Arq Neuropsiquiatr 2006;64(2-A):173-180

SPECIFIC LANGUAGE IMPAIRMENT

Linguistic and neurobiological aspects

Simone Rocha de Vasconcelos Hage[1], Fernando Cendes[2], Maria Augusta Montenegro[2], Dagma V. Abramides[1], Catarina A. Guimarães[2], Marilisa Mantovani Guerreiro[2]

ABSTRACT - Specific language impairment (SLI) occurs when children present language maturation, at least 12 months behind their chronological age in the absence of sensory or intellectual deficits, pervasive developmental disorders, evident cerebral damage, and adequate social and emotional conditions. The aim of this study was to classify a group of children according to the subtypes of SLI and to correlate clinical manifestations with cortical abnormalities. Seventeen children with SLI were evaluated. Language assessment was based on standardized test (Peabody) and a non-standardized protocol, which included phonological, syntactical, semantical, pragmatical and lexical aspects of language. All children, except one, had abnormal MRI. Thirteen children presented perisylvian polymicrogyria. The MRI findings in the remaining three patients were: right frontal polymicrogyria, bilateral fronto-parietal atrophy, and hypogenesis of corpus callosum with Chiari I. The data show that patients with posterior cortical involvement tended to present milder form of SLI (no sign of articulatory or bucofacial praxis disturbance), while diffuse polymicrogyric perisylvian cortex usually was seen in patients who presented severe clinical manifestation, mainly phonological-syntactic deficit. In conclusion, SLI may be associated with perisylvian polymicrogyria and clinical manifestation may vary according to the extent of cortical anomaly.

KEY WORDS: specific language impairment, developmental language disorder, polymicrogyria, malformation of cortical development, perisylvian syndrome.

Distúrbio específico de linguagem: aspectos linguísticos e neurobiológicos

RESUMO - O termo distúrbio específico de linguagem (DEL) é utilizado para crianças que apresentam maturação de linguagem atrasada em pelo menos 12 meses em relação à idade cronológica e que não tenham déficits intelectuais ou sensoriais, distúrbios pervasivos do desenvolvimento, dano cerebral evidente, além de terem condições sociais e emocionais adequadas. O objetivo deste estudo foi classificar um grupo de crianças de acordo com os subtipos de DEL e correlacionar as manifestações clínicas com possíveis anormalidades corticais. Dezessete crianças com DEL foram avaliadas. A avaliação de linguagem foi baseada em teste padronizado (Peabody) e protocolo não-padronizado que incluiu os seguintes aspectos da linguagem: fonológicos, semânticos, pragmáticos e lexicais. Todas as crianças, exceto uma, tiveram RM anormal; treze delas com polimicrogiria peri-silviana. Os achados de imagem nos outros três pacientes foram: polimicrogiria frontal direita, atrofia fronto-parietal bilateral, e hipogenesia do corpo caloso com Chiari I. Os dados mostram que pacientes com comprometimento cortical posterior tenderam a apresentar formas mais leves de DEL (sem sinais de distúrbio práxico articulatório ou bucofacial), enquanto pacientes com polimicrogiria peri-silviana difusa apresentaram manifestação clínica mais grave, principalmente déficit fonológico-sintático. Concluindo, DEL pode se associar a polimicrogiria peri-silviana e as manifestações clínicas podem variar de acordo com a extensão da anormalidade cortical.

PALAVRAS-CHAVE: distúrbio específico de linguagem, polimicrogiria, malformação do desenvolvimento cortical, síndrome peri-silviana.

[1]Department of Speech Therapy, University of São Paulo (USP), Bauru, Brazil; [2]Department of Neurology, University of Campinas (Unicamp), Campinas SP, Brazil.

Received 29 August 2005. Accepted 19 November 2005.

Dra. Marilisa M. Guerreiro - Departamento de Neurologia - FCM / Unicamp - PO Box 6111 - 13083-970 Campinas SP - Brasil. E-mail: mmg@fcm.unicamp.br

Language impairment in children is identified by comparing their language development with other children at the same age. When children present language maturation, 12 months behind their chronological age, it is said that they have a deficit in language development. This deficit can simply indicate a delay in learning or a developmental language disorder. In the case of learning delay, the deficit respects the normal stages of language development and, as time goes by, it decreases with or without therapeutic intervention. In other words, the language manifestations are not persistent. In general, it is a consequence of delayed cerebral maturity or insufficient exposure to linguistic stimulation[1]. Conversely, developmental language disorder is a deviant and persistent impairment with repercussion on written language[2]. Developmental language disorders occur in the absence of sensory or intellectual deficits, pervasive developmental disorders or evident cerebral damage[3]. Furthermore, such cases are not consequences of social or emotional factors. These children present what is called specific language impairment (SLI), one of the most frequent disturbances in the development of higher mental functions in children. One of the main criteria for the diagnosis of SLI is the difference between cognitive linguistic and non-linguistic abilities, evident through the testing of non-verbal intelligence[4], which is usually normal.

Subtypes – SLI cases can present great variability in clinical manifestations concerning language. Some children present difficulties only in expression, others in expression and comprehension of language. Allen and Rapin[5] proposed a SLI classification with six subtypes, divided into three groups. The classification was based on an evaluation of spontaneous and directed language, taking into account the level of linguistic analysis, in terms of phonological, morphosyntactic, semantic-lexical and pragmatic analysis. The subtypes are: *Phonologic-programming deficit:* comprehension is adequate. The child speaks fluently in fairly long utterances, but speech is hard to understand. Sentence structure is generally good, but grammatic markers may be omitted. Speech onset can be either normal or delayed; *Verbal dyspraxia:* comprehension is adequate, but speech is extremely limited, with impaired production of speech sounds and short utterances. There may be signs of oromotor dyspraxia. Some children develop a rich gestural language and profit from learning signs and reading. Speech onset is very delayed; *Phonologic-syntatic deficit:* utterances are short and grammatically incorrect, with omission of functional words and grammatical inflections. Speech articulation is deficient. Word-finding problems are frequent. Comprehension is variable: there may be difficulty in understanding complex utterances and abstract language. Speech onset is very delayed; *Verbal auditory agnosia:* children understand little or nothing of what they hear because they are unable to decode language at the phonological level. Speech is absent or very limited with poor articulation. This syndrome occurs in epileptic aphasia and may be associated with clear EEG abnormalities; *Lexical-syntactic deficit:* children have word-finding problems and difficulty putting their ideas into words. Spontaneous language is superior to language constrained by the demands of conversation or answering questions. Syntax is immature rather than deviant. Production of speech sounds is normal. Comprehension of complex sentences is poor. Onset of speech is usually delayed; *Semantic-pragmatic deficit:* children speak in fluent and well-formed utterances with adequate articulation. However, the content of language is bizarre and the child may be echolalic or use overlearned scripts. Comprehension may be over-literal, or the child may respond to just one or two words in a sentence. Language use is odd, and the child may chatter incessantly or produce language without apparently understanding it. The child is poor at turn taking in conversation and maintaining a topic.

Neurobiological basis – Malformations of cortical development have been seen in children with SLI. Plante[6], using magnetic resonance imaging (MRI), found atypical perisylvian symmetry in six of the eight boys diagnosed with SLI. Duvelleroy-Hommet et al.[7] observed abnormalities in the normal standard of hemispheric asymmetry, especially in the parieto-occipital and parieto-temporal areas.

In a recent study, Guerreiro et al.[8] indicated an association between polymicrogyria on perisylvian region and SLI. Polymicrogyria is an anomaly of cortical development in which neurons reach the cortex but are abnormally distributed, resulting in the formation of multiple small gyri[9]. Perisylvian polymicrogyria has been associated with a wide spectrum of clinical manifestations, such as epilepsy, pseudobulbar signs, cognitive deficits and developmental language disorder or SLI[8,10,11].

The present study has the following objectives: to distinguish linguistic and non-linguistic communicative manifestations of the subtypes of SLI; and to present evidence of the correlation between the clinical manifestations of the different types of SLI and

cortical abnormalities detected on neuroimaging exams.

METHOD

Seventeen children with SLI were evaluated. A comprehensive protocol was applied to study, prospectively, every child presenting language delay as primary complaint. Inclusion criteria were: children should be at least 4 years of age; primary complaint of language delay; normal neurological examination; normal hearing by audiometry; intelligence quotient (IQ) >70; and an informed consent signed by parents or guardians giving permission for their children to take part in this research. The protocol and the informed consent were approved by the ethical committee of our university hospital.

Language evaluation – The language assessment was based on standardized test and a non-standardized protocol.

The standardized test used was the Peabody Picture Vocabulary Test - revised (PPVT), Brazilian standardization by Capovilla and Capovilla[12], to evaluate auditory-receptive vocabulary.

The non-standardized protocol used spontaneous language recorded on VHS video during a one-hour play session. We systematically evaluated, according to a semi-structured protocol, free conversation, repetition, and the following aspects of language: phonological, syntactical, semantical, pragmatical and lexical. Analysis criteria were:

Phonological production. Type of phonological alterations: delayed (phonological simplifications no longer expected at the chronological age, however, observed in the normal language); deviant (phonological simplifications not found in the normal language development); inaccurate (great variation in the articulation of words and increase in the amount of syllable reductions as word extension increases).

Morphosyntactic production (syntax). Sentence structure; nominal and verbal concordance.

Semantic-lexical production. Predominant form of access to lexicon: access using the appropriate lexicon (even with a few words); access using idiosyncrasies; access using periphrases (the use of two or more words instead of an inflected word to express the same grammatical function - example: "that's to eat" instead of "spoon"); and deictics.

Pragmatic evaluation. Conversational abilities (ample, restricted) and communicative functions (ample, restricted).

Comprehension evaluation. Understanding of at least 10 short enunciations (example: "get the pencil"), and 10 long enunciations (example: "get the pencil and put it on the table") with words that have lexical and grammatical meaning.

For children that did not speak or who spoke with restrictions (scattered words and phrases) the language evaluation used the following analysis criteria: intentionality, functionality, engaging in dialogue activities, means of communication and level of comprehension.

Language evaluation was performed by a child speech therapist (S.R.V.H.) specialized in language development. The aim was to categorize abnormal language findings according to the classification proposed by Allen and Rapin[5].

Psychological assessment – Intellectual ability was assessed by the Wechsler Intelligence Scale for Children - III (WISC-III), or the Wechsler Preschool and Primary Scale of Intelligence (WPPSI). Since language delay was required for inclusion into the study, our patients frequently presented verbal IQ scores inferior to performance scores. Low verbal scores jeopardized full scale, therefore we decided to take into account only the performance IQ score because it better represents the cognitive ability of this type of patient.

Neurological examination – A detailed neurological examination was performed and signs of pseudobulbar palsy were specifically investigated. Tongue movements (protrusion, lateral and upward movements) were examined, and the presence of abnormal gag reflex, brisk jaw jerk and automatic-voluntary dissociation of facial movements was specifically noted.

Children with mild developmental motor delay (walking acquisition between 18 and 24 months of age) entered the study providing that developmental language delay was the primary complaint.

Parents or guardians were specifically questioned about a past or present history of drooling, choking, feeding difficulties in the neonatal period, swallowing and sucking problems, and current difficulty of whistling or blowing. A careful family history was searched.

MRI – Neuroimaging investigation was performed in a 2.0 T scanner (Elscint Prestige), using the following protocol: *(a) sagittal* T1 spin-echo, 6 mm thick (TR=430, TE=12) for optimal orientation of the subsequent images; *(b) coronal* T1 inversion recovery, 3 mm thick (flip angle=200°; TR= 2800-3000, TE=14, inversion time TI=840, matrix=130x256, FOV=16x18cm); *(c) coronal* T2-weighted "fast spin echo" (FSE), 3-4 mm thick, (tip angle=120°; TR=4800, TE=129, matrix=252x320, FOV=18x18cm); *(d) axial* images parallel to the long axis of the hippocampi; T1 gradient echo (GRE), 3 mm thick (flip angle=70°, TR=200, TE=5, matrix=180x232, FOV=22x22cm); *(e) axial* T2 FSE, 4 mm thick, (flip angle= 120°, TR=6800, TE=129, matrix=252x328, FOV=21x23cm; *(f) volumetric (3D)* T1 GRE, acquired in the sagittal plane for multiplanar reconstruction (MPR), 1-1.5 mm thick (flip angle=35°, TR=22, TE=9, matrix=256x220, FOV=23x25cm). We performed MPR and curvilinear reformatting in all 3D MRIs[13].

RESULTS

From January 1998 to December 2001, 31 consecutive children with primary complaint of language delay were evaluated. Fourteen were excluded because of a global developmental delay, psychological evaluation showing IQ<70, or because they did

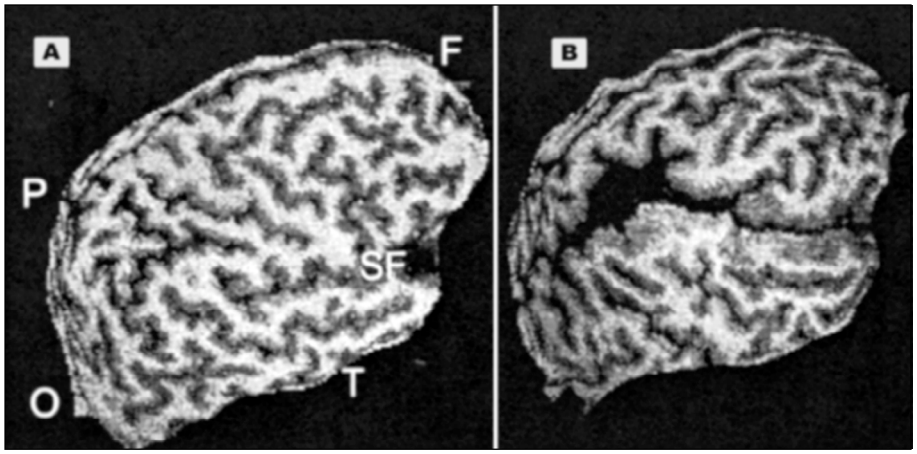

Fig 1. Curvilinear reconstruction from 12 mm of depth from the cortical surface showing (A) normal aspect of the gyri in a normal control [F: frontal lobe, T: temporal lobe, O: occipital lobe, P: parietal lobe and SF: sylvian fissure]; (B) demonstrative display of cortical thickening around the sylvian fissure and focal atrophy at the parietal region in a patient with perisylvian polymicrogyria.

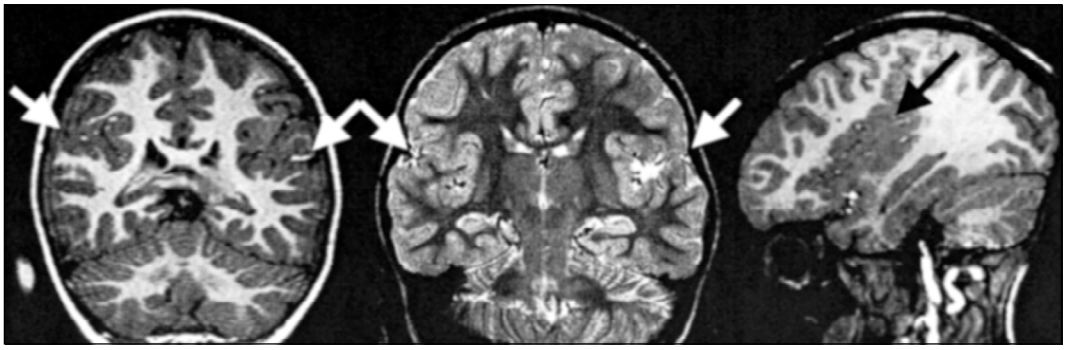

Fig 2. Case 1. Coronal T1-IR and T2 images, and sagittal T1 image showing diffuse polymicrogyria around the Sylvian fissure.

Table 1. Summary data of 17 patients with specific language impairment.

Case	Age	Gender	IQ	Hand	History of pseudo-bulbar difficulties	Family history of SLI	Motor, sensory and coordination systems	Pseudo-bulbar signs
1	4	M	80	R	–	+	Normal	–
2	4	F	100	R	–	+	Normal	–
3	4	M	80	R	–	+	Normal	–
4	4	F	90	R	+	+	Normal	+
5	4	F	80	R	–	–	Normal	–
6	4	F	77	R	+	+	Normal	+
7	4	M	100	R	–	+	Normal	–
8	5	M	88	L	+	–	Mild R hemiparesis	+
9	6	M	107	R	–	+	Normal	–
10	6	M	126	R	–	+	Normal	–
11	7	M	88	R	–	+	Normal	–
12	7	M	88	R	+	–	Normal	+
13	8	F	79	R	+	–	Normal	+
14	8	M	100	R	+	–	Normal	+
15	9	M	75	L	+	–	Normal	+
16	12	M	80	R	–	+	Normal	–
17	14	M	83	R	+	+	Normal	+

Age, age in years; M, male; F, female; IQ, perf o rmance intelligence quotient; Hand, handedness; R, right; L, left; –, absent; +, present; SLI, specific language impairment.

Table 2. Summary data of language assessment.

Case	Type of phonologic alteration	Syntax	Semantics Vocabulary and evocation	Lexical reception Comprehension of sentences	Pragmatics	Subtype
1	NA	NA	Restricted vocabulary	Peabody: 90 Normal	Normal	Expressive deficit
2	NA	NA	Restricted vocabulary	Peabody: 85 Normal	Normal	Expressive deficit
3	NA	NA	NA	Peabody: 60 Short and long sentences: difficulty	Limited communicative ability	Mixed deficit
4	Inaccurate	Abnormal	Restricted vocabulary	Peabody: 85 Short and long sentences: difficulty	Limited communicative ability	Phonologic-syntactic deficit
5	Delayed	Abnormal	Restricted vocabulary	Peabody: 80 Short and long sentences: difficulty	Limited communicative ability	Phonologic-syntactic deficit
6	NA	NA	NA	Peabody: 80 Short and long sentences: difficulty	Good communicative ability with RG	Mixed deficit
7	Delayed	Normal	Adequate vocabulary	Peabody: 110 Normal	Normal	Phonologic-programming deficit
8	NA	NA	NA	Peabody: 55 Short and long sentences: difficulty	Limited communicative ability	Mixed deficit
9	Delayed	Normal	Adequate vocabulary	Peabody: 110 Normal	Normal	Phonologic-programming deficit
10	Delayed	Abnormal	Adequate vocabulary	Peabody:84 Normal	Normal	Phonologic-programming deficit
11	Delayed Deviant	Abnormal	Restricted vocabulary	Peabody: 73 Long sentences: difficulty	Limited communicative ability	Phonologic-syntactic deficit
12	Delayed	Abnormal	Restricted vocabulary Difficulty of evocation Use of periphrases	Peabody: 93 Long sentences: difficulty	Limited communicative ability Prominent word finding difficulty	Lexical-syntactic deficit
13	Inaccurate	Abnormal	Restricted vocabulary	Peabody: 70 Long sentences: difficulty	Limited communicative ability	Phonologic-syntactic deficit
14	Inaccurate	Abnormal	Restricted vocabulary	Peabody: 78 Long sentences: difficulty	Limited communicative ability	Phonologic-syntactic deficit
15	NA	NA	NA	Peabody: 55 Short and long sentences: difficulty	Good communicative ability with RG	Mixed deficit
16	Delayed	Abnormal	Restricted vocabulary	Peabody: 72 Long sentences: difficulty	Limited communicative ability	Phonologic-syntactic deficit
17	Inaccurate	Abnormal	Restricted vocabulary	Peabody: 70 Short and long sentences: difficulty	Limited communicative ability	Phonologic-syntactic deficit

NA, not applicable (no speech); RG, representative gesture.

not complete all steps of the protocol. The remaining 17 children met all inclusion criteria and are the subjects of this study. Fifteen of these 17 children were included in a previous study[8].

Ages ranged from 4 to 14 years (mean = 6.5) and 12 were boys. Demographic data, psychological evaluation (IQ and handedness), history of pseudobulbar difficulties, family history of SLI, neurological examination and the careful search for pseudobulbar signs are presented in Table 1.

Table 2 shows the results of the language assessment. The subjects that did not speak (or who spoke only a few words) could not be classified according to the Allen and Rapin[5] subtypes, which are based upon different levels of linguistic analysis. These children were classified as having a mixed deficit when they showed comprehension difficulties, and an expressive deficit when they did not show any comprehension difficulty.

The correlation between language assessment and MRI abnormalities are presented in Table 3. Regarding imaging abnormalities, the term diffuse polymicrogyria was used when the cortical abnormality occurred around the entire extent of the Sylvian fissure, including the parietal region; while the term posterior parietal polymicrogyria was used when polymicrogyria was restricted to the posterior aspects of the parietal regions, without MRI abnormality at the anterior 2/3 of the Sylvian fissure[14]. Only two children (patients 8 and 11) showed asymmetry of polymicrogyric cortex, which predominated on the left. All other children with polymicrogyria presented symmetric bilateral polymicrogyria (Figs 1 and 2).

The analysis of the results prompted a further division of the findings according to the extent of the polymicrogyric cortex and the severity of the clinical manifestation: patients with posterior parietal cortical involvement tended to present milder form of SLI, while diffuse polymicrogyric perisylvian cortex involving pre-central and frontal regions usually was seen in patients who presented severe clinical manifestation. Only one child had normal MRI. Other three children had different imaging findings. One child had right frontal polymicrogyria on MRI, one had hypogenesis of corpus callosum and Chiari I, and one had bilateral fronto-parietal atrophy.

Table 3. SLI classification and MRI findings.

Case	Language assessment	MRI
1	Expressive deficit	Diffuse bilateral perisylvian PMG
2	Expressive deficit	Normal
3	Mixed deficit	Bilateral posterior parietal PMG
4	Phonologic-syntactic deficit	Diffuse bilateral perisylvian PMG
5	Phonologic-syntactic deficit	Bilateral fronto-parietal atrophy
6	Mixed deficit	Diffuse bilateral perisylvian PMG
7	Phonologic-programming deficit	Bilateral posterior parietal PMG
8	Mixed deficit	Diffuse bilateral perisylvian PMG
9	Phonologic-programming deficit	Bilateral posterior parietal PMG
10	Phonologic-programming deficit	Bilateral posterior parietal PMG
11	Phonologic-syntactic deficit	Bilateral posterior parietal PMG
12	Lexical-syntactic deficit	Bilateral posterior parietal PMG
13	Phonologic-syntactic deficit	Right frontal PMG
14	Phonologic-syntactic deficit	Diffuse bilateral perisylvian PMG
15	Mixed deficit	Diffuse bilateral perisylvian PMG
16	Phonologic-syntactic deficit	Hypogenesis of cc + Chiari I
17	Phonologic-syntactic deficit	Diffuse bilateral perisylvian PMG

SLI, specific language impairment; MRI, magnetic ressonance imaging; PMG, polymicrogyria; CC, corpus callosum.

DISCUSSION

The identification of the subtypes – The phonologic-syntactic deficit seems to be the most common subtype, and could be delayed, deviant or inaccurate. The subjects with phonological deficit of the inaccurate type presented articulation praxis and bucofacial disorders. This fact raised the question of whether it is possible to differentiate some cases of phonologic-syntactic deficit from verbal dyspraxia, as proposed by Allen and Rapin[5]. On neurological examination, most of them presented pseudobulbar signs (Table 1).

Dyspraxia seems to be the basis for the inaccurate phonological alterations and for the limited ability

in producing enunciations. Even the comprehension difficulties can be partially justified by the difficulties in motor planning for speech: the constantly improving repetition of the words allows the child to identify the underlying abstract segments and to form long term representations in the memory[2]. Thus, alterations in production interfere in the perception of language development in children. The lexical-semantic aspect of the language of these subjects was also slightly affected. The access to words was, however, considered appropriate, that is, even though fewer words were used, they were not substituted by deictics or periphrases. The involvement of other linguistic subsystems is justified, even if secondarily, because the process of semantic, syntactic and phonological acquisition is closely connected during the period of language development. Neurophysiological changes influence the emerging phonology, the same way that the cognitive-linguistic development leads to automation of speech motor control. Thus, syntactic simplification strategies interact with phonological simplification strategies, and even with lexical selection strategies, which can justify the restricted vocabulary of the subjects described. It seems that lexicon amplification is also related to the capability of producing them.

The conversational and narrative abilities of the subjects with phonologic-syntactic deficit were shown to be restricted. However, this was not considered to be a fundamental component of this disorder. Children with phonologic-programming deficit presented the same characteristics. They were, however, considered to be secondary to phonological deviations. These children avoid speaking because they are aware of their difficulty. The jeopardized speech intelligibility interferes in conversational abilities, language functionality, and interest for the narrative[4].

Three subjects (patients 7, 9 and 10) were classified as having phonologic-programming deficit because they had problems in the phonological aspect of language. These subjects did not present any articulatory and buccofacial praxis disorder, or phonological deficit of the inaccurate type. Some difficulties in comprehension of long enunciations were observed, however, they were much more subtle when compared with the other children.

One subject was classified as having a lexical-syntactic deficit due to the difficulty of lexical evocation and memorization. The fundamental characteristic of this disorder is the access to lexicon through deictics and periphrases. Lexical-syntactic deficit was observed in this subject during a sample of spontaneous language and of provoked nomination, and fluency was jeopardized. Another aspect found was the good reception for isolated words, but difficulties in the understanding of long enunciates[2]. This child presented these characteristics: the formal evaluation showed difficulties in the understanding of long enunciates, but the Peabody testing indicated average lexical reception.

The subjects that did not speak (or who spoke so little that we were unable to apply the protocol) were classified as presenting a mixed deficit when they had comprehension disorder and expressive deficit when they did not present any comprehension disorder. The comprehension difficulty was evident in the understanding of short or long enunciates as well as in the lexical reception testing (Peabody). Oral language comprehension was jeopardized in all 17 subjects for lexical reception (Peabody: lower score for all subjects) as well as for the comprehension of long enunciations. Even though it is difficult to evaluate comprehension, many children with SLI have difficulties in understanding sentences[2]. Our data reinforce this statement.

The comprehension difficulties observed in most of the subjects in this study are most likely related to what is called processing difficulties. The processing of a sentence in higher levels (comprehension) requires a series of transformation processes, that is, one type of information must be transformed into another: phonetic information, which is a result of the first analysis of spoken language, must be transformed into phonological information, then this information must be transformed into semantic information, and consequently into an idea. Processing limitation means difficulty in transforming one information into another[3].

Neuroanatomic correlations – Most children presented cortical abnormalities in areas related to the oral language. The abnormality most commonly found was perisylvian polymicrogyria. The subjects presenting diffuse polymicrogyria along the Sylvian fissure, extending to the frontal areas, had mainly phonological-syntactic deficit. It is important to note that three of the four subjects who presented absence of speech also had diffuse perisylvian polymicrogyria. The phonological deficit found in the subjects of this group who were able to speak is mainly of the inaccurate type, which is characteristic of articulatory or bucofacial praxis disorder. The two subjects with frontal anomalies (patients 5 and 13) were classified

as having a phonologic-syntactic deficit as well. The frontal lesions may explain the articulatory and bucofacial praxis disorder found in these children.

The posterior parietal polymicrogyria was present in children with mixed deficit, phonologic-programming deficit, phonologic-syntactic deficit or lexical-syntactic deficit. None of the patients presented signs of articulatory or bucofacial praxis disorder. The subjects who were able to speak did not present any phonological difficulty of the inaccurate type. Galaburda et al.[15] presented the neuroanatomical findings in four brains of men with developmental dyslexia. They found perisylvian polymicrogyria in two patients who had had SLI. Worster-Drought[16,17] presented a classification on speech disorders in children. Under the denomination of congenital suprabulbar paresis he described patients who had SLI and weakness of speech musculature due to impaired development of cortico-bulbar tract proceeding from the motor cells of the lower part of the rolandic cortex. He pointed out that the result was varying degrees of paralysis of the lips, tongue, soft palate, and laryngeal and pharyngeal muscles (in other words, suprabulbar paresis). Worster-Drought's description fits several of our patients who presented pseudobulbar signs on neurological examination (Table 1). More recently, Worster-Drought syndrome has been considered as lying in a continuum with congenital perisylvian syndrome, which is clearly associated with polymicrogyria around the Sylvian fissure[8,10,18,19].

In conclusion, this study shows that SLI may be associated with perisylvian polymicrogyria. Considering the spectrum of clinical manifestations and the spectrum of MRI abnormalities, our data allow the proposition of two subgroups: SLI with severe clinical manifestation and neuroimaging showing extensive cortical abnormality around Sylvian fissure; and, SLI with mild clinical manifestation and neuroimaging showing mainly posterior parietal abnormality.

REFERENCES

1. Rapin I, Allen DA, Dunn MA. Developmental language disorders. In SJ Segalowitz, I Rapin (eds). Handbook of neuropsychology. New York: Elsevier; 1992:111-137.
2. Leonard LB. Children with specific language impairment. Cambridge: MIT Press, 1998.
3. Bishop DVM. Uncommon understanding: development and disorders of language comprehension in children. Cambridge: Psychology Press, 1997.
4. Hall DM, Hill PD. Communication disorders. In DM Hall, PD Hill (eds). The child with a disability. London: Blackwell; 1996:173-209.
5. Allen DA, Rapin I. Communication disorders of preschool children: the physician's responsibility. J Dev Behav Pediatr 1988;9:164-170.
6. Plante E. MRI findings in the parents and siblings of specifically language impairment boys. Brain Language 1991;41:67-80.
7. Duvellerоy-Hommet C, Gillet P, Billard C, Loised M, Barthe Z, Santini J. Study of unilateral hemisphere performance in children with developmental dysphasia. Neuropsychologia 1995;33:823-834.
8. Guerreiro MM, Hage SRV, Guimarães CA, et al. Developmental language disorder associated with polymicrogyria. Neurology 2002;59:245-250.
9. Barkovich AJ. Congenital malformations of the brain and skull. In AJ Barkovich (ed). Pediatric neuroimaging, 2nd ed. New York: Lippincot-Raven; 1996:177-275.
10. Guerreiro MM, Andermann E, Guerrini R, et al. Familial perisylvian polymicrogyria: a new familial syndrome of cortical maldevelopment. Ann Neurol 2000;48:39-48.
11. Kuzniecky R, Andermann F, Guerrini R, and CBPS study group. Congenital bilateral perisylvian syndrome: study of 31 patients. Lancet 1993;341:608-612.
12. Capovilla FC, Capovilla AGS. Desenvolvimento lingüístico na criança brasileira dos dois aos seis anos: tradução e estandardização do Peabody Picture Vocabulary Test e da Language Development Survey de Rescorla. Ciência Cognitiva 1997;1:353-380.
13. Bastos A, Comeau RM, Andermann F, et al. Diagnosis of subtle focal dysplastic lesions: curvilinear reformatting from three dimensional magnetic resonance imaging. Ann Neurol 1999;46:88-94.
14. Montenegro MA, Guerreiro MM, Lopes-Cendes I, Cendes F. Bilateral posterior parietal polymicrogyria: a mild form of congenital bilateral perisylvian syndrome? Epilepsia 2001;42:845-849.
15. Galaburda AM, Sherman GF, Rosen GD, Aboitiz F, Geschwind N. Developmental dyslexia: four consecutive patients with cortical anomalies. Ann Neurol 1985;18:222-233.
16. Worster-Drought C. Congenital suprabulbar paresis. J Laryngol Otol 1956;70:453-463.
17. Worster-Drought C. Speech disorders in children. Dev Med Child Neurol 1968;10:427-440.
18. Clark M, Carr L, Reilly S, Neville BGR. Worster-Drought syndrome, a mild tetraplegic perisylvian cerebral palsy. Brain 2000;123:2160-2170.
19. Nevo Y, Segev Y, Gelman Y, Rieder-Grosswasser I, Harel S. Worster-Drought and congenital perisylvian syndromes: a continuum? Pediatr Neurol 2001;24:153-155.