SPECIFIC LANGUAGE IMPAIRMENT: NEUROPSYCHOLOGICAL AND NEUROLINGUISTIC ASPECTS

Maria Pąchalska[1], Grażyna Jastrzębowska[2], Małgorzata Lipowska[1], Anna Pufal[3]

[1] Institute of Psychology, University of Gdańsk, Gdańsk, Poland
[2] University of Opole, Opole, Poland
[3] Center for Cognition and Communication, New York, NY

SUMMARY

This paper discusses neuropsychological and neurolinguistic aspects of specific language impairment (SLI), classified among the developmental speech disorders. SLI constitutes one of the more important manifestations of irregularities in the development process and is among the earliest observed in children. SLI results from the uneven development of linguistic processes – phonological, semantic and/or syntactic – and is caused by hereditary and environmental factors with a negative impact on the activity of the brain. One can speak of SLI in a particular child if the retardation in the development of speech and language reaches 12 months in comparison to the average level of linguistic competence in children of the same calendar age. We do not diagnose SLI if there are other reasons for the observed speech and language symptoms, such as serious sensory and cognitive deficits, permanent developmental impairments (e.g. autism), massive brain injury, or the lack of adequate social and emotional conditions for the development of communication skills. According to WHO categories, SLI manifests itself as an impairment of individual language functions resulting in disturbed communication with other people, which, in turn, leads to a handicap resulting from social isolation, difficulties at school and consequent loss of the opportunity to adapt oneself to adult life in society. Even though the level of impairment in a given patient results from the pathology causing SLI, the handicap level depends on the patient's compensation ability, already available or learned in the rehabilitation process, while the level of limitation depends on the patient's adaptation to the existing situation. The problems of

differential diagnosis and the therapy of such impairments will also be discussed. We will present our own research covering the analysis of 20 children diagnosed with SLI, whose impairments resulted in extremely different symptom profiles. The results will be subjected to discussion in the light of microgenetic theory.

INTRODUCTION

The subject of specific language impairments has been arousing scientific interest for many years. This interest stems from both practical reasons, i.e. the need to help children who have difficulties in communication with the environment, and theoretical reasons relating to the cerebral mechanisms underlying the analysed impairments. In fact, both these aspects are strongly interrelated, as it is difficult to talk about an effective speech and language therapy process without proper theoretical knowledge of the cerebral mechanisms underlying its organisation. It is commonly known that a thorough study of the nature of speech impairments makes it possible to learn more about the rules regulating the operation of the brain in general. As early as 1887, Fournié expressed the opinion that "language is the only window through which a physiologist may watch the life of a brain" (cited by Bianchi 1922).

The correct development of speech and language depends on a range of various factors. where an appropriate level of psychomotor functions plays an important role, which means that there exists a close correlation between the development of cognitive, emotional-motivational and executive processes and the development of speech and language (Robertson & Weismer 1999). On the one hand, this dependency is reflected in differences in the consolidation of language skills and retardation in speech development observed in the majority of children with partial and global developmental impairments – but not only in such children. On the other hand, retardation in speech development is also a common feature of those children whose impairment in language development is a primary (specific) impairment of undetermined etiology and those children who begin to talk late due to slower maturation of the nervous system.

The application of the term *delayed speech development* (DSD) may be useful when a lack of normal speech development of undetermined etiology is observed in the first years of a child's life. This is because the term does not suggest the cause and, hence, the nature of the existing deviation from the developmental standard. but rather signals the existence of differences, statistically more or less significant, and this in turn encourages specific diagnostic and educational action. In such cases, the DSD diagnosis is only a preliminary, cautious diagnosis that should be verified, a statement of the fact that deviations from the norms have appeared in the speech development process, but these deviations are not necessarily of great importance for further development. The same is done with regard to mental impairment

(or intellectual handicap), which, as a rule, remains undiagnosed until the age of three, and the term "delayed mental development" is used. However, this approach does not justify a widespread application of the term "delayed speech development," as the name of a diagnostic unit in all retardation cases (see the typology of speech impairments in Pruszewicz 1992), the more so because it is not compliant with the international medical classifications of ICD-10 and DSM-IV-TR. However, we can agree with Grabias (2001) here that the term "a delay in speech development" is becoming a synonym for the term "impairments in speech development." However, if we accept that the term *DSD only applies to small children in whom it is difficult to specify the cause of the delay* it will assume a different meaning. It will be a cautious preliminary diagnosis in need of permanent verification, and it will be the only justified use of the term "delayed speech development" as the name of a diagnosis.

So, what does the "slower maturation of the nervous system" consist in?

There is no simple answer to this question. However, the neuropsychologist and clinical speech therapist are most interested in neuronal connections. These connections are quite modest in number at birth, but increase in number rapidly in infancy and achieve maximum density at the age of six (Brown 2001). Later, the quantity of synapses is reduced again because redundant connections drop out (see fig. 1) according to the rules of the natural selection: those neuronal solutions that are the most effective and useful remain, while others, less effective, drop out. Adults can keep increasing the quantity of synapses throughout their lives if they learn (acquiring new information and skills) even though the replacement of old solutions with new ones becomes slower and slightly more difficult with years. Unused synapses disintegrate.

Individual neurons in a developing brain rapidly search for organised sets of other neurons to join them. Each cell has to find its place in the system; otherwise, it will perish in a process called apoptosis, i.e. the genetically programmed death of cells. Apoptosis in an immature brain is aimed at strengthening and rationalizing connections between the cells that survive, and it is also supposed not to permit the literal "strangling" of the brain with its own cells. This "pruning" process, although it is necessary, has its price as well. Connections that are destroyed in its course also include those that guarantee a range of intuitive abilities and talents. For example, photographic (eidetic) memory is a relatively frequent phenomenon in small children, but usually vanishes later due to the discarding of the synapses required (Riccio 2007). An incomplete apoptosis may provide an explanation for the astonishing isolated mental talents called the "idiot savant" syndrome, at the same time accounting for their mental deficiencies. On the contrary, apoptosis that goes out of control and destroys too many synapses is considered to be one of the causes of reduced intelligence in Down syndrome. This probably also explains the higher likelihood of Alzheimer's disease in persons with Down syndrome, as well as in those who "vegetate" at an elderly age, avoiding
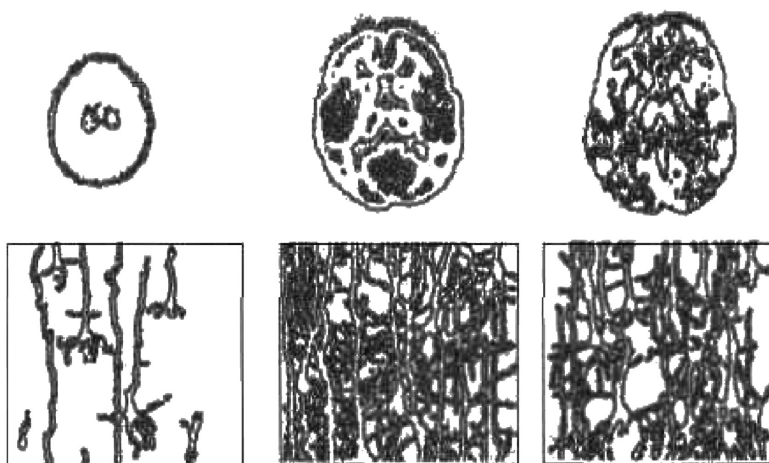
Fig. 1. Neuronal connections: scanty at birth (left), developing in infancy, with maximum density at age six (middle), decreasing due to the dropping out of redundant connections in adults (right). Note: Adults can increase the quantity of connections throughout their lifetimes by learning (acquiring new information). A brain that is not used loses the connections. If the deficit is high (regardless of its developmental or acquired nature), various impairments of cognitive processes appear gradually, including speech and language retardations and impairments [Source: Brown 2001, with the author's consent]

mental activity. It also influences the verbal intelligence ratio decrease observed in children with SLI (see de Vasconcelos Hage et al. 2006).

Differences in forming the ability to use language in children

Many authors stress the differences appearing in children in the fixing of their ability to use a language. In the supplement to the ICD-10 (1997:196), we read that "normally developing children differ considerably from one another when it comes to the age at which they begin to talk, as well as the speed at which the ability to use language is formed. However, such developmental differences are considered relatively unimportant from the clinical perspective. because the observed delay in the development of speech is usually resolved spontaneously before the end of the third year of life without the need for any specialist intervention." ICD-10 defines all irregularities symptomatic for an individual development rate and rhythm as n*ormal variations in development* as opposed to *clinically significant impairments.*

The term *impairment* – in the opinion of the authors of ICD-10 (1997:16) – is a primary concept used in order to "indicate the existence of a system of clinically observed symptoms or behaviours in the majority of cases entailing distress and impairment of individual functions." According to that approach, *speech impairments* should refer to all linguistic behaviour disorders manifesting in various ways that result from the lack of the skills needed to construct or comprehend a verbal statement. In the case of small children (until the third year of age), due to the developmental process still in progress, no

speech impairments are diagnosed, but the term *impairment of speech and language development* is used. Such a differentiation is necessary because the lack of speech development or a delay in the acquisition of language competences and skills can (but need not necessarily) have a pathological background. In the case of impairments where the etiology is difficult to determine, it is not possible to foresee whether the irregularities in speech acquisition existing in a child will disappear spontaneously or whether they will intensify and, consequently, lead to permanent changes in the form of speech defects.

There is little consistency among authors, however, as to the level of retardation in the acquisition of phonological, lexical, and syntactic skills that should be considered pathological; for example, if a child turns three and can only say a few words, this is no more than a warning sign according to Zaleski (1992), while Spionek (1981) states that, if a child at that age actively uses only a few dozen words instead of a thousand or several hundred, such a child's speech should be considered seriously retarded. According to Rapin (1996:649), even though a considerable percentage of such children "talk in a way appropriate for school age, which gave rise to the *theory of retardation or deviation in development and not an impairment* (see also Bishop & Hayiou-Thomas 2007), many of them will draw attention for the second time when they will have difficulties in reading and orthography at school age."

It is worth stressing that this description applies to children with SLI. Numerous studies show that about 7% of children at pre-school age differ from their "normally developing peers" in that they do not learn to talk quickly and effortlessly (Leonard 2000). In their case, the speech development process does not progress according to a typical scheme. Moreover, despite many similarities, its progress does not resemble either that of younger, normally developing children or that of their "late bloomer" peers. A low language acquisition rate is characteristic for this 7% of children; however, the delay is related not only to the speed of the developmental process. As a consequence of restrictions in language capabilities and possibilities, the majority of such children never become fluent in their command of language (at least in some of its domains).

Leonard (2000) stresses that the research done in Western countries shows that 25-50% of 2 year-olds with a slower speech development rate are threatened with SLI. As we can speak of the "risk of dyslexia," we should also assume the existence of "SLI risk" in the case of any 2-year-old in whom there is a delayed beginning and protracted development of the linguistic expression and/or perception (with an undeterminable etiology).

Distinguishing language impairments and speech impairments

Many authors agree that the problem area of impairments in speech (i.e. articulation) should be explicitly separated from language impairments

(Taylor-Sarno 1981; Kaczmarek 1995; Jastrzębowska 2003, 2005; Pąchalska 1999, 2007). The differentiation of language impairments and speech impairments appeared in the neuropsychological and clinical speech therapy literature late in the last century (Robertson & Weismer 1999; see also Pachalska 1999). Thus

1. *language impairments,* from the perspective of developmental impairments, are defined as difficulties in the acquisition of language skills, i.e. impairments of phonology, syntax, semantics or pragmatics (central mechanism). From the perspective of acquired impairments, this means the complete or partial loss of linguistic abilities.

2. *speech impairments,* from the perspective of developmental impairments, are difficulties in the execution of a verbal statement despite normally developed language abilities. They are most frequently identified with impairments in the production of utterances, i.e. articulation, phonation, breath, or fluency of speech (peripheral mechanism). From the perspective of acquired impairments, we can also speak of lesser or greater difficulties in articulation, phonation, breath or fluency of speech, dysarthric speech, etc.

Even though both language impairments and speech impairments result in text deformations, the symptoms of language impairments differ from those of speech impairments, which is important for the clinical practice of both neuropsychologists and speech therapists. The background of disorders in linguistic behaviour is different, and hence the objective of therapy is different as well.

The essence of the specific language impairments (SLI)

To stress that specific disorders are only specific for childhood, as opposed to those that can appear in almost any life period, the authors of DSM-IV-TR (2000) apply the term *developmental language disorders* as opposed to *acquired disorders*. According to this influential manual, development language disorders are divided into four types, namely:

1. *specific language impairment* (SLI), also called the pure form of developmental language disorders (DLD), defined in the older Polish literature as "childhood aphasia" or "alalia" (see: Kordyl 1968; Parol 1989);

2. *non-specific language development disorders* coexisting with such developmental deficits as hypoacusis or mental impairment;

3. *non-specific language development disorders* relating to infantile autism.

4. *other speech disorders* relating to a general impairment of mental development.

The introduction of the term *specific language impairment* in lieu of such terms as "infantile aphasia" or "alalia" has solved some serious terminological problems in the Polish neuropsychological and clinical speech therapy literature, and, at the same time, considerably improved the comparability of results obtained by Polish scientists with global literature. Moreover, termino-

logical precision also facilitates diagnostic and therapeutic procedures.

In turn, the term *specific language impairment* used in the ICD-10 (1997) classification is intended to stress that, in addition to general, extensive (also called global) mental development disorders, among other things, characterized by irregularities in the language development area, such as autism, atypical autism or Rett's syndrome, there are also other disorders whose aetiology is difficult to determine.

Leonard (2000) states that SLI is a pure form of developmental language disorders (DLD) and is defined as a fragmentary, primary or, in other words, *specific* disorder manifesting itself only in developmental difficulties in language assimilation without other, accompanying developmental deficits. Discussing the essence of SLI, Bishop (1992:51) states that "…according to the traditional approach, it is a disorder whose definition was formulated according to the exclusion rule: a child has serious language difficulties that cannot be explained by hearing impairment, low intelligence level, abnormal environment or physical handicap […] language impairment is a primary deficit in this disease entity. The observed language difficulties are not secondary to other limitations of cognitive abilities but reflect an incorrect development of the module specialized in language processes."

Hence SLI is a measurable delay in the assimilation of language, as opposed to apparently typical development of other cognitive and emotional processes. The deficit is mainly limited to the language area, as opposed to cases where a more global model of developmental disorders.

Definition of SLI

According to a review of the latest work in neurosciences (Rapin et al. 1992; Duvelleroy-Hommet et al. 1995; Bishop 1997; Hall and Hill 1996; Leonard 2000; 2006; de Vasconcelos Hage et al. 2006) as well as many years of study carried out by the present authors with regard to patients with various kinds of brain damage, and a deeper knowledge of microgenetic theory, we suggest the following definition of SLI:

Specific language impairment (SLI) consists in a complete or partial lack of the normal organization of language processes (phonological, semantic and/or syntactic) caused by the instability of the brain systems responsible for language, resulting from hereditary and environmental factors, in the absence of sensory and cognitive deficits, permanent developmental disorders (such as autism or similar syndromes), evident brain injury, social or emotional deprivation or other factors potentially causing secondary language and speech impairments..

We can speak of SLI if the delay in the development of speech and language reaches 12 months in comparison with the normal level of language functions in children of the same age,

To help the reader understand more fully the essence of the specific language impairment, it is necessary to analyse individual elements of that com-

pound definition. This will be done in the following subsections.

Partial or complete lack of process organisation

The concept of a "complete or partial lack of process organisation" means a pathological (undesirable) change in the progress of brain processes that guarantee the effective participation of a child in a speech act. In turn, "language processes" are understood here as a set of cerebral functions engaged in the construction and understanding of various kinds of utterances according to linguistic rules:

– *phonological*, i.e. relating to the sound, the acoustic structure of an utterance;
– *semantic*, i.e. relating to the process of associating words with meaning;
– *syntactic*, i.e. the creation and interpretation of sentences (e.g. statements, orders, questions, etc.).

Disorders may appear in a child on the level of the language behavior in one, two or all three of these linguistic domains, or, to put it somewhat differently, there occur signficant violations of the rules mentioned above in speech (or in writing) to a degree endangering the effectiveness of the act of speaking.

However, it should be remembered in this context that, in principle, the entire process illustrated – with great simplification – on fig. 2 takes place in the "black box" of the brain, i.e. in an "invisible place" inside one's skull. Any disturbance of that process in any place along the entire route causes "damage" to the text, on the basis of which the neuropsychologist tries to find the cause of the disorders.

In preliminary testing, then, the first task is to determine whether a disturbance is occuring somewhere in the zone of "linguistic processes," i.e. related to a specific language impairment, or whether it comes from a different zone, e.g. from confusion (in the domain of "prelinguistic processes") or laryngoplegia (belonging to the group of postlinguistic processes). If the observed disorders in a child's speech result rather from disturbances in cognitive or emotional processes, additional study of that area is required. In children with SLI (especially when deep), other cognitive processes can be dis-
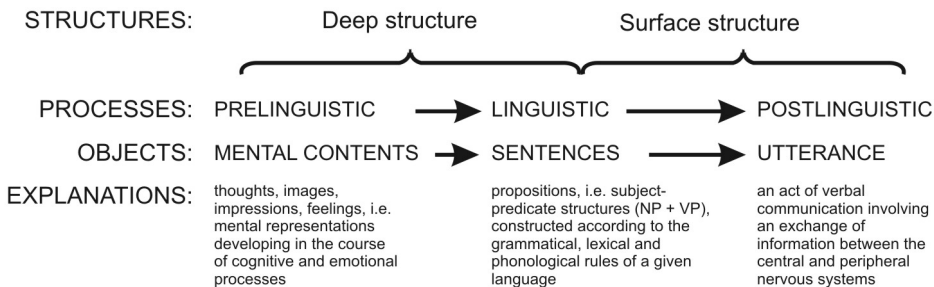

Fig. 2. The speaking process: prelinguistic, linguistic, and postlinguistic processes [Source: Pąchalska 2007]

turbed as well (e.g. perception, memory, attention, thinking). Personality and identity as well as emotional modifications may also occur as a reaction relating to the functioning of the injured brain or as a psychogenic reaction to restrictions in communication. A consultation with a (neuro)psychiatrist is also required in the case of disorders in emotional functioning.

Instability of the language system

The term "instability of the language system" is an important component of the definition proposed here. In systems theory, a "system" is a set of elements that cooperate with one another under the influence of forces present in the system. A system is stable if the influencing forces are so balanced that the elements remain the same with regard to one another or change rhythmically within the foreseeable limits of tolerance. As a result of instability, either the elements cooperate in an unforeseeable or ineffective manner, or they cease to cooperate at all. In the case of language processes, the "cerebral system" consists of neurons and complex neural systems that cooperate in one way or another in the progress of phonological, semantic, and syntactic processes. However, the language system is not inborn. The underdevelopment of specific brain regions in a child or even poor linguistic upbringing may lead to instability of the language system. If these factors exist even before the appearance of hemispheric specialization, i.e. before the development of "speech centers" in the left hemisphere, we cannot speak of the existence of a language system.

A language system is not only a three-dimensional entity, but also – and essentially – a four-dimensional one, i.e. existing in time (Brown 2001, Pąchalska 2007). One weakness of cognitivist brain models is that they assume the existence of stiff, solid brain "processors" (i.e. specific areas or fields of the cerebral cortex) linked with one another with the use of "cables" or neuronal paths. Such models do not take into account the undeniable fact that a neuron is dynamic in its essence because, unlike a silicone chip, it does not contain any recorded information, but only transmits the information by way of changes of polarization of the cell membrane. Hence, neural systems cannot be approached solely through their structural elements with no consideration of dynamic changes in the balance of forces within that system, because it is those changes in time and not their mutual spatial relations that define the operation of a neural system.

SLI affects the language system, understood as the *system transforming our ideas, observations, feelings, etc.* (jointly referred to as "mental events") *into statements structured in accordance with the phonological-graphological, semantic and syntactic rules in force in a given language.* The efficient operation of this system is a necessary condition for effective communication with other people. In turn, successful communication depends on numerous variables: for example, on the ability of both the sender and the addressee to use the same communication channel, i.e. knowledge of the same language.

However, successful communication also depends – and this is probably most important in our case – on the ability of each of the parties (i.e. the sender and the addressee) to process language information. If the language system is instable due to SLI, other systems (cognitive, emotional, family, social) frequently become destabilized as well, and that instability spreads like waves on water when a stone falls in.

Sub-types of SLI

SLI can show a clinically high variability of symptoms relating to language. Some children only present disorders in speaking, others - only in compre-hension, still others - both in speaking and in the understanding of other peo-ple's utterances. Rapin (1996) suggests distinguishing 6 subtypes of SLI, which can be combined into 3 groups. The classification is based on the eval-uation of spontaneous and direct utterances, considering the level of lan-guage analysis, i.e. phonological, morpho-syntactic, semantic-lexical and pra-gmatic (Lum & Bavin 2007).

Subtypes include:

– *Phonological programming deficit.* – Understanding is correct. The child speaks fluently using relatively long utterances, but her speech is difficult to understand. Sentences are usually grammatically correct but inflection may be lost. Speaking may commence in a normal time period or with a delay.

– *Verbal dyspraxia.* Understanding is correct, but speech is limited, with dis-turbed creation of sounds and short utterances. Symptoms of oral dysprax-ia may appear. Rich gesticulation develops in some children, who may use sign language, reading and writing. The commencement of speaking is considerably delayed.

– *Phonological-syntactic deficit.* Utterances are short and grammatically in-correct with omitted functors and inflection. Articulation is incorrect. Problems in searching for words occur frequently. The level of compre-hension varies: difficulties in the understanding of complex utterances and abstract concepts may appear. The commencement of speaking is very delayed.

– *Verbal auditory agnosia:* There is a complete lack of comprehension or a considerably impaired understanding of other people's utterances, be-cause these children have no ability to decode utterances on the level of phonemes. The child does not talk or talks very little and has considerable articulation impairments. This syndrome frequently accompanies epileptic aphasia, and can occur in children in whom EEG irregularities have been observed.

– *Lexical-semantic deficit.* Children have problems finding words and diffi-culties in expressing themselves. Spontaneous speech is better than speech limited by the requirements of conversation or giving answers to questions. Syntax is underdeveloped rather than incorrect. The articula-

tion of individual phonemes is correct. The understanding of complex sentences is limited. The commencement of speaking is usually delayed.

– *Semantic-pragmatic deficit.* The child speaks fluently and correctly in the formal respect; however, the contents of utterances can be bizarre, with echolalia or overuse of verbal stereotypes (e.g. you know, you see, exactly, gosh, etc.). Understanding can be literal, or the child may react only to one or two words in a sentence. The choice of words is atypical (bizarre); the child may also talk incessantly or speak her mind on various topics without understanding what she is saying. The child does not maintain the topic of the conversation and interrupts other speakers.

According to Leonard (2000), the likelihood of the appearance of specific language impairments (SLI) is higher in boys and also in those children whose parents or siblings had difficulties in language assimilation in the past. Robertson and Weismer (1999) observed that these children, despite apparently catching up after a delay (nearly half make up for the deficiencies in lexical resources by the age of 3), continue to show retardation in other spheres of language (phonology, morphology, syntax or narrative abilities). At about age 7, the majority of children with SLI meet expectations relating to speech development and reading skills, but their results are clearly worse than those attained by children from control groups selected according to age and non-verbal cognitive abilities in various functional areas of language (Rescola 1993, cited by Robertson & Weismer 1999).

Guerreiro et al. (2000) stress that scientific research points to certain language areas as being particularly difficult for children with SLI, even though differentiation of language profiles (in other words, differentiation in the SLI subtypes presented above) is considerable in that population. Tager-Flusberg and Cooper (1999) observed that children with SLI experience problems relating to language skills not only at school, but also in later periods. They stress that children with SLI are characterized by adequate mental development, good flexibility of the organs of speech, and correct physiological hearing (even though hearing deterioration may occur in the case of mixed receptive-expressive syndromes). These children show hyperactivity or are prone to seclusion; there are concentration difficulties and high distractibility, no ability to enjoy recreation in the company of their peers (Norrix et al. 2007). No other deviations from the standard – except for speech disorders - are found in the course of a neurological examination. There are no significant impairments in the executive apparatus of speech.

SLI more frequently appears in children with genetically conditioned left-handedness or ambidexterity than in right-handed children. Cases of speech impairments, dyslexia, and dysgraphia can be found in their families (Duvelleroy-Hommet et al. 1995; Dilling-Ostrowska 1990; Galaburda et al. 1985).

Causes of SLI

People with SLI do not form a uniform group when it comes to symptoms

because the causes of SLI are varied. There are several different concepts concerning the organic background of delayed speech development. Historically speaking, we can observe a transfer of emphasis from neurological factors relating to dysfunctions in the prenatal and perinatal period, through the influence of the family environment, in the direction of genetic factors. There is no doubt at present that SLI is a disorder with a heterogeneous etiology (Leonard 2006).

Neurological dysfunctions have been mentioned as a key background for SLI, but unfortunately the data obtained are not unambiguous. Prenatal and neonatal complications have been considered key causes of language dysfunctions for a long time. The role of prenatal risk has been stressed (alcohol abuse by pregnant women or disturbances of the mother's balance of hormones, mainly testosterone). However, this concept has not been verified by sufficient empirical evidence (Merricks et al. 2004).

Searching for the neurological background for delayed speech development, with particular consideration for persons with various language dysfunctions, such as SLI or developmental dyslexia, many authors indicate the occurrence of atypically large vicinity of the fissure of Sylvius in comparison to the healthy population (Bishop & Snowling 2004), in the opinion of many scientists related to polymicrogyria (see de Vasconcelos Hage et al. 2006). Polymicrogyria is an anomaly in the development of the cerebral cortex, in which neurons from deeper layers of the brain reach the cortex level but are incorrectly spaced, as a result of which numerous minute convolutions develop in the cortex instead of or in addition to the normal system of gyri and sulci. De Vasconcelos Hage et al. (2006) state that various abnormal brain formations occur in children with SLI. Plante (1991) using MRI studies discovered an atypical symmetry of the fissure of Sylvius in six out of the eight children with the SLI he analysed. Duvelleroy-Hommet et al. (1995) found the existence of anomalies in the normal, standard asymmetry of hemispheres, mainly in the parieto-occipital and parieto-temporal areas. Within the last few years, Guerreiro et al. (2000) as well as Vasconcelos Hage et al. (2006) have showed a connection between the existence of polymicrogyria in the perisylvian area and the occurrence of SLI. The authors in their clinical studies, corroborated by brain neuroimaging studies (MRI), found that polymicrogyria in the perisylvian region is associated with a range of symptoms and clinical syndromes, including epilepsy, pseudobulbar symptoms (i.e. dysarthria with nasal speech, swallowing difficulties, frequent crying and/or laughing that is difficult to control), cognitive deficits and developmental language disorders (DLD), or SLI (Guerreiro et al. 2000; Barkovich 1996).

Many studies show that children with SLI come from families with varied problems in language development, which applies to ca. 30-60% of this group (Flax et al. 2003). Such an accumulation of symptoms in families may be a consequence of both genetic and environmental factors, and most likely is the result of their combination. Studies searching for the genetic causes

of language difficulties have not brought explicit answers so far, but they are surely polygenetically controlled. Not only questionnaire data gives evidence for this, but also, in particular, studies based on the comparison of twins (Bishop et al. 2006). Barlett et al. (2002) indicated the long arm of chromosome 13 or, more exactly, 13q21, as the localization related to the occurrence of SLI; they also indicated loci 3q21, 2p22 and 17q23 as potentially playing a role in the inheritance of SLI. However, it is impossible to neglect the role of the environment as a factor influencing the appearance of delayed speech development. Two concepts regarding this relationship have been empirically verified. First, as children with SLI come largely from families with language dysfunctions, the utterances addressed to them differ from those heard by children raised in other families. Secondly, it is also possible that adults relieve children of the need to participate in the communication process, and thus no need to model the language system appeared in them (Leonard 2006).

It is worth observing that the correlation between the location of an injury and the nature of disorders in a patient's speech is much weaker in children than in the adults. A child's brain is much more flexible, because the process in the course of which the asymmetry of functions develops has just begun. Only microgenetic theory can explain these facts (see Pąchalska 2003), so a critical view of the entirety and dynamics of symptomatology of SLI is significant. It is also worth observing that brain systems do not operate in a vacuum, which is why disturbances within one system can have unforeseeable consequences within another system.

We can speak of a "delay" if the observed irregularities in a child's speech are only of the quality appropriate for earlier phases of development (i.e. are characteristic for younger, correctly developing children) when, after a period of more or less serious delay, a child's speech attains the level appropriate for a given age group. In the case of significant deviations from the standard, i.e. a slower acquisition of language competence, but also the occurrence in a child's speed of significant quantitative-qualitative differences (quantity of errors and their type), when the attained level of development of language skills (system and/or communication) differs from (is significantly lower than) the level of peers, we should speak of a "deviation," an "impairment," or a "disorder" in development, and not about "delay." This is related to differences in language acquisition models, which are different for "late bloomer" children and different for children with SLI, with mental impairments, autistic or with poor hearing, because the latter never attain the level of fluent knowledge and ability to use language as a means of communication. In such cases, the term "delayed speech development" should be treated only as a tool for the description of these disorders, and not as a diagnosis.

This approach to the problem of delayed speech development is further supported by the fact that the cluster of causes that lead to seemingly identical symptoms is so rich and differentiated in its essence that the term loses its diagnostic power. Depending on the level at which speech mechanisms

are damaged, the objective and progress of speech therapy of SLI children differ, despite the common factor of delay in the development of language skills, which is why this term is also hardly useful in practice. In spite of numerous objections to the approach to this problem reported by many authors, we still lack consensus relating to the approach to this phenomenon, to the meaning of the terms, and to the indications relating to its application in the theory and practice of clinical speech therapy.

The goal of our research was to describe the constellation of individual symptoms and SLI subtypes in a sample of children diagnosed with SLI on the basis of a delay in language development exceeding 12 months in comparison to the commonly accepted norms.

MATERIAL AND METHOD

Patient characteristics

Our research involved 20 children with specific language impairment (SLI) consulted in the Reintegration and Training Center of the Foundation for

Table 1. Biographic characteristics of analysed children with specific language impairments (SLI)

No.	Age	Sex	IQ	Handedness	Swallowing difficulties*	SLI in the family	sensorimotor disorders	pseudobulbar symptoms**
1.	13	m	89	R	-	+	unchanged	-
2.	9	f	107	L	-	+	unchanged	-
3.	16	f	92	L	-	+	unchanged	-
4.	9	f	121	L	-	+	unchanged	-
5.	13	m	101	L	-	-	unchanged	-
6.	15	f	126	R	+	-	slight right-sided pariesis	+
7.	16	f	80	R	+	-	unchanged	-
8.	8	m	88	L	+	+	unchanged	-
9.	6	f	90	R	+	-	slight right-sided pariesis	+
10.	11	f	102	R	-	+	unchanged	-
11.	11	f	107	R	-	+	unchanged	-
12.	12	f	82	L	-	+	unchanged	-
13.	12	f	88	L	-	-	unchanged	-
14.	9	f	101	L	-	+	unchanged	-
15.	7	m	89	L	-	+	unchanged	-
16.	13	f	76	R	+	?	unchanged	+
17.	8	f	79	R	-	-	unchanged	-
18.	13	f	101	R	-	+	unchanged	-
19.	14	m	75	R	-	+	unchanged	-
20.	13	f	89	L	-	+	unchanged	-

* based on the parents' reports.

** based on the related research.

---

[1] *Statistical numbers further in this work will be presented in the following form: arithmetical average ± standard deviation*

Persons with Brain Dysfunctions in Cracow, Poland. The population consisted of 15 girls and 5 boys; the average age was 11.4 ± 2.95 years[1]. The boys were slightly younger (11.0 ± 3.03 years) than the girls (11.5 ± 2.95 years), but the difference was not statistically significant (p = 0.37, n.s.). The youngest girl analysed was 6, while the youngest boy was 7; the oldest patients analysed (2 girls) were 16, while the oldest boy was 13.

The non-verbal intelligence of the children was tested with the use of the WISC-R test. The average score for the entire population was 94.2 ± 14.00 (the lowest IQ was 75, the highest 126).The differences between the girls and the boys were not statistically significant in this respect (p = 0.15, n.s.).

As expected, as many as 13 of the 20 patients came from families in which at least one person (in addition to the analysed child) also had SLI symptoms; in the case of one of the remaining children, it was not possible to determine whether someone else in the family had SLI. However, it is worth noting that half of the analysed population was left-handed (including 3 boys out of 5). Only two persons in the population (both girls) also had other neurological impairments in addition to SLI (in both cases, this was a mild right paresis in right-handed children). According to the information obtained from the parents, 5 children (including 1 boy) had swallowing difficulties, while pseudo-bulbar symptoms (i.e. dysarthria with nasal speech, swallowing difficulties or, potentially, pathological laughing and/or crying) were found in 3 girls only.

Research methods

In all the analysed children, the methodology applied involved the recording and analysis of spontaneous conversations between the child and the therapist or parents in the researcher's presence, as well as simple tests and exercises. The active and passive language skills of the children were evaluated in the areas of phonology, syntax, and lexicon.

In phonology, the spontaneous speech of the children and the repeating of individual words and short sentences were evaluated. To simplify the comparison of results, phonological abilities were evaluated on the following scale:
– 100% = no disorders;
– 75% = the child distorts or omits individual phonemes;
– 50% = speech considerably slower, the child speaks with a visible difficulty;
– 25% = speech very indistinct, sometimes incomprehensible;
– 0% = no speech.

Syntactic abilities in the construction of a verbal utterance were evaluated on the basis of the analysis of sentences spoken by the child in the course of spontaneous speech. These abilities were evaluated according to the following scale:
– 100% = no disorders;
– 75% = the child uses simplified syntax in her own utterances but understands complex sentences in the utterances of other people;

– 50% = numerous agrammatisms in the utterances, difficulties in under-
standing complex sentences;
– 25% = speech is "telegraphic" (nearly all nouns in the nominative case, the
child frequently omits endings);
– 0% = no speech.

The richness of the lexical resources of the analysed children was evalu-
ated in an analogical manner according to the following scale:
– 100% = no disorders;
– 75% = limited vocabulary in own utterances, but the child understands the
majority of words used by the interlocutor;
– 50% = the child frequently cannot express herself, uses periphrases and
gestures instead of the proper word;
– 25% = numerous verbal and semantic paraphasias, neologisms, jargon (it
is difficult to understand what the child is talking about);
– 0% = no speech.

All the children were also tested with the use of an experimental Polish
version of the Peabody test, which involves presentation of a set of drawings
to the child and then asking the child to indicate one of them. The objective
of the test is to verify the child's ability to understand the meanings of words
used by other people regardless of whether the child can use that word in her
own speech or not.

The communicative competence of the child was also evaluated, i.e. the
degree to which the child is able to communicate with people in the environ-
ment regardless of the specific language impairments. These abilities were
evaluated on the basis of an interview with the parents and the clinicist's own
observations, according to the following scale:
– 100% = despite potential language deficits, the child communicates freely
and effectively, at least with her family;
– 75% = difficulties occur in communication with the child, but the majority
of problems can be solved;
– 50% = it is difficult to communicate with the child, and it is not always pos-
sible to overcome these difficulties;
– 25% = the child can communicate with the environment only in scarce,
precisely defined situations;
– 0% = no verbal contact with the child.

An appropriate diagnosis was issued for each child on the basis of the
results of the clinical speech therapy and neuropsychological evaluation car-
ried out with the use of the method of 3 competent jurors, namely:
– F = phonological disorders withour concomitant disorders in syntax and
vocabulary;
– FS = disorders in phonology and syntax with no vocabulary disorders;
– FL = disorders in phonology and vocabulary with no syntax disorders;
– FSL = disorders in the area of all language skills;
– S = syntax disorders with no phonology and vocabulary disorders;

– SL = disorders in syntax and vocabulary with no phonology disorders;
– L = vocabulary disorders with no phonology and syntax disorders.

The results of testing of CT or MRI scans of the head were also available for all the analysed children.

RESULTS

The results obtained from the clinical speech therapy tests are summarized in table 2.

The results presented in table 2 make it possible for us to conclude that:
– no phonological disorders were found in 7 children (35%), while 4 children (20%) showed deformations of individual phonemes, 8 children (40%) showed a slowing down of the fluency of speech, while the speech of 1 child (5%) was incomprehensible,
– no disorders relating to syntax were found in 8 children (40%), disorders occurred in sentences with a complex structure in 5 children (25%), numerous agrammatisms were found in 5 children (25%), while the

Table 2. Summary of results from clinical speech therapy tests of the analysed children with specific language impairments (SLI)

No.	Age	Sex	IQ	language abilities			test Peabody	communication skills	SLI type
				phonological	syntactic	lexical			
1.	13	M	89	100	75	75	90	75	SL
2.	9	K	107	75	50	100	98	75	FS
3.	16	K	92	100	75	50	76	25	SL
4.	9	K	121	75	100	75	90	100	FL
5.	13	M	101	50	50	50	78	25	FSL
6.	15	K	126	25	100	100	109	50	F
7.	16	K	80	100	75	50	77	50	SL
8.	8	M	88	50	100	75	75	75	FL
9.	6	K	90	50	100	100	88	75	F
10.	11	K	102	100	75	75	90	50	SL
11.	11	K	107	50	100	100	95	75	F
12.	12	K	82	50	25	25	90	50	FSL
13.	12	K	88	100	50	50	79	75	SL
14.	9	K	101	75	100	25	91	100	FL
15.	7	M	89	50	100	75	93	75	FL
16.	13	K	76	75	50	50	58	25	FSL
17.	8	K	79	50	25	25	61	25	FSL
18.	13	K	101	50	100	100	86	100	F
19.	14	M	75	100	50	25	65	50	SL
20.	13	K	89	100	75	50	76	75	SL

Table 3. Summary of clinical speech therapy diagnoses and results of neuroimaging tests in the analysed children with specific language impairments (SLI)

Case	SLI type	Neuroradiological results
1.	SL	Bilateral PMG* in the vicinity of the fissure of Sylvius
2.	FS	Bilateral PMG in the vicinity of the fissure of Sylvius
3.	SL	Bilateral atrophies in frontal and parietal lobes
4.	FL	Bilateral PMG in parietal lobes
5.	FSL	Bilateral PMG in the vicinity of the fissure of Sylvius
6.	F	Slight atrophies in frontal lobes
7.	SL	Bilateral atrophies in frontal and parietal lobes
8.	FL	Bilateral PMG in the vicinity of the fissure of Sylvius
9.	F	Bilateral PMG in the vicinity of the fissure of Sylvius
10.	SL	Bilateral atrophies in frontal and parietal lobes
11.	F	Bilateral PMG in parietal lobes
12.	FSL	Bilateral PMG in the vicinity of the fissure of Sylvius
13.	SL	Bilateral atrophies in frontal and parietal lobes
14.	FL	Bilateral PMG in parietal lobes
15.	FL	Bilateral PMG in parietal lobes, dilatation of the subarachnoid space in the posterior cranial cavity with underdevelopment of the vermis, suggesting Dandy-Walker syndrome
16.	FSL	Bilateral PMG in the vicinity of the fissure of Sylvius
17.	FSL	Bilateral PMG in the vicinity of the fissure of Sylvius, frontal atrophies
18.	F	No pathological changes
19.	SL	Bilateral atrophies in frontal and temporal lobes
20.	SL	Bilateral PMG in the vicinity of the fissure of Sylvius

* PMG = polymicrogyria

speech of 2 children (10%) was "telegraphic";

– no deficits in vocabulary were found in 5 of the analysed children (25%), the vocabulary used by 5 children (25%) was limited, the vocabulary was poor (periphrases and gestures) in 6 children (30%), while paraphasias, neologisms and jargon occurred in 4 children (20%),
– as for communication capabilities, no problems were found in 3 children (15%), in 8 children (40%) the communication was difficult but usually possible (partially with the use of gestures and other substitute methods of communication), considerable difficulties in interpersonal communication occurred in 5 children (25%), while in the case of 4 children (20%), their ability to communicate with the environment was limited to specific situations.

In the Polish version of the Peabody test, the average score for the entire analysed group was 83.3 with a standard deviation of 12.79, which suggests
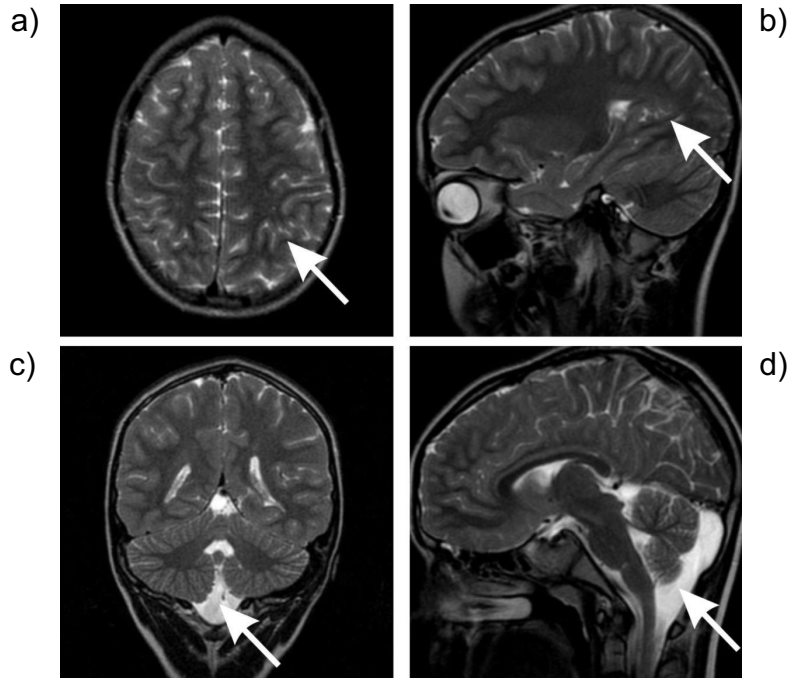
Fig. 3 MRI results for a 13-year-old-boy with SLI a) axial FSE T2, polymicrogyria in left parietal lobe; b) sagittal, FSE T2, polymicrogyria in parietal lobe; c) coronal FSE T2 Dandy Walker variant, d) sagittal FSE T2 Dandy Walker variant

the occurrence of disorders in the child's ability to understand the meanings of words used by other people.

The final analysis showed that, in the analysed population:
– 4 children (20%) showed phonological disorders only;
– 1 child (5%) showed phonological-syntactic disorders;
– 4 children (20%) showed phonological-lexical disorders;
– 4 children (20%) showed disorders in the area of all language skills;
– 7 children (35%) showed syntactic-lexical disorders.

No children with solely syntactic or solely lexical disorders were found.

No significant correlations of these results with other clinical or demographic parameters were found as a result of statistical analysis.

The results of the analysis of CT or MRI scans are presented in Table 3.

The results presented in table 3 show that all of the analysed children except for one showed pathological changes in brain tissue. The most frequent diagnosis, applying to as many as 9 children, was polymicrogyria (PMG) in the vicinity of the fissure of Sylvius, while 4 children had the polymicrogyria (PMG) in the parietal lobes. Cortical atrophies occurred in other children. It is worth observing that these changes were bilateral.

Fig. 3 shows MRI results for a 13-year-old boy with polymicrogyria. As frequently occurred, in addition to PMG there were other anomalies discovered incidentally (dilatation of the subarachnoid space in the posterior cranial cavity, as

seen in Fig. 3, with underdevelopment of the vermis suggesting Dandy-Walker syndrome). Clinically, however, the only symptoms of brain dysfunction were SLI.

DISCUSSION

The results we obtained are comparable to the research done by other authors only to a slight degree (see De Vasconcelos Hage et al. 2006), most likely because our research applied a slightly different approach to the evaluation, and the children we have analysed were slightly older. Similarly to the research done by De Vasconcelos Hage et al. (ibid.) no statistically significant correlations of the parameters determining the nature of the SLI in a given child and other clinical or demographic parameters were found in the children we analysed. It was also impossible to determine a significant connection between the clinical picture of a child with SLI and the results obtained from neuroradiological tests. However, it should be stressed here that our research was based to a great degree on information obtained from the parents. The tests were done in various development periods of the children, which make the comparison of results considerably more difficult. The unification of the execution and analysis of results from the tests could lead to more interesting conclusions.

However, the importance of what we did not find in the children we have analysed is worth stressing.

– *firstly*, there were no cases of pathological changes occurring only in one hemisphere of the brain. Changes in both brain hemispheres were found in nearly all the children; however, these changes existed in the form of polymicrogyria (PMG) in the vicinity of the fissure of Sylvius in nearly half of the patients, as in the case of studies carried out by other authors (Hage et al. 2006). Is this because a child with pathological changes in only one hemisphere develops her language skills in the other hemisphere? It is difficult to answer this question in an unambiguous manner, because we usually do not carry out neuroimaging tests for people in whom no clinical disorders are found. The tests that have been carried out incline us to the hypothesis that SLI symptoms occur in a child only if the opposite hemisphere is unable to replace the functions of the damaged hemisphere.

– *secondly*, we did not find a single case of solely syntactic or solely lexical disorders, while solely phonological disorders were found in 4 children, i.e. 20% of the analysed population. Can we assume that, if a brain has separate processors executing syntactic and lexical functions, these processors cannot be damaged with no influence on other language processes? However, if the brain operates in a holistic manner and executes all functions at the same time, why are only phonological functions impaired in some children? After all, adult patients with aphasia can have Broca's or Wernicke's aphasia without dysarthria. Why should we not have equivalents of such disorders in children?

– *thirdly,* no correlation was found between the location of changes found in

the brain (i.e. the lobe in which changes were found) and the nature of the speech impairments in the child. This raises a similar question: If language functions can be mapped in the brain, e.g. in the style of the famous "house" of Wernicke and Lichtheim (Lichtheim 1885), why are there no explicit location regularities in these children? In addition, if location is not a significant factor, why did changes exist in the vicinity of the fissure of Sylvius and not somewhere else in the brain in as many as 9 children? Neither the former nor the latter hypothesis can explain the results we have obtained.

Of course, the full discussion of the problems raised here is impossible in a single article. Moreover, the studies should be repeated for a larger population, considering more parameters relating to the evaluation of the children's language abilities and including unified neuroimaging study methods. However, our results can suggest some hypotheses, which can be verified in the course of further studies.

Searching for a possible explanation of the observed symptoms in people after brain injuries according to the principles of microgenetic theory (Brown 1979, 2005, Brown and Pąchalska 2003), we should think not only about *where* the damage exists but rather *when*, i.e. at what stage of the formation of the utterance . This is because the nature of a symptom depends on which segment of the normal process is subjected to disorganization or slowing down, which, in turn, depends on location only in part. For this reason, we cannot ignore the location of changes and abnormalities on the one hand, and, on the other hand, we can never be one hundred percent sure of the clinical picture of a given patient on the basis of the location only. In the microgenesis of utterances, subsequent neural activation waves following one another every ca. 30 milliseconds lead contents from deeper, philogenetically older layers of the nervous system to the surface and, at the same time, sculpt that shapeless mass of unconscious contents to form utterances built in line with the rules of the used language. The higher (i.e. later) a disorder occurs in the course of that process, the more the damage affects specific and detailed functions, which makes the impairments occurring, e.g. after injury to the cerebral cortex, more specific and clear than impairments occurring, e.g. after damage to subcortical structures.

It is not exactly known how, when and why highly specialized groups of neurons executing strictly defined tasks relating to the process of verbal expression are created in the brain, usually in the left hemisphere in the vicinity of the fissure of Sylvius. We are not born with the Broca's or Wernicke's area defined in advance in a structural sense. These areas becomes specialized in the course of the acquisition of knowledge of the language. According to microgenetic theory, ontogenesis, phylogenesis and microgenesis are one and the same process all the time, but executed within various time scales. Modifications taking place in the course of the evolution of the human brain are repeated in the development of the brain of each one of us

from birth to death, and the same changes, the same transfers from an older processing phase to a newer one, are also executed in the microgenesis of a given state of the mind. In other words: ontogenesis creates solutions that microgenesis uses when the functions are transformed into structure as one learns. This is what the plasticity of the brain involves, and, as we know, it is highest in a child and decreases gradually with the passing years (Pąchalska 2007).

In the light of the facts presented above, a more complete understanding of SLI symptoms in children can be obtained not thanks to the question: *where in the brain do changes exist*, but rather *when in the brain and speech-modelling process does the "derailment" of that process took place?* We can assume that the process of verbal expression is a certain reflection of the process of learning of speech and language, which, in turn, reflects the brain's development process. With further development, dynamic solutions repeated many times become structural solutions, even though the move-ment from function to structure is always rather liquid and flexible. This is why the traditional approach assuming the reverse reaction between structure and function in the brain cannot explain the phenomena observed in the clin-ic in children with SLI, or in adults with aphasia, in a satisfactory and consis-tent manner.

CONCLUSIONS

1. Specific language impairments (SLI) occur when language abilities in a child develop with at least one year's delay in comparison to the major-ity of children at the same age.
2. SLI differs from ordinary speech development delay in the fact that disor-ders in a child with SLI do not disappear spontaneously with time.
3. SLI differs from the general impairment of mental development in that other cognitive functions except for speech and language progress normally in a child with SLI.
4. In the population we tested, neuroanatomical irregularities were found in all children with SLI except for one, but the results obtained do not make it possible for us to indicate a relationship between the nature and location of these changes and the clinical image of the analyzed child in terms of language and speech.
5. The arrangement of individual language abilities and disorders existing in children with SLI varies depending on age, and on other factors not exact-ly known and analysed.
6. The disorders existing in children with SLI can be explained with the use of microgenetic theory.

REFERENCES

American Psychiatric Association (2000). Diagnostic and Statistical Manual of Mental

Disorders (4th ed., text revision). Washington, DC: Authors.

Barkovich, A.J. (1996). Congenital malformations of the brain and skull. In: A.J. Barkovich (ed.), Pediatric neuroimaging, II ed. (177-275). New York: Lippincott-Raven.

Barlett, C.W., Flax, J.F., Logue, M.W., Vieland, V.J., Bassett, A.S., Tallal, P., & Brustowicz, L.M. (2002). A major susceptibility locus for specific language impairment is located on 13q21. American Journal of Human Genetics, 71, 45-55.

Bianchi, L. (1922). The mechanism of the brain and the function of the frontal lobes. Edinburgh: Livingstone

Bishop, D.V. (1992). The underlying nature of specific language impairment. Journal of Child Psychology and Psychiatry, 33(1), 3-66.

Bishop, D.V. (1997). Uncommon understanding: development and disorders of language comprehension in children. Cambridge: Psychology Press.

Bishop, D.V. & Snowling, M. J. (2004). Developmental dyslexia and specific language impairment: same or different? Psychological Bulletin, 130(6), 858-886.

Bishop, D.V., Hayiou-Thomas, M.E. (2007). Heritability of specific language impairment depends on diagnostic criteria. Genes, Brain and Behavior.5, e-article.

Bishop, D. V., Laws, G., Adams, C. V. Norbury, C. F. (2006). High heritability of speech and language impairments in 6-year-old twins demonstrated using parent and teacher report. Behavior Genetics, 36, 173-184.

Brown, J.W. (1979). Language representation in the brain. W: H. Steklis, M. Raleigh (red.), Neurobiology of social communication in primates. New York-San Francisco-London: Academic Press.

Brown, J.W. (2001). Microgenetic theory: reflections and prospects. Neuropsychoanalysis, 3, 61-74.

Brown, J.W. (2005). Process and the authentic life: Toward a psychology of value. Heisenstamm, Germany: Ontos Verlag.

Brown, J.W. & Pąchalska, M. (2003). The nature of the symptom and its relevance for neuropsychology. Acta Neuropsychologica, 1, 1-11.

De Vasconcelos Hage, S.R., Cendes, F., Montenegro, M.A., Abramides, D.V., Guimaraes, C.A. & Guerreiro, M.M. (2006). Specific language impairment: linguistic and neurobiological aspects. Arquivos de Neuro-Psiquiatria, 64(2A), 173-80.

Dilling-Ostrowska, E. (1990). Zaburzenia mowy. In: J. Czochańska (ed.), Neurologia dziecięca, ed. II. Warsaw: PZWL.

Duvelleroy-Hommet, C., Gillet, P., Billard, C., Loised, M., Barthe, Z. & Santini J. (1995). Study of unilateral hemisphere performance in children with developmental dysphasia. Neuropsychologia, 33, 823-834.

Flax, J.F., Realne-Bonilla, T., Hirsch, L.S., Brustowicz, L.M., Bartlett, C.W. & Tallal, P. (2003). Specific language impairment in families: Evidence for co-occurrence with reading problems. Journal of Speech, Language, and Hearing Research, 46, 530-543.

Galaburda, A.M., Sherman, G.F., Rosen, G.D., Aboitiz, F. & Geschwind, N. (1985). Developmental dyslexia: four consecutive patients with cortical anomalies. Annals of Neurology, 18, 222-233.

Grabias, S., ed. (2001). Zaburzenia mowy. Mowa - teoria - praktyka. Lublin: Wydawnictwo UMCS.

Guerreiro, M.M., Andermann, E., Guerrini, R. et al. (2000). Familial perisylvian polymicrogyria: a new familial syndrome of cortical maldevelopment. Annals of Neurology, 48, 39-48.

Hall, D.M., Hill, P.D. (1996). Communication disorders. In: D.M. Hall & P.D. Hill (ed.), The child with a disability (173-209). London: Blackwell.

Jastrzębowska, G. (2003). Zakłócenia i zaburzenia rozwoju mowy. In: T. Gałkowski & G. Jastrzębowska (ed.), Logopedia – pytania i odpowiedzi. Podręcznik akademicki (9-66). Opole: Wydawnictwo Uniwersytetu Opolskiego.

Jastrzębowska, G. (2005) Opóźnienie rozwoju mowy. In: T. Gałkowski, E. Szeląg & G. Jastrzębowska (ed.), Podstawy neurologopedii. Podręcznik akademicki (360-378). Opole: Wydawnictwo Uniwersytetu Opolskiego.

Kaczmarek, B.L.J. (1995). Mózgowa organizacja mowy. Lublin: Agencja Wydawniczo-

Handlowa AD.

Kordyl, Z. (1968). Psychologiczne problemy afazji dziecięcej. Warsaw: PWN.

Leonard, L.B. (2000). Children with specific language impairment. Massachusetts: MIT Press.

Leonard, L.B. (2006). SLI - Specyficzne zaburzenie rozwoju językowego. Gdańsk: Gdańskie Wydawnictwo Psychologiczne.

Lichtheim, L. (1885). On aphasia. Brain, 7, 435-436.

Lum, J.A. & Bavin, E.L. (2007). Analysis and control in children with SLI. Journal of Speech, Language, and Hearing Research, 50(6), 1618-30.

Merricks, M.J., Stott, C.M., Goodyer, I.M. & Bolton, P.F. (2004). The aetiology of specific language impairment: no evidence of a role for obstetric complications. Journal of Neural Transmission, 111(7), 773-789.

Norrix, L.W., Plante, E., Vance, R. & Boliek, C.A. (2007). Auditory-visual integration for speech by children with and without specific language impairment. Journal of Speech, Language, and Hearing Research, 50(6), 1639-51.

Pąchalska M. (1993). Zaburzenia komunikacji u chorych z afazją. In: Opuscula logopaedica in honorem Leonis Kaczmarek (265-283). Lublin: UMCS.

Pąchalska, M. (1999). Afazjologia. Warsaw & Cracow: Wydawnictwo Naukowe PWN.

Pąchalska, M. (2003). Diagnoza chorego z afazją. In: T. Gałkowski & G. Jastrzębowska (red.), Logopedia. Pytania i odpowiedzi. Podręcznik akademicki (611-699). Ed. II. Vol. 2: Zaburzenia komunikacji językowej u dzieci i osób dorosłych. Opole: Wydawnictwo Uniwersytetu Opolskiego.

Pąchalska, M. (2007). Neuropsychologia kliniczna: Urazy mózgu. Warsaw: Wydawnictwo Naukowe PWN.

Parol, U. Z. (1989). Dziecko z niedokształceniem mowy. Warsaw: WSiP.

Plante, E. (1991). MRI findings in the parents and siblings of specific language impairment boys. Brain & Language, 41, 67-80.

Pruszewicz, A. (1992). Opóźniony rozwój mowy. In: A. Pruszewicz (ed.), Foniatria kliniczna. Warsaw: PZWL.

Rapin, I. (1996). Developmental language disorders: A clinical update. Journal of Child Psychology and Psychiatry, 6, 643-655.

Rapin, I., Allen, D.A. & Dunn, M.A. (1992). Developmental language disorders. In: S.J. Segalowitz & I. Rapin (ed.), Handbook of neuropsychology (111-137). New York: Elsevier.

Riccio, C.A., Cash, D.L. & Cohen, M.J. (2007). Learning and memory performance of children with specific language impairment (SLI). Applied Neuropsychology, 14(4), 255-61.

Robertson, S.B., Weismer, S.E. (1999). Effects of treatment on linguistic and social skills in toddlers with delayed language development. Journal of Speech, Language, and Hearing Research, 42, 1234-1248.

Spionek, H. (1981). Zaburzenia psychoruchowego rozwoju uczniów a niepowodzenia szkolne. Warsaw: PWN.

Tager-Flusberg, H. & Cooper, J. (1999). Present and future possibilities for defining a phenotype for specific language impairment. Journal of Speech, Language, and Hearing Research, 42, 1275-1278.

Taylor-Sarno, M. (1981). Recovery and rehabilitation in aphasia. In: M. Taylor-Sarno (ed.), Acquired aphasia.(485-522).New York: Academic Press.

World Health Organization (1997). The ICD-10 Classification of Mental and Behavioral Disorders: Diagnostic Criteria for Research. Geneva: Authors.

Zaleski, T. (1992). Opóźniony rozwój mowy. Warsaw: Państwowy Zakład Wydawnictw Lekarskich.

Correspondence address:

Prof. Maria Pąchalska, Ph.D.

Institute of Psychology, Gdansk University,ul. Pomorska 68, 80-343 Gdańsk

m.pachalska@medscimonit.com