

SCHOOL DISADAPTATION IN CHILDREN WITH SPEECH UNDERDEVELOPMENT AND ITS PREVENTION

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350 7 to 14-year-old students of a special school for children with severe speech and language disorders (SLD) were tested through psychological, neurolinguistic and clinical methods. It was a longitudinal study. During 4-5 years of studying at the special school, the majority of speech and language impairments reduce and communicative skills grow up. But the other students demonstrate regress of adaptation skills, intellectual efficiency and increase in restlessness, aggressive behavior, mood disorders, and neurotic disorders. We recognize it as the state of exacerbation. Comparative statistic analysis reveals that this state occurs mostly between 9.5 and 11.5 years of life. 50% of the decompensation cases observed occurs in this age range. The data obtained gave us an incentive to develop a prevention program. The target group included a population aged 9.5-10 with PSU (PPMI-program). The 2-year experience of incorporating the risk subgroup of SLIC into the PPMI-program gave a positive return. The amount of decompensations reduced significantly. Intensity of psychopatological disorders decreased.

Keywords: speech, language, disorders, exacerbation, prevention.

Many researchers point to high co-occurrence of speech underdevelopment and behavior disorders. Speech and language disturbed children are at the increased risk of developing psychiatric disorders. According to Horwitz et al. (2003), parents report that 30 month-old subjects with expressive language and speech disorders are four times more likely to have externalizing behavior problems in comparison with the population. In an epidemiologic study of 705 3-year-old children in a London suburb, Stevenson & Richman (1978) found that 59% of children with delayed language and speech had behavioral problems as well, whereas such disorders were typical of 14% of the total population.

Jenkins, Bax, and Hart (1980) conducted a longitudinal study of behavior problems in 418 preschool children of London that included studying them at the age of between 6 weeks and 4.5 years. A reliable connection between delayed speech-language and behavior problems was established. Children in the group with atypical language development manifested behavior problems twice as often in comparison with the general population. Stevenson, Richman, and Graham (1985) examined 3-year old children, re-examined them when they turned 8 years old and found that behavior problems persisted at the age of 8, too.

Beitchman et al. (1996) noted that psychiatric disorders in 12.5 year-old individuals who had originally been diagnosed with speech and language disorders at the age of 5 were more likely to occur in individuals with language underdevelopment than in those with speech disorders, even if such disorders were partially compensated. Severe speech-language impairment found at the age of 5 are accompanied with continuous and increasing risk of deficiency in language skills and poor academic progress at the age of 12.5, with symptoms of language and speech underdevelopment being retained in 72% of 12.5 year-old children. On the contrary, Redmond and Rice (2002) report reduction in behavioral disorders in such children after they have reached the age of 6–8.

Clinical observations and statistics demonstrate that these disorders are compensated not in all children with language and speech underdevelopment in preschool years. In school years, about 3–4% of children with language and speech underdevelopment continue to lag behind. Most of them develop such problems as dyslexia, dysgraphia, and different behavior deviations. Many authors discuss a causal relationship between severity and persistence of language-speech impairments, on the one hand, and contingency of psychiatric disorder development, on the other hand (Lindsay, Dockrell, & Strand, 2007).

Baker and Cantwell (1987) reported an increase in psychiatric disorders from 46% to 60% over an observation period of five years (the initial age of sample was 5.7 years, the catamnesis age was 9.1 years). One fourth of children developed psychiatric disorders prior to the reexamination, although they had been diagnosed psychiatrically well at first.

The data given above support our own experience. In previous publications, we reported about attention deficit, aggressive behavior, as well as over anxiety and excessive shyness in children with primary speech

underdevelopment (PSU) (Kornev, 2003). Obviously, high prevalence of these disorders is typical of severe and persistent PSU cases. In this connection, it would be reasonable to discuss how secondary behavioral complications in children with PSU can be prevented.

It was the main objective of this study to explore behavioral abnormalities, emotional and social difficulties at school in a sample of PSU-affected children aged 7–14 and to create a program of preventing the same. The study consisted of two parts: the 1st related to clinical, psychological and psychopathological follow-up study of schoolchildren having PSU. Moreover, the psychopathological phenomena dynamics for the age range of 7–14 years was analyzed. The 2nd part of the study aimed at elaborating and testing a prevention program, and assessing its efficiency.

Experiment 1

Methods

350 students from a special remedial school for children with severe speech underdevelopment took part in a longitudinal study. All students were 1st–3rd graders (the mean age is 8 years) in the initial step of the study. In the end of this study, they were 5th–7th graders (the mean age is 12.8 years).

All of them were diagnosed as having PSU after a clinical and psychological examination. The children were included in the experimental group on the grounds of the following criteria: presence of expressive speech delay and impressive speech safety. The experimental assessment of syntactic abilities by means of a Sentence Repetition test (Kornev, 2003) revealed that the average syntactic development level of these children corresponds to the age of 4 to 5. By the time the children started to attend school, no explicit psychopathologic symptoms had been found in all the children. Nonverbal IQ below 80 measured by WISC and hearing loss was employed as the exclusion criteria. Children having PSU were examined through a complex of psychological, neurolinguistic and psychometric methods¹. Psychopathological and selective psychological evaluation repeated annually.

¹ Detailed description of the procedure and methods of this study can be found in our earlier publications (Kornev, 1997, 2003, 2006).

Results

Clinical study of 7–9 year-old children with PSU revealed that 90% of them had a number of psychopathological syndromes, emotional immaturity, attention deficit, and easy fatigability. No correlations between a nature of psychopathological symptoms and a structure of speech disorders were found. For example, only attention deficit and easy fatigability was observed in some severe PSU cases. Several less severe cases manifested pronounced emotional and behavioral disorders. On the other hand, an integrated psychological study revealed an important connection between severity of speech underdevelopment and frequency of multiple mild cognitive deficits of successive functions, working memory, selective attention, short-term verbal memory and verbal-logic reasoning.

The follow-up study revealed that the developmental paths of children with PSU vary considerably in different children. During 4–5 years at the remedial school, the majority of speech and language impairments compensated in full or in part, and communicative skills grew up noticeably. 1.5% of the sample had only subtle advance, though. Re-examinations demonstrated that several psychological indices varied considerably within the entire period of study. A period of sustainable improvement in children having PSU sometimes changed into decompensation conditions. In this case children's adaptation skills decreased unexpectedly. The cognitive skills deficiency grew more severe, fatigability scaled up, behavior became aggressive, emotional and neurotic disorders took place. We recognize this condition as mental decompensation. We found 101 cases of such decompensations within the entire follow-up study. One of the questions we tried to answer was at what age such decompensations occur more often.

A comparative statistic analysis of decompensation frequency in different ages was conducted. The analysis of decompensation occurrence age revealed 3 critical age ranges when decompensation conditions occur the most frequently: 6–7 years, 9.5–11 years, and 14–15 years. These age ranges coincide with the known crisis periods in development of children. Mental processes may worsen in these rather unstable periods of development (Vygotsky, 1984). In some children having PSU, the above-discussed processes look like psychological deadadaptation manifesting itself in several psychopathological symptoms. The statistical analysis revealed reliably that such conditions were most often observed between

9.5 and 11.5 years. As much as 50% of all decompensations occurred in this age (Table 1). Consequently, children with PSU are at the highest risk of decompensation at the age of between 9.5 to 11.5.

Table 1

Age distribution of 101 decompensation cases in a group of 350 children with PSU

Age range	Cases of decompensation	
	N	%
7–8 years	21	21
9.5–11.5 years	50	50
12–12.9 years	12	12
13–15 years	18	18
Total	101	100

Table 2

Decompensation occurrence in different age ranges

Age range	Children with PSU	Children with PSU that had decompensation	%
A) 7–8 years	65	11	17
B) 9.5–11.5 years	110	30	27*
C) 12–12.9 years	60	5	09
D) 13–15 years	115	16	14

* Adequacy of differences (t-criterion): $p < .001$ (B–D) and $p < .05$ (A–B)

The cross-sectional method was used in the majority of researches devoted to comorbidity in subjects having PSU (Baker & Cantwell, 1987; Beitchman et al., 1996; Baltaxe & Simmons, 1988; Horwitz et al., 2003). Changes in psychological and psychopathological indices over time were not assessed even in longitudinal studies. Such information is crucial for planning prevention and remedial treatment programs. We conducted a corresponding research in this connection.

Duration of decompensation conditions varied widely in children having PSU. Some decompensations lasted for 6 months, others lasted for up to 2 years. In mild cases, decompensation revealed itself in increase of fatigability and intensification of attention deficit symptoms. The be-

ginning of more severe cases was similar. But several months later these initial symptoms were accompanied by affective disorders and divergent behavior phenomena. As far as the most prolonged decompensations are concerned, several successive stages could be singled out (Table 3).

Table 3

Psychological characteristics of decompensation stages

Decompensation stages	Psychological manifestations
Stage I cognitive resource deficit	Easy fatigability, attention deficit, high distractibility
Stage II Affective disorders	Increased irritability, temper tantrums, increased anxiety and shyness, neurotic depressive disorders;
Stage III Divergent behavior	Aggressive behavior, incomppliance, obstinacy, avoidance, protest and oppositional behavior, escapes;
Stage IV Psychotic disorders	Consciousness disorder syndromes, affective psychoses

A group of children (101 individuals) suffered from decompensation was divided into 4 sub-groups on the grounds of the comparative analysis of psychopathological patterns. Subgroup 1 (30%) included those who had symptoms typical of only the 1st decompensation stage during the entire observation period: easy fatigability, attention deficit, high distractibility. Subgroup 2 (32%) included the children in which an integrated symptomatology relative both to the 1st and the 2nd decompensation stages was observed, specifically: irritability, temper tantrums, high anxiety, shyness and neurotic depressive disorders in combination with the symptoms of the 1st stage. Subgroup 3 (35%) included children having aggressive behavior, manifestations of obstinacy, avoidance, protest and oppositional behavior, along with escapes (which correlates to the 3rd stage). Subgroup 4 (3%) was composed of those who had psychotic symptomatology.

In connection with the observations described above, it would be reasonable to put two questions: why is the risk of decompensation is particularly high within this very age range (9.5–11.5 years)? What factors do produce modulating influence on specific features of decompensation symptoms? We are still unable to give an exact answer to the first question. However, it is worth noting that the age range of between 10

and 11 years is critical for brain α -activity maturation (Kovaleva, 1990). This age range may be a critical period in terms of ontogenesis, yet it is significantly less studied than pubertal and preschool periods.

Finding an answer to the second question requires considering both biological and psychological determinants. The data referring to importance of speech disorder severity were cited above (Baltaxe, Simmons, 1988). Our findings revealed that 85% of children with PSU have symptoms of minimal brain injury occurred in the perinatal period (Kornev, 2003). On the other hand, a child is severely affected by frustrative environmental factors. One of them is poor academic progress at school. When decompensation develops in a child, the latter cannot understand why his/her academic progress is becoming poorer. Frequently it is followed by numerous reproofs from teachers and parents that are unpredictable for a child. These conditions emerge for reasons unknown for teachers and parents, while obvious, external causes seem to be absent. Usually they have no reasonable explanation for such sudden deterioration of learning abilities of a child. Sometimes they impute it to laziness, dullness or negligence. As a consequence, a child often gets punished. Children are frustrated by unexplainable changes, lack of understanding and support from grown-ups. It induces the feeling of confusion, weariness, or makes them irritable. Both biological and psychological factors combined in the majority of the cases under investigation.

Experiment 2

The data obtained in the first part of our study gave us the opportunity to determine an age range at which main decompensation prevention efforts should be aimed. For this plan to be carried out, intervention should precede the occurrence of decompensation symptoms. The main goal of 2nd part of our study was to verify our hypothesis that relevant preventive actions can prevent decompensation from occurring or reduce severity of its manifestations.

We worked out a program the objective of which was to prevent decompensation or relieve the same. This program included 3 main components: a) psychological support, b) pedagogical support and c) medical support (PPM-program).

110 children at the age of 9.5–11 suffering from PSU were selected as a focus group. The grounds for this decision were the data on the maxi-

mum decompensation risk age obtained in the first part of the experiment. Clinical evidence of PSU and appropriate age were the inclusion criteria. All these children took part in PPM-program.

All participants were divided in 2 subgroups: a) children who had no symptoms of decompensation (82 individuals), and b) children who had initial manifestations of decompensation (28 individuals). Therefore, the main task concerning the 1st subgroup was to prevent decompensation, while the task associated with the 2nd subgroup involved remedial support to lessen decompensation severity and prevent its further development. The control group was comprised of 100 children of the same age having PSU that did not participate in the PPM program and were studying under routine academic conditions in the same remedial school.

Psychological support included psychotherapeutic consulting targeted to difficulties associated with poor academic progress, low self-esteem, frustration, problems in relations with peers. A wide range of techniques was applied: art therapy (creative block design activity, drawing), play therapy, and other creative activities. The therapeutic aim was to minimize acuity of unsuccessfulness experience, to raise self-esteem, to maximize cognitive resources for problem-solving, to get a social support, to encourage children in extra-curricular athletic or other activities they are interested in, and to help them to make decisions relative to their further education. We helped them to cope with multiple difficulties they faced. The latter was especially important, because many children had a sense of guilt.

Pedagogical part of the PPM-program included the following (in subgroup b)): providing the decompensation-affected children with extra rest as a short 1–2-week holiday, logopedist consultancy. The children from subgroup a) were under more fixed observation as to learning efficiency, occurrence of new difficulties or increase in fatigability. As soon as these risk features were found, the children were provided with the same support as the ones from subgroup b) described above. The children were offered medicamentous support if there were relevant indications. All specialists who took part in the PPM-program were cooperating with each other. The PPM program lasted for 2 years. Yet, the duration of prevention/remedial actions for each child was 2–4 months.

The 2-year experience of running the PPM-program for children having PSU allowed obtaining positive results (Table 4). The whole amount of decompensations reduced. It is especially true for the most severe manifestations. Severity of all psychopathological symptoms decreased. The PPM-program prevented development of more severe decompensation forms in all cases.

Table 4

Incidence of exacerbation conditions among 9.5–11.5 year-old children with PSU from the control and the experimental groups

	Control group n=100		Experimental group n=110	
	Children with decompensation	%	Children with decompensation	%
Stage I	8	8	11	10
Stage II	12	12	3	3
Stage III	6	6	2	2
Stage IV	2	2	0	-
Total	28	28	16	15

Summing up the data obtained in second experiment, we arrived at the conclusion that the so called “comorbidity symptoms” in children with primary speech underdevelopment can be prevented or reduced to less severe manifestations. Fruitful experience of running the PPM-program gives certain reasons for such an optimistic statement.

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