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ASSESSMENT AND TREATMENT OF DYSLEXIA

Evelin Witruk

This article will overview the current state-of-the-art of the different methods of assessment and treatment of dyslexia. On the basis of the modification and extension of the multilevel model of Valtin (1989, modified by Witruk, 1993b) the methods were discussed regarding their main aims. Assessment and treatment methods were described regarding the primary causes (biological risk factors), secondary causes (partial performance deficits), primary symptoms (reading and writing failures) and regarding the secondary symptoms (emotional and behavioural disorders).

Key words: Multilevel model of dyslexia; magnocellular deficit; partial performance deficit; working memory; complex training programme.

1. Definitions

The history of the dyslexia research is controversial and led to many contradictory theories and results up to the present day. It is now just over 110 years since Morgan (1896) first published his famous account of Percy, a dyslexic boy of 14 years. The state-of-the-art of dyslexia research can be characterised by the distinction of scientists in groups of protagonists of a visual versus a phonological/auditory deficit on the one hand and in groups of protagonists of a low, basic level versus a higher level deficit on the other hand. A lot of opposite results and theories lead to the question about the specificity and homogeneousity of different deficits in dyslexic individuals. The model of Habib (2000) gives an integration of perceptive and cognitive deficits on the basis of a common temporal processing deficit, which can be analysed on a low, basic level and/or on a higher complex level of performance. The individual combination of partial deficits on the low and the higher level produces the specificity of the symptoms.

Dyslexia is defined by the World Health Organisation (WHO) as a restricted developmental disorder in the acquisition of reading often connected with a disorder in acquisition of writing. These disorders are usually contrasted by the better, normal or over-averaged intelligence. During the last century hundreds of scientists searched the specific sources of these disabilities. A lot of opposite results lead to the question about the dyslexia specificity and subtype specificity of deficits. The different prevalence rates of dyslexia in the world varied from 1 % in Scandinavian countries, 2 % around the region of Beijing, 3-5% in Germany, 8-10 % in UK and USA lead to the question of the cultural/language impact on the development of dyslexia. The relation of boys to girls is about 4:1.

2. The multilevel model of dyslexia

The multilevel model of dyslexia calls for two causal and two symptomatic levels which are

Evelin Witruk — Prof. Dr. University of Leipzig, Faculty of Biosciences, Pharmacy and Psychology, Institute of Psychology II; Seeburg str. 14-20, 04103 Leipzig, Germany; e-mail: witruk@rz.uni-leipzig.de.

superimposed in time, with one flowing from and having repercussions on the others (cf. Table 1).

On the level of primary causes, it is assumed that there exist biological risk factors interacting with environmental stressors and believed to express themselves in functional and structural neuro-anatomical characteristics of dyslexic individuals. The intervention on this level can aim at the compensation or at the restoration of these biological risk factors.

On the one hand, the secondary causes that grow out of the primary ones refer to partial performance deficits in the fields of visual and auditory perception, motor patterns and long-term and working memory. Here, the treatment involves a functional training, which is highly selected in the main imported deficit function and assumes a generalisation and stabilisation of the complex action system of reading and writing. In principle, the cause levels can be identified even before such children start formal education.

Table 1. Multilevel model (developed by Valtin, 1989, modified by Witruk, 1993b)

		Assessment	Treatment
Causes	***Primary***	Biological risk factors	Compensatory training
	Secondary	Partial performance deficit	Training of basic functions
Symptoms	***Primary***	Reading and writing tests	Rehabilitative exercises of reading and writing
	Secondary	Personality questionnaire, observation	Complex training, Psychotherapy

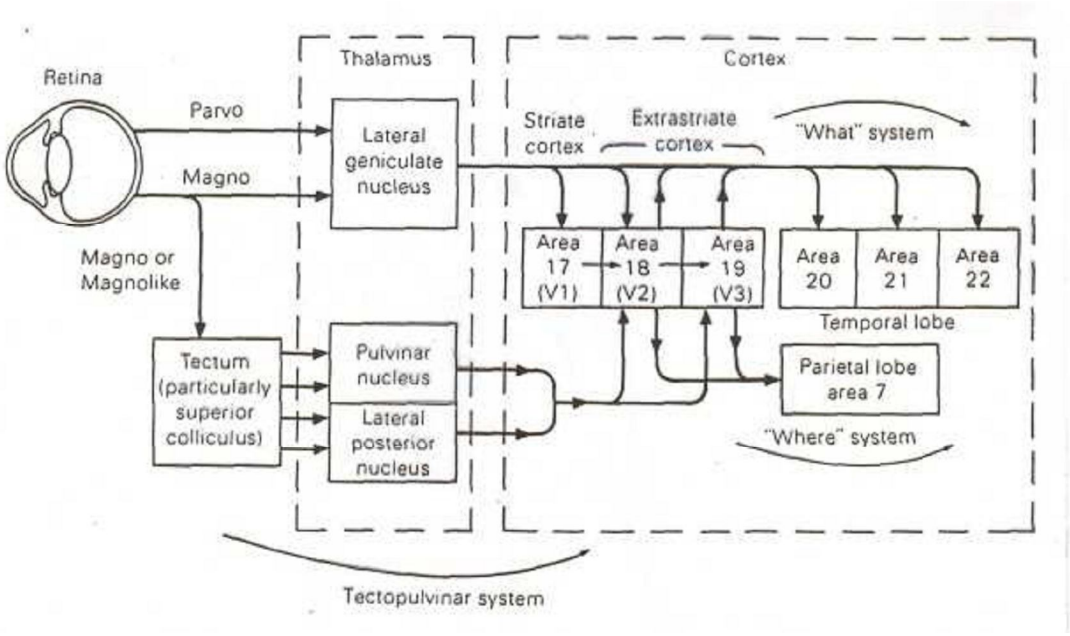

Fig. 1. Schematic figure of the visual system

On the other hand, primary symptoms can be detected only in the specificity of failures in reading and orthography, for example on the basis of error, time and eye movement analyses. In time, these latent failures and the responses from these children`s environment lead to a vicious circle of secondary symptoms made up of the four stages of anxiety, blocking, avoidance, compensation and lowering of motivation, as described by Betz and Breuninger (1982). These effects underscore the existential observational relevance of written language and the consequences of its impairments. Secondary symptoms may have repercussions on primary symptoms and on such causes as destabilisation and blocking, though there has hardly been any research on this yet.

Thus, the psychopathology of dyslexia provides some clues about possible working memory deficits and the way they must be integrated into a person`s overall pattern of

disability. Let us now examine, with the help of a demand-related information processing approach, the relevancy of working memory to the regulation of our behaviour.

3. Assessment and treatment regarding primary causes

The genetic basis of dyslexia was discussed by the familial clustering and the concordance between parents and children (44 % in Schulte-Koerne et al., 1998) and between monozygotic twins (90 %) in comparison to dizygotic twins (32 %). The primary reasons of dyslexia seem to be represented in genetic anomalies on the chromosomes 6 and 15 (Wilcke & Boltze, 2010).

The most spectacular evidence on the international research area has been presented by Galaburda and Livingstone (1993). They compared brain sections of 5 dead dyslexic adults with those of 7 healthy individuals of comparable age and occupations. When they compared these brains of each group, they detected disorganised magnocellular layers in the dyslexic geniculate laterale with smaller cells which seemed highly variable in size and shape. By contrast, they found no peculiarities in the parvocellular layers, which are mainly responsible for colour perception, while the magnocellular layers are responsible for contrast perception and hence the recognition of script (see Figure 1, 2a and 2b).

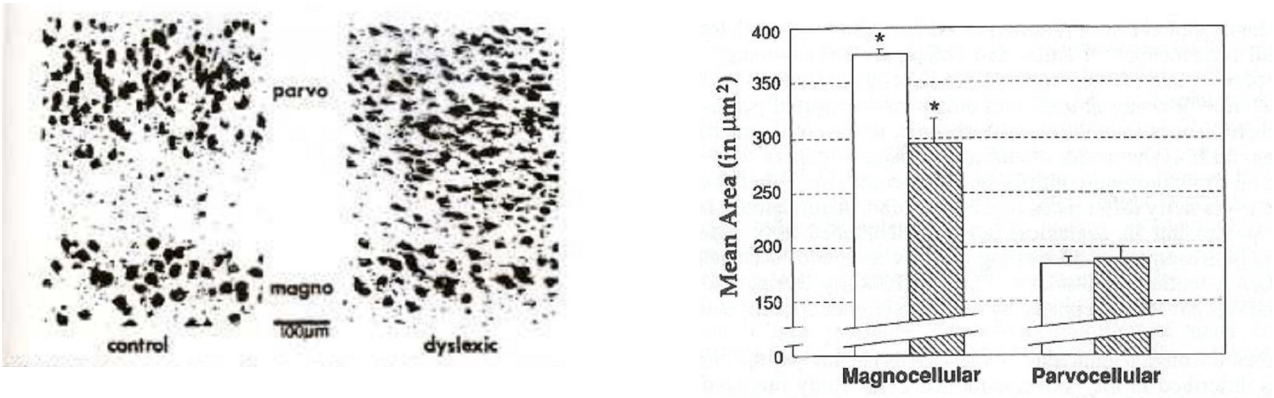

Fig. 2a and 2b. Significant smaller magnocells in dyslexic adults but the same size of parvocells (Galaburda & Livingstone, 1993)

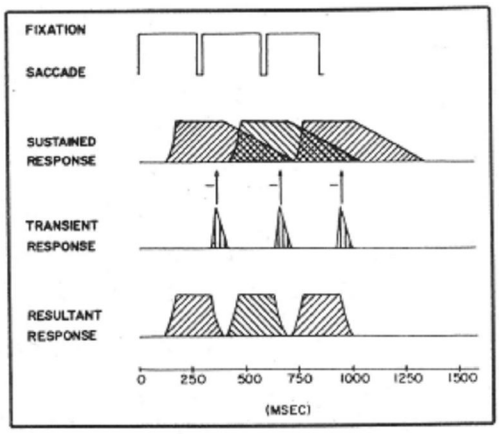

Fig. 3a

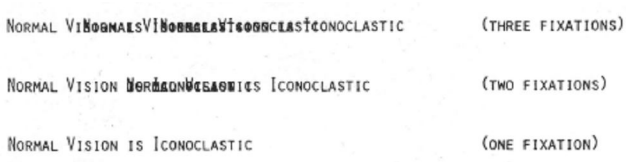

Fig. 3b

Fig. 3a. The Breitmeyer effect — interaction effect between the parvo- and magno-system in the visual system [Note: Sustained Response = Parvo System (P); Transient Response = Magno System (M)].

Figure 3b. The overlapping/smearing effect as a result of the Breitmeyer effect.

Details concerning the Breitmeyer effect are shown in Figure 3a and 3b.

Regarding the magnocellular deficit hypothesis of dyslexia positive and negative evidence was presented. Our research included the interaction hypotheses of the magno- and the parvocellular systems measured by the visible persistence duration.

Visible persistence is one of the effects of the early or low level visual processing. Impairments of the early visual functions in dyslexics have been found with a variety of tasks, in particular with tasks involving dynamic stimuli like flicker fusion tasks and coherent, apparent and real motion perception tasks. Lower temporal and spatial contrast sensitivity and longer duration of visible persistence as deficits in dyslexics could be shown, which are linked to abnormalities in the magnocellular pathway. This weakness in the transient system was investigated in its direct impact on the temporal processing of information by Galaburda and Livingstone (1993), Stein and Walsh (1997), Eden (1996). For example Chase and Jenner (1993) found only deficits in contrast fusion tasks, but not in colour fusion tasks in dyslexics. Another group of authors explain the indirect impact on the impairment of the interactions between the magnocellular, parvocellular and the koniocellular pathways, which are investigated only for the both first by Breitmeyer and Ritter (1986) and Slaghuis et al. (1996). A new approach to the magnocellular deficit is linked to the concept of attention spotlight. Vidyasagar and Pammer (1999) proposed how magnocellular input may be vital for controlling sequential attention during reading by a magnocellular mediated feedback input to the striate cortex. Some studies can not verify the magnocellular/transient deficit theory, for example Walther-Müller (1995), Gross-Glenn et al. (1995) or can show that the presence of a magnocellular deficit depends on the type of dyslexia, for example Spinelli et al. (1997).

Some studies are based on the assumption of a deficit in the interaction of the magno- and parvocellular pathways in dyslexia and its consequences for the visible persistence as an effect of early visual functions. Visible persistence may be defined as any continued visible response to a stimulus after stimulus offset that is phenomenally indistinguishable from that occurring during the actual presence of the stimulus (Haber and Standing, 1970). In this sense visible persistence is the amount of time for which a visual stimulus continues to be seen after it has been terminated. Breitmeyer and Ritter (1986) and Lovegrove (1996) suggested that after a saccade has been made from one fixation point to another, magno/transient channels inhibit parvo/sustain channels so that the previously fixated stimulus does not remain visible and mask the next stimulus which is fixated. Such masking would interfere with reading. When dyslexics' eyes move across a line, the high visible persistence masks the currently fixated text and makes it difficult to identify letters or words. Therefore the visual deficit can be explained on the basis of this theory as an inadequate inhibition of the parvocellular system by the magnocellular system. Studies of Lovegrove (1996), Slaghuis et al. (1996), Slaghuis and Ryan (1999) show an increased duration of visible persistence in dyslexics in comparison to normal readers. The authors explain the longer duration of visible persistence in dyslexia as a consequence of a disorder in transient channel inhibition of sustained channels. Cestnick and Coltheart (1999) could find no group and no subtype effect, but Slaghuis and Ryan (1999) could show a significant subtype effect of an increased visible persistence in dyslexics.

In our experiments the role of visible persistence in dyslexia as a biological factor, which is cultural independent, was investigated by using the Ternus apparent movement tasks, which are used as a measure of duration of the visible persistence and as an index of the integrity of functioning of sustained and transient channels in dyslexia. Only in one of three subtypes of dyslexia we could find a significant increased visible persistence as a biological risk factor for the "smear over effect", which was first found by Breitmeyer and Ritter (1986).

A very strong other assumption has been developed during the last twenty years about the hemispheric co-ordination deficit in dyslexia. Research by Larsen, Hoien, Lundberg and Odegaard (1990) pointed to symmetric patterns of the left and right Planum Temporale in 15-year-old dyslexic children, who also had phonological deficits and who were either left-handed or both left- and right-handed somewhat more frequently than a control group. For the diagnostics it is possible to estimate the hemispheric co-ordination and dominance relation by the Hand Dominance Test (HDT) developed by Lienert (1976).

An additional biological basis of dyslexia was found by Robinson (2001) regarding an immune system dysfunction in the visual deficit subtype of dyslexia, indicating differences in connective tissue turnover due to infection or stress, which can cause a macular degeneration of the eye.

Examples of ***treatment methods*** regarding primary reasons are the following ones:

- Coloured glasses, transparencies and coloured paper of books should reduce the contrast and therefore the magnocellular activation. A compensation of the magnocellular deficit is expected.
- Reading windows should reduce the Breitmeyer- or "smear over effect".
- Prism glasses were developed for the stabilisation of the fixation point on the line.
- The cinesiological training like the "Brain Gym" program from Dennison and Dennison (1991) is based on the assumption of a hemispherical co-ordination deficit and wants to activate both hemispheres in the same time by special body exercises (e.g. symmetrical and cross-middle movements of the arms and legs).
- Dietary intervention, targeting specific biochemical anomalies were also investigated as a feasible treatment option.

4. Assessment and treatment regarding secondary causes — Partial performance deficits

The secondary causes of dyslexia were proofed in partial deficits in basic functions and not in complex actions of reading and writing. In principle, primary and secondary causes can be identified even before such children start formal education. Intervention and prevention can therefore also start before. Partial deficits can involve auditory and/or visual perception, tactile perception, working memory, long-term memory, motor functions and integration functions (Figure 4).

Partial deficits are related to basic functions, which are preconditions for higher complex activities like language, reading and writing (Figure 5).

The results regarding the deficits of auditory or phonological working memory seem to be present with relatively high consistency (Witruk & Ho, 2010). Deficits of visual working memory appear to depend strongly on the types of material used. Studies in which visual but nameable stimuli were used could be related to phonological decoding and to the phonological loop. The lower automation of Central Executive processes in dyslexics can be verified.

4.1. Working memory in dyslexia

Impairment of working memory performance in dyslexic children and adults has been found for visual and auditory presentation of stimuli with different paradigms and types of material.

4.1.1. Visual working memory in dyslexia

Regarding deficits in visual working memory, several results are available. So and Siegel (1997) found deficits for Chinese poor readers in visual working memory tasks (free visual reproduction of character lists with and without phonological, visual and semantic similarities). Ho and Bryant (1997) have reported that early visual memory skills are predictive of later reading performance in Chinese. Recent findings of Ho and her colleagues (2002, 2004, 2006) also suggest that the major difficulties of Chinese dyslexic children lie in visual-orthographic processing while some dyslexic children have difficulties in visual motion perception.

Ellis (1981) reported four visual matching experiments based on the Posner Paradigm with different material in which he was not able to find deficits for dyslexics if the two stimuli were not nameable. Significant deficits for dyslexics were shown if the visual stimuli were phonologically similar letters. He interpreted these results as naming deficits. Vellutino's findings (1987) also speak against a general deficit of the visual working memory. His dyslexic children were able to reproduce unknown Hebrew words and letters just as well as normal reading children. If the word list was in English, the dyslexic children performed significantly poorer than the control group. Vellutino's interpretation refers to a deficit of dyslexics during storage and recall of linguistic information. Likewise Barnea, Lamm, Epstein, and Pratt (1994) mainly found deficits for Hebrew speaking dyslexic children with series of lexical and visual stimuli.

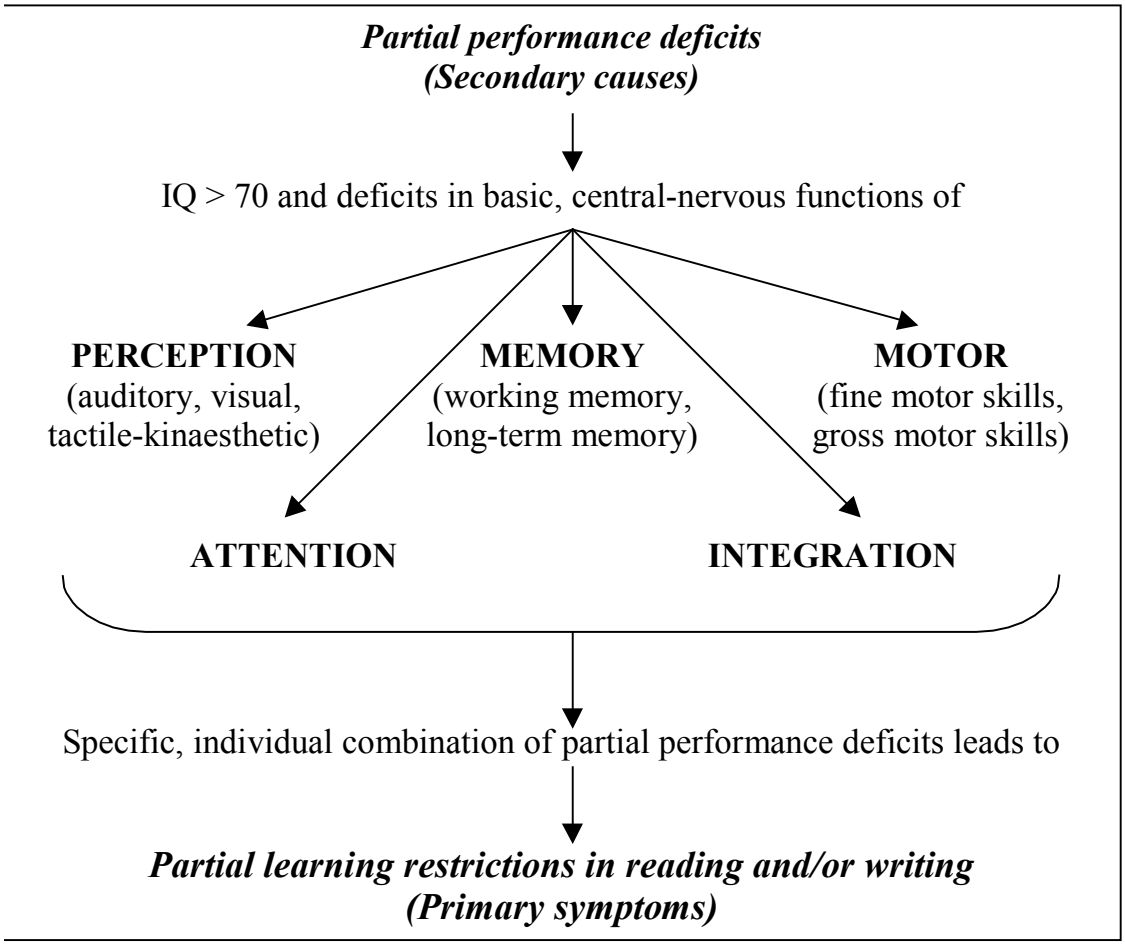

Fig. 4. Partial performance deficits

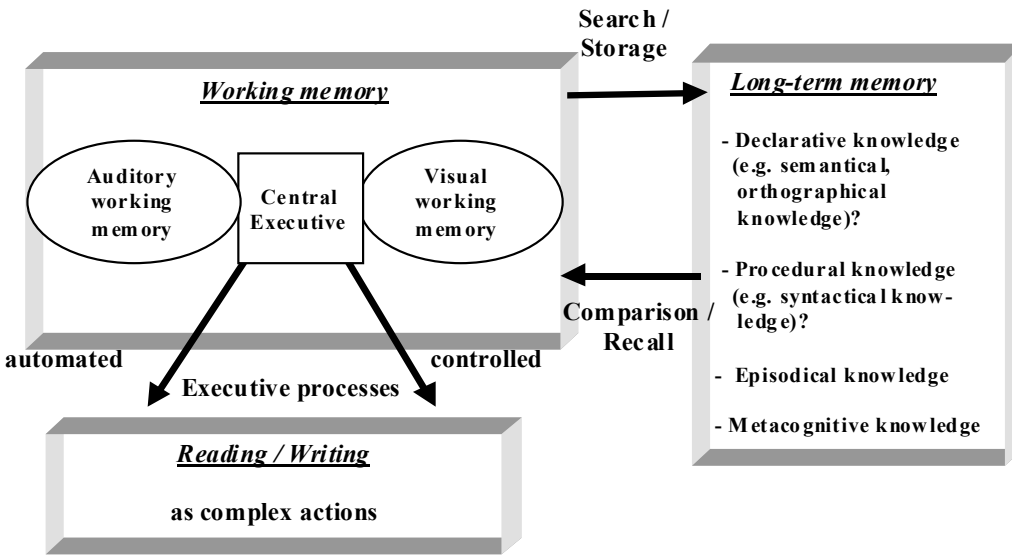

Fig. 5. Working memory functions during reading and writing (Witruk, 2003)

Using visual matching tasks, Willows, Kruk, and Corcos (1993) found deficits of dyslexic children with letters from the — to them unknown — Hebrew alphabet. These deficits in accuracy and speed were stronger in 6-year-old children than in 8-year-olds.

Compensation effects for deficits of visual working memory were shown in a study by Witruk and Rosendahl (1999) for visual matching tasks and visual serial recall tasks. For these visual working memory tasks they found significant adaptations towards the control group in a longitudinal and cross-sectional comparison between 7- and 9-year-old dyslexic children. For visual matching tasks Witruk (1993a, 1999) and Witruk, Ho and Schuster (2002) found a material-specific, nongeneral deficit in dyslexic children. For the accuracy parameter, significantly higher error rates were observed for dyslexic children with letters and dot patterns but not with line patterns.

4.1.2. Auditory working memory in dyslexia

The current discussion explores whether the reading and spelling difficulties of dyslexic children are based on auditory working memory deficits or on specific phonological working memory deficits with linguistic material like phonemes, syllables or words. Some studies show that the dyslexia deficit is based on the auditory field in general and also involves phonology. For example, Lachmann (2007) found a lower Mismatch Negativity in dyslexic children in comparison to nondyslexic children for linguistic stimuli but also for tone series. Mismatch Negativity represents auditory perception, discrimination, and memory processes on different levels (prior to semantic representations). Measured as a component of an acoustical evoked potential, it represents vast pre-attentional stimulus discrimination and memory comparisons. Auditory working memory deficits for nonlinguistic material were also found by Fischer (2007) for tone pairs, by Helenius, Uutela and Hari (1999) and, Hari and Renvall (2001) for tone series. Schulte-Koerne (2001) found that a smaller value of the Mismatch Negativity occurs in German dyslexic children compared to nondyslexic children for the passive perception, discrimination and memory comparison of verbal stimuli but not for nonverbal, auditory stimuli (sine tones).

Regarding the deficits of phonological working memory, research evidence has been more convergent. The most often used paradigm is the so-called memory span for numbers, words, and pseudowords. Deficits in phonological abilities and of phonological working memory in dyslexic Canadian children are described by Siegel and Linder (1984). Ho, Law and Ng (2000) and Ho and Lai (1999) were able to validate these phonological deficits in Chinese dyslexic children. According to Everatt, Groeger, Smythe, Baalam, Richardson, and McNamara (2001), phonological working memory deficits on sequential information (such as in digit span tasks) could be the root-cause of some other deficits and are evident across child and adult populations.

Gathercole and Baddeley (1993) found delays of development regarding articulation speed, rehearsal of nonwords, and memory span for words in 8-, 11- and 15-year-old dyslexic children. Phonological deficits which were found in 8- and 11-year-old dyslexic children were not found in 15-year-old dyslexic children. Thus, with regard to phonological working memory, one can call it a later onset in the tendency of compensation. Gathercole, Alloway, Willis and Adams (2006) found that phonological working memory skills represent an important constraint on the acquisition of skills and knowledge in reading and mathematics.

4.2. Central Executive functions in dyslexia

Proof of deficits in dyslexics in relation to Central Executive functions were found only in a few investigations. Schneider (2001) reported a stronger activation of the frontal lobe in dyslexic children during mental rotation and sound connecting tasks. She interpreted these results as a stronger involvement of the Central Executive in dyslexic children on the basis of an inefficient automation. The tasks used by Siegel and Ryan (1989) involved executive functions during word recognition after sentence completion and counting. They found generalized working memory deficits in dyslexic children (age 7-13), while children with arithmetic deficits had only a deficit in processing numerical information.

4.3. Intervention regarding secondary causes

- Tachistoscopic visual perception training (Gutezeit, 1977) is based on a very short presentation (0.5 sec.) of single words and word groups, which can be repeated until the child or the children group can read or write them. The training can be realized by an overhead projector or by a PC with LCD projector. Gutezeit has evaluated this training for dyslexic children of the 3rd grade and found significant improvements of the reading and writing performance.

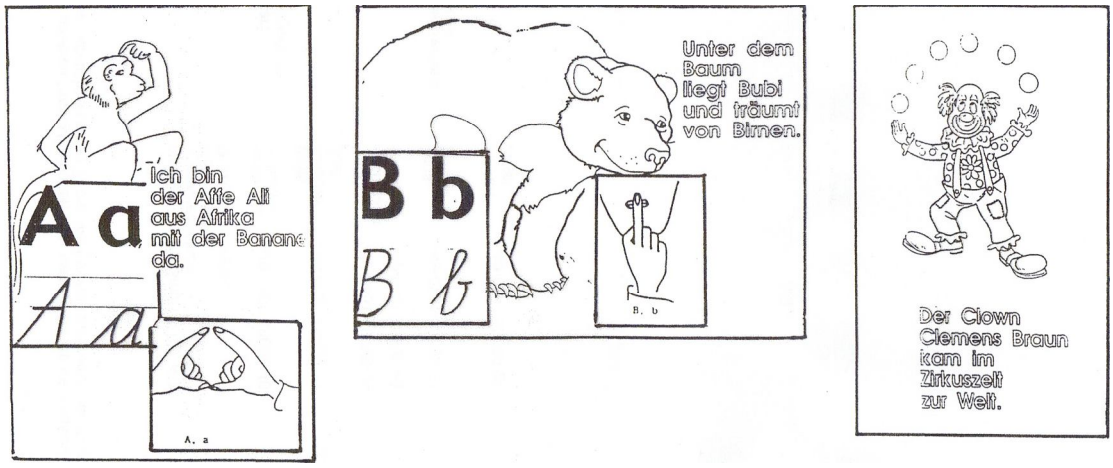

Fig. 6. Special learning material in the rehabilitative classes

- Working memory training (Witruk, 2003)
- Visual perception training (Frostig, 1974)
- Training of auditory functions (Warnke, 1998)
- Training of integration functions (Karma, 2003)

5. Assessment and treatment of primary symptoms — Failures in reading and writing

The diagnostic criteria in the ICD 10 are based on the analysis of failures in reading and writing in comparison to a better intelligence. This discrepancy between the IQ and reading and writing performance is the basic assumption. Sometimes this discrepancy is quantified. The typical errors in reading and writing are:

- Loss of letters, word parts, whole words,
- Reversal errors of letters or mirror errors (like "u" and "n", "b" and "d"),
- Adding of letters, word parts or whole words,
- Low reading speed,
- Low level of reading understanding.

5.1. Assessment of primary symptoms

For the assessment of the primary symptoms it is necessary:

Fig. 7. Reading exercise for dyslexic children "Which letter is in which picture?" (von Maydell & Vogel, 1977)

- to measure the reading performance with a reading test (for example the Zurich Reading Test (ZRT) from Linder and Grissemann, 2000),
- to measure the writing performance with a writing test (for example the Westermann writing test (WRT 6+) from Rathenow, Vöge and Laupenmühlen, 1980),

- to use a combined reading and writing test like the Salzburg reading and writing test (SLRT) from Landerl, Wimmer, & Moser (1997) and
- for the proof of the discrepancy to use an intelligence test like the Cultural Fair Test (CFT) from Weiss (1987).

5.2. Intervention regarding primary symptoms

- Special rehabilitative classes (2nd and 3rd grade) offer particular didactics developed by Weigt (1994) with script oriented playing therapy, with additional supporting hand signals, graphical signs for peculiarities of orthography and with a morphemic rule system for the better understanding of the construction of the German script. These special rehabilitative classes were evaluated by Witruk (1993b). A very good impact on the school career and the personality development of the dyslexic children could be found.
- Reading and writing learning institutes offer special didactics.

6. Assessment and treatment of secondary symptoms

Secondary symptoms can be developed in dependence on the feedback of the environment. The interactive behaviour of parents, peers and teachers with the dyslexic child has a high relevance for its self-esteem. The labilisation and the decrease of the self-esteem are the beginning of the development of emotional and behavioural disorders.

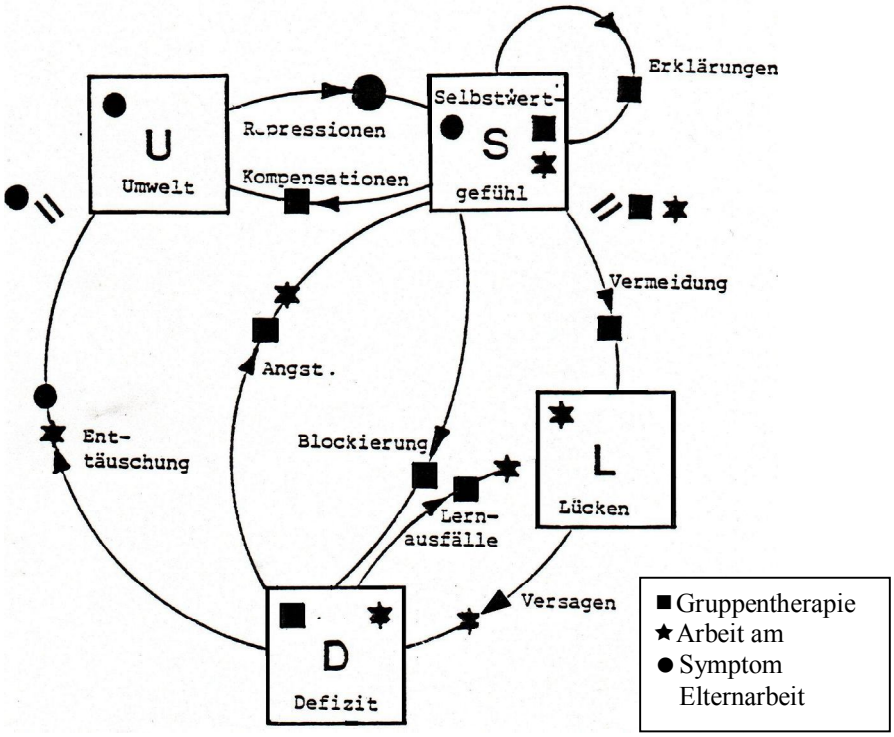

Fig. 8. Complex training programme developed by Betz and Breuninger (1982)

Betz and Breuninger (1982) describe four stages of the development of emotional and behavioural disorders:

1. After the first weeks in school negative attributions (of the failures) developed by the child and by the environment are starting. The first supporting activities by the parents will be experienced by the child as repression.
2. The dyslexic child tries to get success by the producing of compensating behaviour like clownery, violent behaviour or stealing presents for peers. But often the environment can not accept this behaviour and punishes it.
3. In the third stage the anxiety increases and leads to avoidance behaviour like absence

from school, blocking and avoidance of reading and writing demands.

4. In the fourth stage these disordered emotions and behaviour will be stabilized by the decrease of motivation, disidentification and the misunderstanding of the environment (for example the mistrust of parents in cases of successful performance of the child).

If a dyslexic child has reached this fourth stage it is not possible to exercise reading and writing but it is necessary to reduce the disordered behaviour and the anxiety and to stabilize its self-esteem. That means a psychotherapy or a complex program including the parents are strongly recommended.

Intervention regarding secondary symptoms

- The complex training programme developed by Betz and Breuninger (1982) is integrating three modules:

1. Group psychotherapy with children differentiated in children with high anxiety and children with violent behaviour,
2. Parent working meetings with psycho-education, exchange of experiences and information about the progress of the intervention,
3. Training of the orthography by using special didactics of success (for example exercise by self-control system, optimization of the learning organization, registration of correct responses — not of errors — in the dictate).

- Single- or group psychotherapy of the dyslexic child (client-centred, non-directive psychotherapy, behaviour therapy or psychoanalyses) or systemic therapy are integrating the whole family.

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ДИАГНОСТИКА И ЛЕЧЕНИЕ ДИСЛЕКСИИ

Эвелин Витрук

Лейпцигский университет, Институт психологии II, Германия

Статья посвящена краткому обзору различных современных методов диагностики и коррекции дислексии. На основе модификации и расширения многоуровневой модели Valtin (1989, модифицированной Witruk, 1993b) эти методы рассматриваются в контексте их основных задач. Описываются методы диагностики и коррекции в отношении первичных причин (биологические факторы риска), вторичных причин (парциальные дефицитарные функции), первичных симптомов (нарушения чтения и письма) и последующих вторичных симптомов (эмоциональные и поведенческие нарушения).

Ключевые слова: многоуровневая модель дислексии; магноцеллюлярный дефицит; парциальные дефицитарные функции; оперативная память; комплексная программа.