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Motor Learning and Chunking in Dyslexia

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ABSTRACT. The authors investigated whether participants with dyslexia had problems with executing discrete keying sequences and with switching between chunks in those sequences. Participants with dyslexia and participants in the control group executed 2 6-key sequences each, with 1 sequence consisting of 2 successive instances of 1 3-key segment (2 × 3 sequence) and the other not involving such a repetition (1 × 6 sequence). The authors assumed that during execution of the 2 × 3 sequence, the same chunk could be reused, whereas during execution of the 1 × 6 sequence a switch between chunks had to be made. Participants with dyslexia were slower than participants in the control group in executing the 1 × 6 sequence, but not the 2 × 3 sequence. The authors suggest that the smaller amount of repetitions of the chunks in the 1 × 6 sequence or the increased difficulty of the 1 × 6 sequence led to the slowed execution of the 1 × 6 sequence in participants with dyslexia.

Keywords: chunking, dyslexia, motor performance, sequence learning

People with dyslexia have difficulties with learning to read, spell, and write, despite normal intellectual capacity, adequate sociocultural and educational opportunities, and intact sensory abilities (Habib, 2000; Shaywitz, 1998). Approximately 5–12% of the world's population is affected by dyslexia (Katusic, Colligan, Barbaresi, Schaid, & Jacobsen, 2001). In addition to language problems, participants with dyslexia often suffer from sensory problems unrelated to reading (Eden & Zeffiro, 1998; Habib; Stein & Walsh, 1997) and have problems with processing rapidly successive information in the auditory and visual domains (Habib; Hari & Renvall, 2001).

At present, there are several theories regarding the cause of dyslexia. Most established is the phonological processing theory. This theory states that dyslexia is caused by a deficit at the level of phoneme representation, which leads to difficulties in using and manipulating phonemes when learning to read (Manis et al., 1997). The phonological processing theory only accounts for language-related deficits in participants with dyslexia, whereas numerous researchers have found additional problems in dyslexia unrelated to language. For example, researchers have found deficits in motor skills (Fawcett & Nicolson, 1999), balance (Nicolson & Fawcett, 1990), low-level visual and auditory processing (Talcott & Witton, 2002), and information-processing speed (Nicolson & Fawcett, 1994). In the present study, we investigated the relation between motor learning and dyslexia, and therefore we discussed theories regarding motor deficits in dyslexia in more detail.

One theory regarding motor deficits in dyslexia is the temporal processing theory. This theory suggests that different impairments that participants with dyslexia show (e.g., in language, visual, and sensorimotor tasks) all stem from a fundamental deficit in the processing of rapidly changing stimuli or rapidly successive stimuli (Habib, 2000; Tallal, Stark, & Mellits, 1985). Auditory experiments have suggested that for the typical participants with dyslexia', problems with reading, writing, and spelling are caused by difficulties with the perception of rapid acoustic elements in human speech (Tallal & Piercy, 1973, 1975; Tallal et al.). A related hypothesis, the cerebellar-deficit hypothesis, attributes timing problems in dyslexia to cerebellar dysfunction. The cerebellum is supposed to play a crucial role in timing and in motor and sequence learning (e.g., Ivry, Keele, & Diener, 1988). Researchers have found support for the cerebellar-deficit hypothesis by showing timing deficits in participants with dyslexia (Nicolson, Fawcett, & Dean, 1995) and by showing a diminished activation of the cerebellum in participants with dyslexia during motor learning (Jenkins, Brooks, Nixon, Frackowiak, & Passingham, 1994; Nicolson et al., 1999). Nicolson and Fawcett (2000) showed that even after extended practice, participants with dyslexia are slower and more prone to error on a keyboard spatial task and on a choice response task. Nicolson and Fawcett's (2000) finding supported the cerebellar-deficit hypothesis, which suggests difficulties with new and well-learned motor skills in participants with dyslexia. In addition, the cerebellum is thought to be responsible for the ability to establish associations between stimuli and responses and to be linked to implicit sequence learning (Menghini, Hagberg, Caltagirone, Petrosini, & Vicari, 2006), which suggests that participants with dyslexia have difficulties, in particular, when learning is implicit. Thus, the temporal processing theory states that participants with dyslexia have difficulties with the processing of rapidly changing stimuli or stimuli presented in rapid succession. The cerebellar-deficit hypothesis attributes these problems in dyslexia to the cerebellum and suggests problems with timing, motor learning, and implicit sequence learning.

In this regard, a second hypothesis regarding motor deficits in dyslexia, the sluggish attentional shifting (SAS)

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hypothesis, is interesting because it suggests that participants with dyslexia have difficulty with the disengagement of attention once their attention is engaged (Hari & Renvall, 2001). The SAS hypothesis suggests that because of disengagement problems, participants with dyslexia prolong chunks and have difficulties with switching between chunks. *Chunking* is a process of segmentation or reorganization that results in a reduced number of information units, called *chunks*. For example, a telephone number is more easily remembered as 06-23-24-25 than as 0-6-2-3-2-4-2-5. In this way, regularly used large quantities of information can be segmented or reorganized in practical units to overcome short-term memory limitations.

Therefore, the cerebellar-deficit hypothesis suggests problems with learning sequential movements occur because of timing difficulties, whereas the SAS hypothesis suggests problems with learning sequential movements occur because of difficulties with attention disengagement. A frequently used task for investigating learning of sequential movements is the serial response time (SRT) task. In the SRT task, participants react to a spatial cue by pressing the spatially corresponding key. Unbeknownst to participants, on some blocks the stimuli are presented in a particular sequence. Participants generally respond more quickly when the stimuli are presented in a sequence than when stimuli are presented pseudorandomly, even if participants cannot explicitly report the sequence and thus rely on implicit knowledge (Willingham, Nissen, & Bullemer, 1989). Previous studies have shown mixed results of the SRT task in participants with dyslexia. Some researchers found sequence learning deficits with the SRT task in participants with dyslexia (Howard, Howard, Japikse, & Eden, 2006; Menghini et al., 2006; Nicolson et al., 1999; Stoodley, Harrison, & Stein, 2006; Vicari et al., 2005; Vicari, Marotta, Menghini, Molinari, & Petrosini, 2003), whereas others did not (Kelly, Griffiths, & Frith, 2002; Rüsseler, Gerth, & Münte, 2006; Waber et al., 2003). Still, other researchers suggested that participants with dyslexia have difficulties with implicit sequence execution and not with explicit sequence execution (Vicari, Marotta et al.; Rüsseler et al.). Rüsseler et al. suggested that the lack of sequence learning deficits in dyslexia found in some studies with the SRT task was caused by the development of explicit knowledge with some versions of the SRT task.

In the present study, we used the discrete sequence production (DSP) task, which gave us the opportunity to study chunking (Rhodes, Bullock, Verwey, Averbeck, & Page, 2004; Verwey & Dronkert, 1996). In a typical DSP task, participants practice two discrete sequences by responding to series of three to six key-specific stimuli. All but the first stimuli are presented immediately after the response to the previous stimulus. Participants with dyslexia and participants in the control group practiced two DSP sequences, one consisting of two successive instances of one three-key segment (2 × 3 sequence) and the other not involving such a repetition (1 × 6 sequence; Verwey, Lammers, & van Honk, 2002). We imposed chunking during practice by inserting a variable response-stimulus interval (RSI) between the third and fourth key of both sequences (Verwey, 1996). The RSI induced a sequence with two identical three-key chunks (2 × 3 sequence) and a sequence with two nonidentical three-key chunks (1 × 6 sequence). Compared with the frequently used SRT task, the DSP task was characterized by a small number of key presses per sequence and more repetitions per sequence. Ultimately, with practice, the key-dependent cues were no longer needed because the first cue acted as an imperative stimulus for the entire sequence (Verwey, 1999). We investigated whether participants with dyslexia experience difficulties with executing DSP sequences. The cerebellar-deficit hypothesis suggests difficulties in dyslexia during the initial stages of sequence learning and less problems with practice (Nicolson & Fawcett, 2000). In addition, we examined whether participants with dyslexia have problems with switching between chunks in sequences. The cerebellar-deficit hypothesis suggests that participants with dyslexia are slowed on both the 2 × 3 and the 1 × 6 sequence because of an automatization deficit that is unrelated to chunking. The SAS hypothesis predicts that participants with dyslexia are only slowed on the 1 × 6 sequence, as a switch between two different chunks has to be made.

Method

Participants

Participants were 40 students from the University of Twente and the Saxion College, including 19 participants with dyslexia (12 men, 7 women) and 21 participants without dyslexia (11 men, 10 women). All participants were right-handed and between 18 and 28 years old. The participants with dyslexia had a documented history of the condition, as was shown by an official medical report. The participants with dyslexia were paid €24 for their participation, whereas participants in the control group (participants who were not dyslexic) received course credits for their participation. All participants were right-handed according to Annett's (1970) handedness inventory, were native Dutch speakers, and signed informed consent forms before the start of the experiment. There was no difference in handedness between participants in the control group and participants with dyslexia (see Table 1). The ethics committee of the University of Twente approved the study.

Apparatus

We controlled stimulus presentation and response registration with E-Prime (Version 1.1, Psychology Software Tools, Pittsburgh, PA) on a 2.8-GHz Pentium 4 PC running under Windows XP. Participants sat in a dimly lit room in front of a 17-in. computer screen with a viewing distance of approximately 60 cm.

DSP Task

Participants placed their right-hand fingers on four keys of a computer keyboard: the index finger on the C key, the

middle finger on the V key, the ring finger on the B key, and the little finger on the N key. Four horizontally aligned squares (2.5°) presented in the center of the screen functioned as placeholders for the stimuli. The four horizontally aligned squares subtended 13° and had the same alignment as the four response keys. The squares presented in silver on a black background and at the start of a sequence were filled with the background color (black). After a 1,500-ms interval, one square filled with yellow to which the participant reacted by pressing the corresponding key. Immediately after a key press, another square filled, and so on. If a participant pressed a wrong key, an error message appeared and the same square refilled until the correct response was given. With a premature first response, feedback indicated that the response was too early and the 1,500-ms fore-period started again. One sequence involved six key-specific cues, all of which the participant had to react to.

Each participant executed two six-key sequences, one sequence with two successive instances of one three-key segment (2 × 3 sequence) and one that did not involve such a repetition (1 × 6 sequence). The sequences appeared in a random order and were combinations of the keys C, V, B, and N. We used the following four combinations of 2 × 3 and 1 × 6 sequences with different participants: vnc–vnc and bcn–cbv; bcn–bcn and nvb–vnc; nvb–nvb and cbv–bcn; and cbv–cbv and vnc–nvb. Finger-specific effects are largely controlled by using—across participants in each group—each key in each position of the sequence.[1] We instructed participants to react as accurately and fast as possible to each stimulus and we measured response time (RT) from target onset to the next response. Participants practiced the sequences in four blocks of 160 sequences, yielding a total of 320 practice trials for each sequence. The fifth block was the test block, which also comprised 160 sequences. During practice, the RSI between the third and fourth key of all sequences varied randomly between 0, 200, 400, and 600 ms to enforce the same segmentation across participants. In the test block, the RSI between all keys was 0 ms. Halfway through every block there was a break for 20 s during which the participant could relax. During this break and at the end of each block, the participants received feedback about their mean RT and the number of errors since the previous feedback. Every practice block was followed by a short break of approximately 2 min, and we offered a break of at least 10 min between Blocks 2 and 3.

Procedure and Design

Before starting the experiment, we presented participants with the Dutch version (translation of the English version) of the Dyslexic Screening Test (DST; Fawcett & Nicolson, 1996; Kort et al., 2005). The DST includes a test of single-word reading, spoonerism, spelling, verbal working memory, writing, two tests of rapid naming, and two tests of phonemic awareness. All tests were paper-and-pencil based. Subsequently, participants performed the DSP task. After the DSP task, participants filled out a paper-and-pencil questionnaire that first asked them to recall the practiced sequences (recall test) and then asked them to identify the two sequences (of the 16 sequences) that they had practiced (recognition test).

Data Analysis

Time 1 (T1) indicated the time between stimulus onset and depression of the first key. The *interkey interval* was defined as the time between the onsets of two consecutive key presses in a sequence (stimulus onset co-occurred with depression of the previous key). The interkey intervals T2–T6 preceded key presses 2–6, and executing one sequence denoted a trial. We excluded from analyses the first two trials of every block, the first two trials after every break, and trials in which participants made one or more errors. We also eliminated from the analysis those sequences in which the sequence execution time—the sum of the six RTs in a sequence—lasted longer than the mean sequence execution time across participants per group and within blocks, plus 3 standard deviations. In this last procedure, we removed 1.6% of the trials (2.5% for the participants with dyslexia, 0.8% for the controls). We analyzed the number of sequences in which participants made one or more errors. However, we did not use the total number of errors because one error in a sequence could easily lead to additional errors in subsequent keypresses because of the high execution rate. We used the Greenhouse–Geisser correction with corrected values of the degrees of freedom whenever the sphericity assumption of the F test was violated.

Results

Dyslexia Tests

We analyzed scores of the dyslexia tests using a multivariate analysis of variance (ANOVA). We found a significant difference between the groups on dyslexia test performance, $F(9, 30) = 4.6$, $p < .005$. Table 1 shows the univariate tests comparing groups' scores on the dyslexia tests. It appears that participants with dyslexia scored significantly worse on the tests of picture naming, letter naming, reading, spelling, phonemic awareness (nonsense sentences), and writing, whereas there were no significant differences between the groups on the spoonerism test and the verbal working-memory test.

Practice Phase

Figure 1 shows the results of performance on the DSP task, Sequence × Group. We evaluated RTs using a repeated-measures ANOVA with the variables Block 1–4, RSI (0, 200, 400, or 600 ms), sequence (2 × 3 or 1 × 6), and key (T1–T6) as within-subjects variables and group (participants with dyslexia or control) and version (1–4) as between-subjects variables. During the practice blocks, the difference in RTs between the 2 × 3 sequence and the 1 × 6 sequence was larger for participants with dyslexia than for controls, as was shown by the significant interaction between sequence and group,

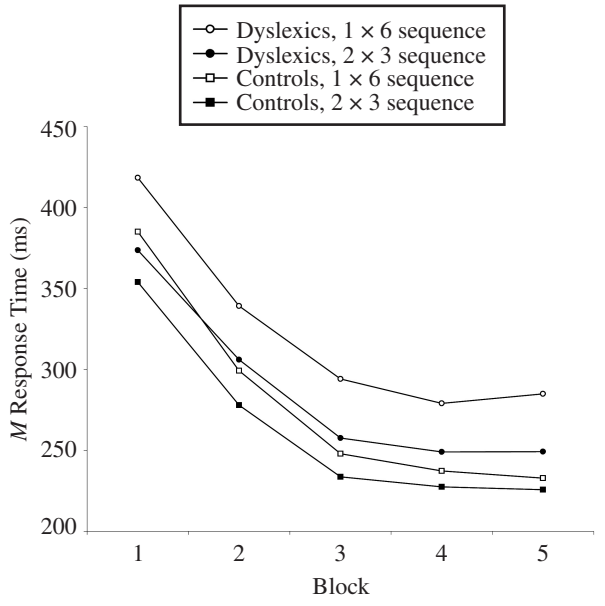

FIGURE 1. The results of performance on the Discrete Sequence Production Task × Sequence and Group.

$F(1, 31) = 5.7$, $p < .05$. There was no significant difference in RT across sequences and between groups during the practice phase, $F(1, 31) = 2.7$, $p > .1$. Furthermore, we performed a repeated-measures ANOVA with Blocks 1–4 and sequence (2 × 3 or 1 × 6) as within-subjects variables and group (participants with dyslexia or control) as between-subjects variable on error rates to investigate group differences in the practice phase. Participants made more errors during the 1 × 6 sequence than during the 2 × 3 sequence, $F(1, 38) = 8.0$, $p < .01$ (11.3% vs. 9.3%, respectively), and participants with dyslexia made more errors than participants in the control group, $F(1, 58) = 5.1$, $p < .05$ (12.1% vs. 8.6%, respectively).

Test Phase

We performed a repeated-measures ANOVA on RT with sequence (2 × 3 or 1 × 6) and key (T1–T6) as within-subjects variables and group (participants with dyslexia or control) and version (1–4) as between-subjects variables to investigate group differences in the test phase when RSI was 0 ms. A trend emerged that showed participants with dyslexia as slower compared with participants in the control group, $F(1, 32) = 3.9$, $p = .058$. A significant interaction between sequence and group showed that the difference in RT between the 2 × 3 sequence and the 1 × 6 sequence was larger for participants with dyslexia than for participants in the control group, $F(1, 32) = 11.1$, $p < .005$ (see Figure 1). Planned comparisons showed a significant difference between participants with dyslexia and participants in the control group for the 1 × 6 sequence, $F(1, 32) = 6.7$, $p < .02$, and no difference between the groups for the 2 × 3 sequence, $F(1, 32) = 1.6$, $p > .2$.

To investigate if there were differences among the sequences in the initiation of the sequence, the execution of the chunk, and chunk transition, we performed an additional ANOVA with the variable phase (initiation for Key 1; execution for mean Keys 2, 3, 5, and 6; and transition for Key 4). We found no significant interaction among phase, group, and sequence, $F(2, 76) = 0.5$, $p > .5$, or between phase and group, $F(2, 76) = 0.8$, $p > .4$. Last, we performed a repeated-measures ANOVA on error rates with sequence (2 × 3 or 1 × 6) as the within-subjects variable and group (participants with dyslexia or control) as the between-subjects variable to investigate group differences in the test phase. The ANOVA showed that participants with dyslexia made more errors than participants in the control group, $F(1, 38) = 4.7$, $p < .05$ (14.7% vs. 10.0%, respectively). Therefore, we did not observe any indications that transitions were slowed more than executions in participants with dyslexia.

Recall and Recognition Test

Table 1 shows the results of the recall and recognition tests for the two groups, per sequence. For recall, the maximum score was 6, as six keys could be recalled correctly. Participants in the control group had a perfect recall score for the 2 × 3 sequence ($M = 6$, $SD = 0$) and a near perfect score for the 1 × 6 sequence ($M = 5.90$, $SD = 0.44$). Participants with dyslexia had mean recall scores of 5.79 and 5.53 for the 2 × 3 and the 1 × 6 sequences, respectively (SDs = 0.92, 1.02, respectively). For recognition, the score was either 1 or 0, respectively, and correct or incorrect, respectively. Participants in the control group had a mean recognition score of 0.95 for both the 2 × 3 and 1 × 6 sequences (SDs = 0.22), and participants with dyslexia had a mean recognition score of 1 and 0.95 for the 2 × 3 and 1 × 6 sequences, respectively (SDs = 0, 0.95, respectively). We found no significant differences between the groups for the mean recall and recognition tests for both sequences (see Table 1). Their near perfect recall scores suggest that explicit knowledge had developed to the same extent for both groups.

Discussion

The goal of the present experiment was to investigate whether participants with dyslexia would have problems with executing learned movement sequences, specifically with switching between chunks in sequences. Seven of nine subtests of the dyslexia test battery showed significant differences between the participants with dyslexia and participants in the control group, confirming that the participants with dyslexia could be classified as such. The verbal working-memory test did not yield significant differences between the groups, and therefore verbal working-memory capacity can be excluded as a possible reason for group differences.

The DSP task involved two sequences, 2 × 3 and 1 × 6. The differences between the two sequences were that (a) the 2 × 3 sequence had the same chunk repeated, which leads to double exposure and (b) the 1 × 6 sequence included a shift between two different chunks, which was more

TABLE 1. Mean Scores, Standard Deviations, and Ranges for Each Group and Significance of the Difference Among Groups of Each Dyslexia Screening, Handedness, Recall, and Recognition Test

	Dyslexic			Control			
Variable	*M*	*SD*	Range	*M*	*SD*	Range	*p*
Dyslexia screening test							
Picture naming	35.53	9.31	25–69	30.29	3.85	25–40	.023
Letter naming	20.53	4.46	15–33	16.62	2.96	11–22	.002
Reading	44.21	10.61	29–71	32.10	6.07	23–44	< .001
Spoonerism	8.68	2.54	0–11	9.71	1.42	6–11	*ns*
Spelling	30.16	3.62	22–34	34.57	1.33	31–36	< .001
Working memory	6.32	1.92	3–11	6.24	1.58	4–10	*ns*
Nonsense sentences A	78.42	4.03	70–83	81.48	2.27	76–83	.005
Nonsense sentences B	129.95	45.32	71–235	66.10	17.04	43–109	< .001
Writing	26.68	5.28	13–36	32.10	3.85	22–37	.001
Annett's handedness inventory test	19.32	4.89	8–24	19.33	3.69	11–24	*ns*
Recall test							
2 × 3	5.79	0.92	2–6	6.00	0.00	6–6	*ns*
1 × 6	5.53	1.02	3–6	5.90	0.44	4–6	*ns*
Recognition test							
2 × 3	1.00	0.00	1–1	0.95	0.22	0–1	*ns*
1 × 6	0.95	0.23	0–1	0.95	0.22	0–1	*ns*

difficult than a shift between two identical chunks. Results showed that participants with dyslexia were slower than participants in the control group in executing the 1 × 6 sequence, but not slower in executing the 2 × 3 sequence. Because this slowing was found at all sequence positions, there seems to have been a general problem with executing the 1 × 6 sequence, rather than a chunk-transition problem as the SAS hypothesis predicted. The cerebellar-deficit hypothesis suggests that participants with dyslexia would initially be slower in executing both keying sequences, but with practice less slowing would occur. This was not confirmed because only the 1 × 6 sequence was slowed for participants with dyslexia in the test phase. We suggest that the smaller amount of repetitions of the chunks in the 1 × 6 sequence or the increased difficulty of the 1 × 6 sequence led to the slowed execution for the 1 × 6 sequence in participants with dyslexia. Further research needs to clarify the reason for the slowing of the 1 × 6 sequence for participants with dyslexia. Still, a trend was shown in the test phase, indicating that participants with dyslexia were slower than participants in the control group overall in sequence execution. We observed a similar trend during the practice phase, though it is not evident from the ANOVA. These two trends of slowing in participants with dyslexia indicated that, overall, participants with dyslexia were slowed in sequence learning compared with participants in the control group, which agreed with the automatization deficit in participants with dyslexia that Nicolson and Fawcett (1990) suggested, which is also in line with the cerebellar-deficit hypothesis. Nicolson and Fawcett argued that participants with dyslexia have a deficit related to automatization in all modalities and all tasks and thus also in gross and fine motor skills. These automatization deficits are thought to be related to a cerebellar deficit (Nicolson et al., 1995), for which researchers have found behavioral and neuroanatomical evidence (Fawcett, Nicolson, & Dean, 1996; Finch, Nicolson, & Fawcett, 2002; Nicolson et al., 1995).

Last, recall rates showed that all participants had developed explicit knowledge of the sequences. Previous research has shown that implicit and explicit sequence mechanisms were involved in parallel during sequence learning (Jiménez & Méndez, 2001; Willingham & Goedert-Eschmann, 1999). For the DSP task, researchers could hypothesize that initially, when participants responded to key-specific cues, execution relied on implicit knowledge. With practice, the implicit knowledge was repeated so many times that participants became aware of the repeating sequence, which led to explicit mechanisms becoming more important. With additional practice, as execution speed increased, implicit mechanisms became more important again, because execution became automatic. If the slowed execution of the 1 × 6 sequence in participants with dyslexia is related to the amount of repetitions of the chunks, then it is possibly related to the reliance on implicit and explicit mechanisms underlying sequence learning. Future research is needed to clarify this.

In conclusion, the present experiment showed that participants with dyslexia were slower than participants in the control group in executing the 1 × 6 sequence, but not slower in executing the 2 × 3 sequence. The slowing in the 1 × 6 sequence could not be related to the chunk transition in the sequence. We suggest that the smaller amount of repetitions of the chunks in the 1 × 6 sequence or the increased difficulty of the 1 × 6 sequence led to the slowed execution

of the 1 × 6 sequence in participants with dyslexia in the test phase.

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NOTES

1. The number of participants did not allow for perfect counterbalancing. However, removing 4 participants in the analyses to achieve perfect counterbalancing did not change the results. Therefore, we kept the number of participants unchanged.

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