Psychol Res (1981) 43:201–218
(Developmental Dyslexia Issue)

Psychological Research

Visual and Name Coding in Dyslexic Children

Nick Ellis

Department of Psychology, University College of North Wales, Bangor LL57 2DG, Wales, UK

Summary. Four experiments are reported which were designed to test for differences between dyslexic and non-dyslexic subjects at a number of visual information processing functions. It is argued that the older dyslexic child's reading problems cannot be ascribed to slowness of visual code production, to the limited capacity of the system, or to an extra rapid rate of decay. The results are compatible with the theory that, as a group, the dyslexic children tested show a slowness or inadequacy at a non-visual, name or linguistic coding level. It is suggested that this deficiency does not lie in the area of articulatory encoding but at an earlier stage where phonological or lexical codes are produced from visual stimuli.

Introduction

The suggestion that dyslexia can be usefully regarded as a deficiency in information processing is reflected in the work of Stanley and Hall (1973a, 1973b), Miles and Wheeler (1974, 1977), Wheeler (1977), Ellis and Miles (1977, 1978a, b, 1980), and Jorm (1979). In particular, Stanley and Hall (1973b) report that dyslexic children performed less efficiently than controls did on a task involving recall from 6-letter arrays presented for brief durations, while Ellis and Miles (1978a) found that processing time for 5-digit arrays was over four times as long for dyslexic children as it was for matched controls. They also found a high correlation between the speed of visual information processing (as indexed by stimulus acquisition from briefly presented arrays of digits) and reading speed. This phenomenon merits further study. The present experiments have been designed to elucidate the processes underlying this deficiency and to trace their relationship to reading processes.

Now the expression 'difficulty with reading' is too imprecise for use in strictly controlled research, the phenomena to which it refers being varied in their manifestations and attributable to a number of different causal factors (cf. Vernon 1977, 1979). There is little doubt however, that dyslexic children constitute a *relatively* homogeneous

0340–0727/81/0043/0201/$ 03.40

group of reading retardates since by definition their reading disability cannot be ascribed to low intelligence, primary sensory defects, or emotional disturbance.

Whether dyslexic children form a qualitatively homogeneous group will depend upon the level of study. The group is, again by definition, uniform in its members' difficulties at reading and spelling, and by experiment (Ellis and Miles 1977, 1978a) uniform in its members' slowness at processing simple alphanumeric arrays from a single fixation.

Several studies have claimed that there are various types of dyslexic children. Boder (1971) distinguishes between the dysphonetic (those children showing difficulties at phonic analysis and synthesis) and the dyseidetic (those poor in the perception of whole visual structures, be they words or letters); Ingram (1960) distinguishes between visuo-spatial deficiencies, auditory-linguistic deficiencies, and the cross-modal deficiencies in relating visual symbols to their phonemic equivalents, while Vernon (1979), in reviewing this area, categorises into five groups of disability involved in reading retardation, viz., inability 1) in the analysis of complex visual shapes, 2) in the analysis of whole word sounds into phonemes, 3) in the acquisition of simple regular grapheme-phoneme *associations*, 4) in the grasping of irregularities in these, and 5) in the grouping of single words into phrases or sentences. A deficiency in the processing of simple numeric arrays may be an eventual result of almost any of the above proposed functional deficiencies.

A promising initial distinction is that between visual and name functions: is there uniformity in that the dyslexic child's single fixation reading slowness is a result of problems in the visual encoding and analysis of stimulus information, or alternatively in the creation of name representations for visual stimuli? The evidence so far seems inconclusive. The fact that some dyslexic children show unusual persistence in confusing 'b' and 'd' both in reading and writing (Critchley 1970; Vernon 1971) might seem at first glance to suggest a visual code problem, whereas the slow reaction time of dyslexic subjects in colour and picture naming (Denckla and Rudel 1976) points rather towards a name encoding deficiency.

Experiment I

Posner (1969) has demonstrated, in a letter matching task, that responses of 'same' to physically identical letters (AA) occurred more quickly than responses of 'same' to physically dissimilar letters (Aa). He therefore distinguished between visual code comparisons and name code comparison; and it is possible as a result of his technique to collect speed and error data for the production and matching of both types of stimulus representations. If dyslexic subjects are slower or less efficient at encoding the visual features of the stimulus independently of their ability to name, then more errors and relative slowness as compared with controls would be expected in situations of visual code comparison. In contrast, if their deficiency is primarily one involving the name-code, then they would be slower and/or make more errors specifically in the name-coding conditions.

In the present experiment a variation on the Posner procedure was introduced. This involved pairs of letters which, though different, were either visually similar (e.g., OQ)

Table 1. Subject data for Experiments 1 and 2. Means and (SDs)

	Dyslexics n = 21	Controls n = 21
Chronological age	11.8 (0.7)	11.8 (1.0)
Reading age	9.0 (0.5)	12.7 (1.3)
Spelling age	8.3 (0.6)	12.6 (1.1)

or phonologically similar (e.g., Gd). These 'similar' conditions were included since it is to be expected that a deficiency in the production or comparison of a given type of stimulus feature representation will be associated with slower and/or more error-prone performance with letter pairs that are confusable on that stimulus feature dimension. Thus, for example, longer latencies or more errors may be expected for the phonologically similar letter pairs if the subject has difficulty at name encoding.

Subjects

Two groups of 21 boys were tested. The dyslexic subjects were chosen from a private school which specialised in dyslexia. A check was made of the school records so as to ensure that no child was chosen as a dyslexic unless all of the following conditions were satisifed: a) reading age (RA) on the Schonell R_1 test was at least two years behind chronological age (CA), b) spelling age (SA) on the Schonell S_1 test was at least two years behind CA, c) there was no evidence of any gross behavioural problems or of any gross organic disorder, and d) there was average intelligence or above, as determined by recognised intelligence tests, usually the Wechsler or the Terman.

Members of the control group were also chosen from private schools. Inclusion was conditional upon a) a score of average or above on a recognised intelligence test and b) RA and SA not more than one year behind CA.

Table 1 shows the characteristics of the two groups in terms of chronological, reading, and spelling ages.

Materials and Procedure

Six categories of test stimuli were used, each category comprising pairs of letters. The types of pairs and the actual letters used are shown in Fig. 1.

For the visually different condition the pairs were chosen so that they were as far as possible neither physically nor phonologically confusable, while for the visually similar condition the letters, though different, were designed to be visually but not phonologically similar. Visual similarity was achieved by the presentation of two upper case letters, choice of letter pairs being influenced by the findings of Townsend (1971). For the phonologically different condition the letters were designed to be neither visually nor phonologically confusable, while for the phonologically similar condition the letters were designed to be phonologically but as far as possible not visually similar. Guidelines for choice were taken from the confusion data of Conrad (1964) and Wickelgren (1965).

SAME CASE PAIRS

Visually Identical run1	Visually Identical run2	Visually Dissimilar	Visually Similar
OO	OO	OB	OQ
RR	RR	RM	RP
FF	FF	FS	FE
CC	CC	CT	CG

DIFFERENT CASE PAIRS

Phonologically Identical run1	Phonologically Identical run2	Phonologically Dissimilar	Phonologically Similar
Bb	Bb	Ba	Bd
Mm	Mm	Mb	Mn
Dd	Dd	Ds	Dp
Gg	Gg	Gw	Gd

Fig. 1. Test letter pair stimuli used in Experiment 1

Letter pairs, printed centrally on white card with 28pt folio light letraset were presented in an Electronic Developments 3-field tachistoscope. Each trial began with the warning signal 'ready', followed by the presentation of a fixation cross for 1 s at an intensity at the subject's eyes of approximately 0.15 lux. The offset of the fixation cross was followed by the exposure of a test letter pair for 2 s at an intensity at the subject's eyes of approximately 1.8 lux. The on-set of the letter pair started a Dawes digital meter, counter and timer, type 3000A, accurate to 1 ms, and the subject's vocal response stopped the timer by means of a voice key. A record was kept of this time.

Testing occurred in a quiet, dimly lit schoolroom. The subject was told that he would be seeing pairs of letters printed on cards held in front of him and that these pairs would consist either of two capital letters or of one capital and one small letter: if the two letters were the same (i.e., of the same name) he was to say 'yes,' and if they were different he was to say 'no', and he was to give his answer as quickly as possible. He was then given eight practice trials with flash cards and was corrected if he made a mistake or was confused. He was then moved to the tachistoscope where, under the same instructions, he received eight further practice trials. None of the different letter pairs in any of the practice trials were either acoustically or visually confusable.

After the practice trials, the subject then participated in 32 test trials with the stimuli shown in Fig. 1; these were presented in quasi-random order and were counterbalanced for both condition and response-type in such a way that the sequence of 'yes' and 'no' trials varied randomly, with the constraint that no sequence of any one kind was longer than three trials.

A baseline reaction time measure, measured by the same timing arrangement and under exactly the same conditions, was also taken for each child. As the experiment involved two responses ('yes' and 'no'), a two-choice reaction-time procedure was used rather than a measure of a simple reaction time: the child was instructed to say 'yes' as soon as possible after the onset of a solid blue circle and 'no' after the

Table 2. Means and (SDs) for response times (ms) in Experiment 1

Condition	Dyslexics		Controls	
Two-choice RT				
Baseline	838	(211)	780	(137)
Visually identical	960	(254)	933	(240)
Visually dissimilar	1065	(276)	1023	(234)
Visually similar	1219	(306)	1200	(359)
Phonologically identical	1156	(360)	1039	(277)
Phonologically dissimilar	1159	(309)	1059	(257)
Phonologically similar	1304	(366)	1188	(336)
Group X type interaction response times				
Same case	1051		1022	
Different case	1194		1081	

onset of a faint circle outline. Again practice was given with a series of flash cards. Then four practice trials were conducted with the tachistoscope, followed by eight test trials.

Results

The mean reaction times and their standard deviations for the dyslexic and the control children are shown in Table 2. Errors were too infrequent to analyse, error rates being approximately 1% for the controls and 4% for the dyslexics.

The two-choice reaction time baseline means do not differ significantly ($t = 1.5$, df = 40). The remaining data were analysed as a 3-way factorial with subjects nested within groups: 2 groups (dyslexic, control) × 2 types (visual, phonological) × 4 conditions (totals for each subject of 'run 1 identical,' 'run 2 identical,' 'dissimilar,' 'similar'.). The 'type' ($F \approx 51.84$; df 1,40)[1] and 'condition' ($F = 50.9$; df 1,40) factors were significant at the .01% level. The 'group' x 'type' interaction ($F = 9.01$, df 1,40) and the 'type' × 'condition' interaction ($F = 8.38$, df 1,40) were significant at the 1% level. No other factors or interactions were significant.

Discussion

Within the present framework, the 'group' × 'type' interaction is the most interesting one, especially as there was no significant difference overall between the two groups. It can be seen from Table 2 that the dyslexic subjects do not respond more slowly than the controls on the same case letter pairs that are considered to be judged same or different on the basis of their visual characteristics, but they are slower at judging the different case pairs where the comparison is one of name codes ($t = 5.69$, $P < .01$). Supramanian and Audley (1976) have found a similar pattern of results with poor readers.

[1] Since this is a repeated measures design all dfs used are conservative

There are a number of possible conceptualisations of the processes and the order of operation of processes involved in such tasks in which stimuli are sometimes visually identical (*RR*) requiring a 'Same' response, sometimes phonologically but not visually the same (*Mm*) requiring a 'Same' response, and sometimes different both visually and in name (*Rm* or *Ds*) requiring a 'Different' response.

All the models assume that letter stimuli can be encoded both for their visual features and for their name representations. Another common assumption is that the production of name codes takes longer than the production of visual codes (cf. Posner 1969; Coltheart 1972). These assumptions fit the present data: the different case pairs that are assumed to involve name code comparisons take on average 101 ms longer to judge than the same case pairs. This difference was highly significant.

The models differ in the temporal ordering of these processes. A *parallel* encoding possibility assumes that visual code comparison operates in parallel with name code comparison (see, e.g., Cohen 1969). In *serial* encoding models (see, e.g., Egeth 1966) visual and name comparisons are organised in series, and visual comparisons are made first.

On the parallel view visual codes affect the response times *only* when two stimuli are physically identical; in all other circumstances the stimuli are dealt with by the name code system; and in this case visual similarity (*RP*) or dissimilarity (*RM*) would not be expected to influence the 'Different' latencies. On the serial view, however, it will take longer for visual comparisons to reach a 'Different' decision when stimuli are visually *similar* and hence the implementation of name matching will begin later for such stimuli. This would result in longer 'Different' latencies for visually *similar* stimuli (*RP*).

The present data are more consistent with the serial view: the average latency for visually similar pairs was 1210 ms, the average latency for visually dissimilar pairs was 1044 ms, a highly significant difference.[2]

It is important to emphasise that while there appears to be a deficiency in the name coding processes in the dyslexics, there is no suggestion of a deficiency in dealing with the visual codes of letters.

Now it is regularly claimed that young dyslexic children show confusion in both the reading and the writing of certain letters (see, e.g., Critchley 1970; Vernon 1971; Liberman et al. 1971). This is especially true when the letters are reversible, e.g., b/d and p/q. The findings of the present experiment showed that visually similar letter

---

[2] Both the serial and parallel views, as stated, assume that the 'Different' response to same-case different stimuli (OQ, RP, EF, CG, OB, RM, ES, and CT) requires the comparison of name codes. Such responses could not be a result of visual code comparisons since when visual code comparisons reveal that two stimuli are visually different a 'Different' response may not be warranted because the stimuli may be of the 'Mm' kind: visually different but requiring a 'Same' response. Hence dyslexic children should be slower in this condition just as they were reliably slower than the control children in responding on the basis of name code comparisons with the different case stimuli. However, this was not so. A plausible explanation is that the physical characteristics of the large upper case second letter of the same case pairs are, as a class, so distinctly different from those of lower case second letters of different case pairs that 'Different' responses to visually different upper case letters are warranted without recourse to name code comparison.

pairs (*RP*) were responded to as being different more slowly than visually different (*RM*) pairs. However this trend was seen to an equal extent in both dyslexic and control children. Furthermore the same pattern is also found in the phonological conditions: the addition of confusability appears to slow down both dyslexic and control subjects equally. Hence it is unlikely that the letter confusions so often seen clinically in dyslexic children are simply due to a limitation at the visual code level. On the contrary it is necessary to push back the level at which these confusions occur.

Vellutino et al. (1972) and Vellutino et al. (1975), presented their subjects (good and poor readers in the range between second and sixth grade) with tachistoscopic exposures of both verbal and non-verbal stimuli and asked them to identify and/or reproduce these stimuli both orally and in writing. The poor readers manifested considerably greater accuracy in copying and naming letters in words than they did in pronouncing these same words. The poor readers also differed from the controls in the types of errors they made, but this occurred only in the case of oral responses, not in the case of written ones. Here, too, therefore, the same general pattern of results is found, in that poor readers perform normally in dealing with graphemic stimuli as such but once they are set a task that requires grapheme to phoneme translation they perform less well.

Experiment 2

The dyslexic children tested here behaved uniformly as a group in that they showed no impairment in the speed or accuracy with which they judged letters on the basis of their visual features, and in that they were consistently slower than the controls in comparing letters on the basis of name features. This does not prove, however, that there is no visual code problem in poor readers. A deficiency may have been masked by learning overlay: the children tested with the Posner task were 10 to 12 years old with a reading age of 9 and had long mastered the skills necessary for correct identification of single letters. A different result might have been obtained with younger dyslexic children, or with unfamiliar visual material. Therefore a visual matching task was devised with confusable non-alphanumeric stimuli. Since these stimuli were new to the children there was no possibility of the immediate results being affected by learning overlay. Also they were not nameable (or at least not nameable at first glance) and thus only visual coding was involved. Letter-like forms similar to those devised by Gibson et al. (1962) were used.

Method

The same subjects as in Experiment 1 took part. The procedure was basically the same as that of Experiment 1. Again baselines for two-choice reaction time were determined for each child. He was then told that he would be seeing pairs of shapes, the two shapes being exactly the same or different; if they were the same, he was to say 'yes,' and if they were different he was to say 'no' as quickly as possible. Sixteen letter-like pairs were then shown sequentially on flash cards for practice and all errors were corrected. Next came 8 practice trials on the tachistoscope, followed by 32 test trials. Exposure sequences on the tachistoscope and timing procedures were identical with those used

in the previous experiment. The test stimuli are shown in Fig. 2. The four basic stimuli were chosen to be examples of simple and complex straight and curved features. The transformations used included reversed and rotated versions of the basic stimuli, since these might be expected to create special difficulties for dyslexic children in view of their alleged difficulties over 'left' and 'right' and over 'up' and 'down'. The presentation order of the stimuli was counterbalanced in respect of both response type ('yes' or 'no') and in respect of the different variants at the basic stimuli.

Results and Discussion

The mean reaction times and the standard deviations these means for the two groups of subjects are shown in Table 3.

The data were analysed as a 3-factor ANOVA (groups, conditions, type of stimulus).

As in Experiment 1, the main group factor ($F = 1.28$, df 1,40) failed to reach significance, even though the means for all conditions of the dyslexic group were larger than those of the control group. This was due to the large subject variability. The conditions and type of stimulus factors were both highly significant, but are of little interest here. As all interactions with groups were insignificant, it must be concluded that the dyslexic and control children were affected by all the variables in a similar manner.

Errors were again too infrequent to allow analysis across the two factors of groups and conditions. When, however, errors across the different conditions were summed

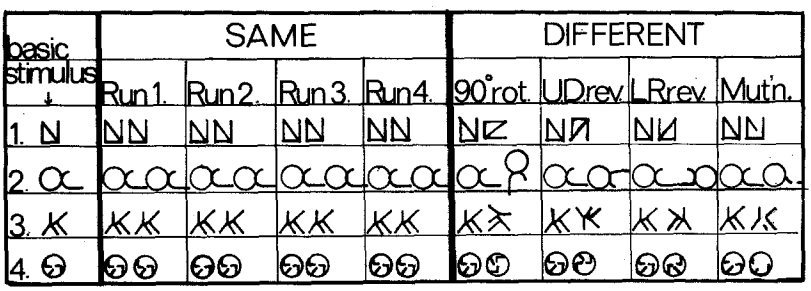

Fig. 2. Test letter-like form stimulus pairs used in Experiment 2

Table 3. Means and (SDs) for response times (ms) in Experiment 2

Condition	Dyslexics		Controls	
Same				
Run 1	1185	(404)	1117	(376)
Run 2	1296	(510)	1160	(353)
Run 3	1266	(434)	1217	(463)
Run 4	1269	(459)	1173	(351)
Different				
90° rotation	1263	(405)	1182	(307)
UD reversal	1317	(339)	1251	(370)
LR reversal	1422	(498)	1269	(357)
Mutation	1427	(458)	1356	(478)

for each subject, the difference in error rate across groups (there were 32 test trials; mean and (sd) errors for the controls was 1.62 (1.53) and for the dyslexics was 3.38 (3.23)) just reached significance ($N_1 = N_2 = 21$, $Z = 2.20$, $P < .05$, Mann Whitney normal approximation).

It must be concluded that the dyslexic group showed no impairment in terms of either the speed or the accuracy with which they could judge two letters to be the same or different on the basis of their visual characteristics in Experiment 1. The same conclusion applies with regard to the speed at which they judged whether two non-letter stimuli were the same or different in Experiment 2. It would be incautious, however, to conclude that there is *no* impairment in dyslexics when comparing the visual features of novel and confusing nonsense shapes since the errors, though too few to subject to their intended analysis, lend some small support to the notion that the dyslexics may be slightly less accurate.

Experiments 3a and 3b

It is still open to argument that there may be a deficit in the visual coding system of dyslexic children. For example, dyslexic and control children may differ with respect to the capacity or decay rate of the visual code. One possibility is that visually encoded information serves as the data-base for name encoding functions (Coltheart 1972; Mitchell 1976). Thus if the visual code were to decay more rapidly in dyslexic children, then name-coding would be impeded. If the visual code is not a locus of deficiency in dyslexia, then all such possibilities need to be excluded.

For this purpose a procedure used by Phillips and Baddeley (1971) and Phillips (1974) was chosen. This involves showing the subject a matrix of cells similar to a chess board. In its simplest form this was a 4 x 4 (16 cell) matrix with half the cells filled in a random arrangement. First one such matrix is shown, and then, after a variable interstimulus interval (ISI), a second which is either identical with the first or, if different, different in respect of one cell only. The subject has to report if the two matrices are the same or different. This procedure, like that of Experiment 2, excludes the possibility of learning overlay, since the material was unfamiliar to the subjects and, unlike alphanumeric stimuli, had no symbolic significance. In contrast with the procedures of Experiments 1 and 2, however, it has the advantage of making possible the determination of the rate of decay of the visual code. Posner (1969) did in fact claim that this could be done if one determined the duration (with the two letters presented sequentially) at which physical-match reaction time was no quicker than name-match reaction time. It is possible, however, as Phillips and Baddeley (1971) have pointed out, that in the case of letters there may have been both visual code decay and increased use of name code, since the subject may use the name code in preference to the decaying visual code even though there is still some information in the latter. This objection does not apply to the present procedure.

Subjects

These were i) 61 male dyslexic children (selected from the same school as before) with a mean CA of 12.3 years (s.d. 1.0), a mean RA of 9.9 years (s.d. 1.5), and a mean SA of 9.0 years (s.d. 1.3); ii) 22 male control children with a mean CA of 11.9 years (s.d.

1.0), a mean RA of 12.8 years (s.d. 1.3), and a mean SA of 12.5 years (s.d. 1.1); and iii) 26 first-year undergraduates with a mean CA of approximately 19 years. The criteria detailed in Experiment 1 were again applied to determine membership of the dyslexic and control groups. The undergraduate subjects were included to investigate the possibility of group performance differences associated perhaps with age or superior reading ability.

Method

Experiment 3a. 4 x 4 square cell matrices were constructed on white cards with half the cells blacked in at random. A new pattern was used on each trial. The matrices were photographed with a Bolex cine camera on 16 mm Kodak High Contrast negative film 7457. Each trial consisted of 120 frames of fixation cross followed by 24 frames of the randomly filled 4 x 4 matrix. There was then a variable ISI (either 1, 2, 5, 10, 48, or 143 frames) before the second matrix appeared, again for 24 frames. The second matrix either was identical with the first or had one cell more or one cell less filled. During the ISI there was either a blank field (dark when projected) or a pattern mask consisting of a larger matrix with cell size linear dimension half of that used with the test matrices, approximately half of the cells of the mask matrix being filled randomly. Each trial followed the sequence shown in Fig. 3.

The film was constructed so that after 6 practice trials there were 48 test trials (4 trials at each ISI with ISI blank, 4 trials at each ISI with ISI filled by a mask) with order of presentation of trials counterbalanced for ISI, mask/no mask, and same/different, matrix 1 being identical with matrix 2 in half the trials and in half the trials differing from it by one cell.

The subjects saw the film in large groups in a dimly lighted room and were instructed to mark an 'S' on their score sheet if the cell arrangements in the two matrices were the same, and a 'D' if they were different. The subjects were questioned during the practice trials so as to ensure that they understood the instructions and that they did not 'get lost.' The number of each trial was spoken before each pair of presentations. There was a break of approximately 5 s between each trial.

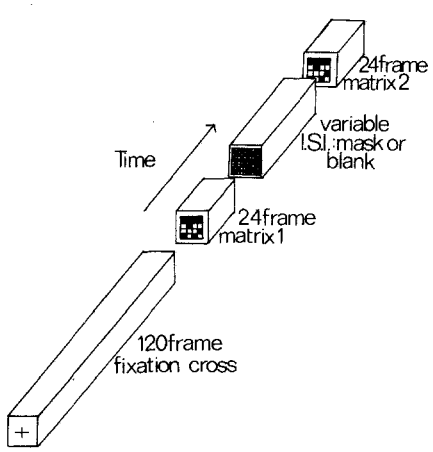

Fig. 3. Exposure sequence of a single trial from the matrix match: Experiment 3

Table 4. Percentage of subjects correct in the different conditions of Experiment 3

	4 × 4 Matrix, ISI blank			4 × 4 Matrix, ISI mask			5 × 5 Matrix, ISI blank		
ISI frames	Dyslexic subjects	Control subjects	Undergraduate subjects	Dyslexic subjects	Control subjects	Undergraduate subjects	Dyslexic subjects	Control subjects	Undergraduate subjects
1	88	91	94	77	69	87	78	86	90
2	77	83	83	72	74	81	87	86	82
5	97	99	97	80	82	83	82	59	83
10	90	98	95	77	74	90	81	71	80
48	86	91	91	73	69	81	61	78	69
143	70	72	69	60	70	85	68	72	72

The film was projected on to a white screen at a speed of 24 frames per second by means of a Bell and Howell 644 projector. The complete procedure for each trial was thus: fixation cross (5000 ms), matrix 1 (1000 ms), variable ISI (42, 83, 208, 417, 2000, and 6006 ms), matrix 2 (1000 ms).

The percentage of subjects in each group correct on each trial was then calculated.

Experiment 3b. After a break of approximately 10 min the subjects took part in a further experiment. The equipment instructions, and method of projection were identical with those of Experiment 3a. More complex matrices were used, however, consisting of 5 × 5 cells. Exposure time was increased for both matrix 1 and matrix 2 to 48 frames (approximately 2 s); the same ISIs were used but no ISI was filled by a mask.

Results and Discussion

The percentages of dyslexic children, control children, and undergraduates giving correct answers in the different conditions are shown in Table 4.

The results of Experiment 3a were analysed as a 4-factor ANOVA: 3 groups (dyslexic children, control children, undergraduates) × 6 ISIs × 2 mask (mask, no mask) × 4 blocks. The mask factor ($F = 4.15$; df 5,105); $P < .01$) was significant but no other factors or interactions reached significance.

The presence of a mask in the ISI decreases the likelihood of correct responding: mean percent correct with the ISI blank is 87.1%, whereas with the mask the percentage falls to 76.7%. This finding replicates that of Phillips (1974), who found out that the presence of a mask resulted in fewer correct responses, especially on ISIs less than 100 ms.

The data of Experiment 3 b were analysed as a 3-factor ANOVA: 3 groups (dyslexic children, control children, undergraduates) × 6 ISIs × 6 blocks. Only the ISI factor ($F = 3.82$; df 5,58; $P < .01$) was significant. Both the group factor and the group × time interaction were insignificant which shows that even with these more complex matrices, there was no significant difference between the three groups in respect of either capacity or decay rate of the visual code as measured by this method.

These results therefore strengthen the argument that there is no major impairment of the visual code in dyslexic children. Neither the production nor the capacity nor the decay characteristics of the visual code can in any way be distinguished from controls. They can thus not be the basis for the name coding deficiency, indicated by the findings in Experiment 1.

Experiment 4

We now need to focus on the nature of the name code since it was only here that dyslexic children were impaired. Since Posner first introduced the paradigm used in the first two experiments it has become recognized that there is more than one type of internal name code. The accepted distinction is between a *phonological code* (the form of code that represents an auditory image or that may result directly from the application of grapheme-phoneme conversion rules to a letter string, Coltheart 1978) and an *articulatory code* (the form of code that is the immediate precursor of an overt articulation: 'inner speech'). The question then arises as to whether it is the phonological or

the articulatory (or conceivably both) type of name code that is the locus of deficiency in dyslexia. Ellis (1980) has attempted to throw light on this question by using articulatory suppression (which can be assumed to interfere with articulatory encoding) in Posner-type tasks with undergraduate subjects; he found that neither 'visual code' nor 'name code' matching was affected. This may mean that at least for undergraduates the name code used in such tasks is non-articulatory, but phonological. However, the question remains as yet open.

There are several interesting hypotheses for processes involved in name coding that deserve exploration. Sperling (1967) suggested that as a result of a 'scan' of the visual information store there arises a 'program of motor instructions' for later articulation. Allport (1978a, b) postulates the production of a 'lexical code' as a consequence of input logogen activation, and other models (e.g., Baddeley 1979; Baddeley and Lewis 1979; Coltheart 1978) acknowledge the possibility of direct grapheme-phoneme conversion by means of pre-articulatory phonological coding or 'auditory imaging.'

However, before considering these possibilities, it is necessary to test the simple hypothesis, namely that the name code deficiency in dyslexic children is to be found at the level of articulatory encoding. A direct test of this hypothesis is to compare dyslexic and control children in respect of the speed at which they can repeat auditorily presented words. In this case the need to create a non-articulatory name code or lexical code from graphemic stimuli is bypassed but the stimuli still require articulatory encoding for output.

Subjects

The subjects were 13 dyslexic boys (mean CA 11.8 years (range 11.0-12.9), mean RA 8.9 years, and mean SA 8.0 years), from the same school as those of the earlier experiments, and 13 control boys of mean CA 11.8 years (range 10.9-13.0), mean RA 12.8 years, and mean SA 12.8 years). The criteria for dyslexic and control group inclusion (average or above average intelligence etc.) were those detailed in Experiment 1.

Method

The following words were pre-recorded on a tape recorder: 'hot,' 'big,' 'cold,' 'huge,' 'bright,' 'square,' 'high,' 'red,' 'wrong,' 'regular,' 'purple,' 'afternoon,' 'rectangular,' 'dangerous,' 'transparent,' 'elliptical,' 'miscellaneous,' and 'professional.' The first nine are short, the latter nine long.

The child was told that he was to hear some words on the tape recorder and that he was to repeat the words as quickly as he could. The onset of each word started a Dawes timer by means of a voice key, while the onset of the child's response stopped the timer by means of a second voice key whose microphone was positioned directly in front of his mouth. Errors and response times accurate to 10 ms were recorded.

It should be noted that the time taken to activate a voice key varies as a function of the initial phoneme of the word being spoken. Since the 'short' and 'long' words were not matched with respect to initial phoneme, comparison of repetition latencies between 'short' and 'long' words are not strictly legitimate.

Results

The response time data were analysed as a 3-factor ANOVA: 2 groups (dyslexics, controls) × 2 word lengths × 9 words. The groups factor ($F = 2.63$; $df = 1,24$) was insignificant, the mean response times on the 'short' words being 820 ms for the controls and 850 ms for the dyslexics, and on the 'long' words being 1010 ms for the controls and 1110 ms for the dyslexic subjects. Both the word length factor ($F = 299.8$; $df = 1,24$) and word factor ($F = 14.6$; $df = 1,24$) were significant, but there was no interaction with groups. There were hardly any mispronunciations.

Discussion

When single words were presented auditorily the dyslexic children did not differ significantly from the controls in the speed or accuracy with which they made the words articulate. Both groups were affected equally by word length. Whether this articulatory encoding is the same process as that involved in reading is a matter of debate; in adults its initiation is certainly very fast, automatic, and with practice, apparently independent of vocabulary size for nonsense syllables (Davis, Moray, and Treisman 1961). These findings may or may not apply with articulatory encoding in the postulated grapheme to lexical code to articulatory code pathway. There is, however, no doubt that the overt articulatory output is the same whether the initial input be visual or auditory, and the experiment shows that when the need to create a non-articulatory name or lexical code from graphemic stimuli is bypassed dyslexic children perform no differently from controls at articulatory encoding. This fits in with other evidence (Ellis 1980; Ellis and Miles 1980) for normal articulatory encoding in dyslexic children. We can therefore rule out a simple explanation of the name code deficit in dyslexic children and are forced to consider the more complex processes hypothesized to be involved in name coding.

The Nature of the Name-Encoding Deficiency in Dyslexia

A distinction is commonly made between at least two available strategies for reading to name. The first is an orthographic mechanism which makes use of general relations between letters or letter groups and their sounds (grapheme-phoneme correspondences), thus allowing naming by synthesis. The second is a lexical mechanism whereby knowledge of the pronunciation of whole words is utilized. Thus in both cases naming involves access to an internal phonological code, albeit by a different route. Both involve also visual analysis and articulation which have been ruled out as major sites of the deficiency under consideration. The main difference is as follows: 'In the orthographic mechanism the effective units for accessing pronunciation are letters or letter groups; in the lexical mechanism, the units are whole words or morphemes' (Baron and Strawson 1976). These theories owe much to the Morton logogen model (1969, 1977, 1979, 1980).

Crucial for this discussion is that both lexical and non-lexical naming rely on a 'naming code,' i.e., pronunciation which is internally represented has to be retrieved. This is where the dyslexic child appears to show the deficit. We would therefore predict that dyslexic children's performance on tasks involving *either* the lexical *or* the phonological route would be impaired. In fact, evidence from many different sources suggests

that this is the case. The lexical naming route may well be used for stimuli such as objects, colours, or digits, which are not alphabetic and which therefore are not open to grapheme-phoneme conversion. Similarly, the production of the names (rather than the sounds) of letters, and the naming of frequent yet irregular words (e.g., *eye*) which cannot be named correctly by application of grapheme-phoneme conversion rules, are likely to involve the lexical naming route. There is evidence that dyslexic children are deficient at all these tasks. Thus Ellis (1980) has shown that dyslexic children, who at 10-14 years old have had much exposure to the letters of the alphabet, are reliably slower than controls at naming single *letters* of the alphabet. Denckla and Rudel (1974), Spring (1976), and Ellis (1980) all give evidence of relatively slow *object* and *colour* naming in dyslexic children. When the Oldfield and Wingfield (1965) stimuli are used, the less frequent the object name, the slower the dyslexic children are at naming the object and the greater the likelihood that the former group will be unable to find the picture name at all (Ellis 1980). Ellis (1980) has shown that dyslexic children (10-14 years) are slower than control children at reading common and frequent *words* (e.g., 'bird') which are well within the children's reading vocabulary. Ellis and Miles (1978a) demonstrate dyslexic children to be slow at processing from arrays of *digits*.

The synthetic phonemic route to naming must be used when the stimuli are nonwords which, by definition, have no lexical entry. Seymour and Porpodas (1978), Baddeley et al. (in preparation), and Snowling (1980) show that dyslexic children are both slower and more error prone at reading *orthographically regular nonwords*. Similarly Firth (1972) found that dyslexic children were poor at naming nonsense words like *nate*.

Therefore, there is a wealth of evidence for a double impairment in developmental dyslexia: dyslexic children are impaired both i) in lexical access and lexical retrieval of pronunciation and ii) in retrieval of phonology by means of application of grapheme-phoneme rules. This is also the conclusion reached by Seymour and Porpodas (1978), although others (e.g., Jorm 1979; Snowling 1980) would argue that the impairment in the use of the synthetic phonemic route is considerably more severe.

It is interesting that this double-deficiency hypothesis is essentially that proposed by Marshall and Newcombe (1973) for surface dyslexic patients. It is worth noting in this context that both Holmes (1973) and A.W. Ellis (1980) reach the conclusion that the reading errors made by developmental dyslexic children are 'of a piece' with those made by surface dyslexic adults. Such similarities of symptom give support to the view of similar functional disabilities underlying the two conditions.

However, since both processes may well hinge crucially on the involvement of the same name code that is impaired in dyslexic children, we could state more parsimoniously that there is just one single deficit. This view would also fit in with more recent criticisms of dual-route models in reading (Glushko 1978. Marcel 1979, 1980). If these views are accepted then both lexical naming and a large part of orthographic naming involves lexical retrieval of phonology. This being the case, a simple description of a single functional impairment associated with develomental dyslexia can be seen in the specific name coding deficiency so clearly demonstrated by the present experiments.

Acknowledgments. The author wishes to thank Tim Miles for constant support and discussion, Alec Vardy for statistical advice, the headmasters Gerald Trump, John Walters, and Kenneth Underwood who allowed him into their schools, and Alan Allport, Alan Baddeley, Max Coltheart, and Uta Frith for helpful comments at various stages of this work. Willingness to help is not an implied endorsement.

References

Allport DA (1978a) Word recognition in reading: a tutorial review. In: Bouma H, Kolers PA, Wrolstad M (eds) Processing of visible language. Plenum, New York

Allport DA (1978b) On knowing the meaning of words we are unable to report: the effects of visual masking. In: Dornic S, (ed) Attention and performance, 6. Erlbaum Associates, Hillsdale, NJ

Audley RJ (1976) Reading difficulties: the importance of basic research in solving practical problems. Presidential Address to the 1976 BAAS meeting

Baddeley AD (1979) Working memory and reading. In: Kolers PA, Wrolstad M, Bouma H (eds) Processing of visible language. Plenum, New York

Baddeley AD, Lewis V (1979) Inner active processes in reading: the inner voice, the inner ear and the inner eye. In: Lesgold AM, Perfetti CA (eds) The Proceedings of the Pittsburgh Conference on Interactive processes in reading, September 1979. Erlbaum, Hillsdale, NJ

Baron J, Strawson C (1976) Use of orthographic and word-specific knowledge in reading words aloud. J. Psychol: Percept Perform 2:386–393

Boder E (1971) Developmental dyslexia: Prevailing concepts and a new diagnostic approach. In: Myklebust HR (ed) Progress in learning disabilities, Vol. II. Grune & Stratton, New York

Coltheart M (1972) Visual information processing. In: Dodwell P (ed) New horizons in psychology: 2. Penguin, Harmondsworth

Coltheart M (1978) Lexical access in simple reading tasks. In: Underwood G (ed) Strategies of information processing. Academic Press, London

Conrad R (1964) Acoustic confusion in immediate memory. Br J Psychol 55:75–84

Critchley M (1970) The dyslexic child. Heinemann, London

Davis R, Moray N, Treisman A (1961) Imitative responses and the rate of gain of information. Quart J Exp Psychol 13:78–89

Denckla MB, Rudel R (1974) Rapid 'automized' naming of pictured objects, colours, letters and numbers by normal children. Cortex 10:186–202

Denckla MB, Rudel R (1976) Naming of object-drawings by dyslexic and other learning disabled children. Brain Lang 3:1–15

Egeth HE (1966) Parallel versus serial processes in multi-dimensional stimulus discrimination. Percept Psychophys 1:246–252

Ellis AW (1980) Developmental and acquired dyslexia: some observations on Jorm (1979). Cognition (in press)

Ellis NC (1980) Functional analyses of reading and short-term memory in dyslexic children. Unpublished Ph.D. thesis, University of Wales

Ellis NC, Miles TR (1977) Dyslexia as a limitation in the ability to process information. Orton Soc Bull XXVII:72–81

Ellis NC, Miles TR (1978a) Visual information processing as a determinant of reading speed. J Read Res 1:108–120

Ellis NC, Miles TR (1978b) Visual information processing in dyslexic children. In: Gruneberg MM, Morris PE, and Sykes RN (eds), Practical aspects of memory. Academic Press, London

Ellis NC, Miles TR (1980) A lexical encoding deficiency I: experimental evidence. In: Pavlidis G Th and Miles TR (eds) Dyslexia research and its applications to education. Wiley, Chichester (forthcoming)

Firth I (1972) Components of reading disability. Unpublished doctorate dissertation, University of New South Wales, Australia

Gibson EJ, Gibson JJ, Pick AD, Osser H (1962) A developmental study of the discrimination of letter-like forms. J Comp Physiol Psychol 55:897–906

Glushko RJ (1978) The organization and activation of orthographic knowledge in reading aloud. Occasional papers, 1. Center for Human Information Processing, University of California, San Diego

Holmes JM (1973) Dyslexia: a neurolinguistic study of traumatic and developmental disorders of reading. Unpublished PhD thesis, University of Edinburgh

Ingram TTS (1960) Pediatric aspects of developmental dysphasia, dyslexia and dysgraphia. Cerebral Palsy Bull 2:254–277

Jorm AF (1979) The cognitive and neurological basis of developmental dyslexia: A theoretical framework and review. Cognition 7:19–33

Liberman IY, Shankweiler D, Orlando C, Harris KS, Berti FB (1971) Letter confusions and reversals of sequence in the beginning reader: implications for Orton's theory of developmental dyslexia. Cortex 7:127–142

Marcel A (1979) Surface dyslexia and beginning reading – a revised hypothesis of the pronunciation of print and its impairments. In: Coltheart M, Patterson KE, Marshall JC (eds) Deep dyslexia. Routledge & Kegan Paul, London

Marshall JC, Newcombe F (1973) Patterns of paralexia: a psycholinguistic approach, J Psycholing Res 2:175–199

Miles TR, Wheeler TJ (1974) Towards a new theory of dyslexia. Dyslexia Rev 10:9–11

Miles TR, Wheeler TJ (1977) Responses of dyslexic and nondyslexic subjects to tachistoscopically presented digits. IRCS Med Sci 5:149

Mitchell DC (1976) Buffer storage modality and identification time in tachistoscopic recognition. Quart J Exp Psychol 28:325–337

Morton J (1969) Interaction of information in word recognition. Psychol Rev 76:165–178

Morton J (1977) Word recognition. In: Morton J, Marshall JC (eds) Psycholinguistics: Series II. Elek, London

Morton J (1979) Facilitation in word recognition: experiments causing change in the logogen model. In: Kolers PA, Wrolstad M, Bouma H (eds) Processing of visible language 1. Plenum Press, New York

Oldfield RC, Wingfield A (1965) Response latencies in naming objects. Quart J Exp Psychol 17:273–281

Phillips WA (1974) On the distinction between sensory storage and short-term visual memory. Percept Psychophys 16:283–290

Phillips WA, Baddeley AD (1971) Reaction time and short-term visual memory. Psychonom Sci 22:73–74

Posner MI (1969) Abstraction and the process of recognition. In: Spence JT, Bower G (eds) The psychology of learning and motivation, Vol. 3. Academic Press, London

Seymour PHK, Porpodas CD (1980) Graphemic and lexical processing in dyslexia. In: Frith U (ed) Cognitive processes in spelling. Academic Press, London

Snowling MJ (1980) The development of grapheme-phoneme correspondence in normal and dyslexic readers, J Exp Child Psychol

Sperling G (1967) Successive approximations to a model for short term memory. Acta Psycholog 27:285–292

Spring C (1976) Encoding speed and memory span in dyslexic children. J Spec Educ 20:35–46

Spring C, Capps C (1976) Encoding speed, rehearsal, and probed recall of dyslexic boys. J Educ Psychol 66:780–786

Stanley G, Hall R (1973a) Short-term visual information processing in dyslexics. Child Develop 44:841–844

Stanley G, Hall R (1973b) A comparison of dyslexics and normals in recalling letter arrays after brief presentation. Brit J Educ Psychol 43:301–304

Supramanian S, Audley RJ (1976) The role of naming difficulties in reading backwardness. Paper presented to the British Association Annual Conference.

Townsend JT (1971) Theoretical analysis of an alphabetic confusion matrix. Percept Psychophys 9:40–50

Vellutino FR, Smith H, Steger JA, Kaman M (1975) Reading disability: age differences and the perceptual-deficit hypothesis. Child Develop 46:487–493

Vellutino FR, Steger JA, Kandel G (1972) Reading disability: an investigation of the perceptual deficit hypothesis. Cortex 8:106–118

Vernon MD (1971) Reading and its difficulties. CUP, Cambride

Vernon MD (1977) Varieties of deficiency in the reading process. Harvard Educ Rev 47:396–410

Vernon MD (1979) Variability in reading retardation. Br J Psychol 70:7–16

Wickelgren WA (1965) Acoustic similarity and intrusion errors in short-term memory. J Exp Psychol 73:165–171

Wheeler TJ (1977) A study of information processing in dyslexic subjects. Unpublished PhD thesis, University of Wales