Dyslexia: Challenging Theories

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Summary

Experiments presented challenge theories on their ability to provide causal explanations of the pattern of performance in dyslexia. Studies 1a and 1b employed a prism adaptation paradigm to investigate the Cerebellar Deficit Hypothesis (CDH). No group differences were found, although unfortunately it was concluded that the paradigm could not satisfactorily isolate cerebellar function from other compensation mechanisms. Studies 2a and 2b exploited a sequential stereopsis technique to test the visual deficit hypothesis. No group differences were found, although the dyslexic group did exhibit a fatigue effect on one condition. Using an attention shifting paradigm, Study 3 found a dissociation between focus and shift attention conditions in dyslexic children, but that they sustained their attention as well as controls. In Study 4, supporting the Dyslexia Automatisation Deficit (DAD) as opposed to a general resources deficit, control performance suffered most under visually degraded conditions of the same attention paradigm. Study 5 further investigated attention on a test thought to be sensitive to attentional lapses; dyslexic children did make more errors, although conclusions were limited by their qualitatively normal performance.

Deficits in dyslexia were found to be wider reaching than many theories of dyslexia would suggest. At a cognitive level of explanation the DAD was able to account successfully for many of the findings. However, like the Phonological Deficit Theory the DAD specifies no neurological mechanism for the deficit; this is provided by the CDH (for which no evidence was found here). Analyses do point towards the need for either a very general explanation, or the identification of a smaller number of core deficits, for the apparently disparate deficits found. The fatigue effect found only in the dyslexic group on part of the vision experiment has further direct and immediate implications for future research.

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Contents

1. Introduction	
1.1 Definition and Diagnosis of dyslexia	1
1.2 Visual Deficit Theory	7
1.3 Phonological Deficit Theory	8
1.4 Rapid Temporal Processing Deficit Hypotheis	14
1.5 Dyslexia Automatisation Deficit Hypothesis	21
	21
1.6 Cerebellar Deficit Hypothesis	
1.7 Attention Deficit?	27
1.8 Scope of thesis	31
2 Driam Adaptation in Duplayie	
2. Prism Adaptation in Dyslexia	25
2.1 Prism Adaptation	35
2.2 Individual Differences in Adaptation	39
2.3 Rationale of experiments	40
2.4 Experiment 1: Straight Ahead Shift Experiment	42
2.5 Experiment 2: Prism Adaptation	50
2 Winnel Definite in Devile in 9	
3. Visual Deficits in Dyslexia?	(0)
3.1 Introduction	68
3.2 What is sequential stereopsis?	76
3.3 Experiment 1: Binocular Control	79
3.4 Experiment 2: Automatisation of Binocular Control	96
3.5 Pooled Data	103
3.6 Experiment 2 and general discussion	104
3.7 Further examination of Enright's theory	108
3.8 Summary and conclusions	111
4. Attention Shifting Deficits in Dyslexia?	
4.1 Attention	113
4.2 Selective vs sustained attention in ADHD and dyslexia	115
4.3 Reorientation and direction of attention	120
4.4 The Experiment	123
• • • • • • • • • • • •	
5. Attention, Resources and Dyslexia: a further look	
5.1 Introduction	143
5.2 The Experiment	146
6. Performance of children with dyslexia on the SART	
6.1 Introduction	174
6.2 The Experiment	177
7. General Discussion and Conclusions	
7.1 Issues addressed	191
7.2 Summary of results	192
7.3 Limitations of research and future directions	201
7.4 Summary and conclusions	210
8. References and Appendices	
References	213
Appendices	238

Chapter 1 General Introduction

Introducing the major theories of dyslexia and the foci of this thesis

Summary:

This chapter presents a general introduction to dyslexia. Definitions and methods of diagnosis are discussed, before introducing the current major theories of dyslexia, their supporting evidence and their limitations in terms of providing a causal explanation of dyslexia. The aim of the thesis is to evaluate the different theories of dyslexia with regard to their abilities to account for disparate evidence. In order to fulfil this aim, novel evidence is presented throughout the thesis to throw further light on the theories and to present new and testing challenges for them. In this chapter, a brief overview of the experiments to be presented is given as well as explaining how the various and wide-ranging ideas for these experiments arose.

1.1 Definition and diagnosis of dyslexia

Kussmaul (1877) first introduced the concept of 'word-blindness' claiming that 'a complete text-blindness may exist even though the power of sight, the intellect, and the power of speech are intact". Following Kussmaul's report, several more case studies were reported, sometimes involving loss of one alphabet, but leaving another intact (see e.g. Michel, 1892; Charcot, 1892, both cited in Hinshelwood, 1895). Hinshelwood (1895) wrote about word blindness in terms of visual memory and described one of his patients who was even unable to read what he himself had written, therefore gaining no help from vision in the spelling of the words and the formation of the letters. He expressed surprise at the fact that the patient could read figures with ease, but had so much difficulty with letters. Mierzejewski (1892; cited in Hinshelwood, 1895), however, was undoubtedly one of the first to describe a form of word-blindness he called "caecitas syllabaris et verbalis, sed non litteralis". His patient was able to recognise individual letters, but could not unite them into syllables and words. Badal (1888), also cited in Hinshelwood (1895), described a similar case, where highly familiar words, such as the patient's own name, were recognised and pronounced, whereas the patient could not combine even the simplest syllables of novel words. Badal explained this phenomenon in terms of familiar words forming a 'graphic unity', whereas for unfamiliar words the *relationship* between letters needed to be resolved.

The concept of 'congenital word blindness' followed an increasing number of reports of word-blindness in adults. In 1900, Hinshelwood (cited in Miles and Miles, 1990) commented that "I have little doubt that these cases of congenital word blindness are by no means so rare as the absence of recorded cases would lead us to infer". He further stated that "Their rarity is, I think, accounted for by the fact that when they do occur they are not recognised." and that "It is a matter of the highest importance to recognise the cause and true nature of this difficulty in learning to read which is experienced by these children, otherwise they may be harshly treated as imbeciles or incorrigibles, and either neglected or punished for a defect for which they are in no wise responsible."

Orton (1937), cited in Miles and Miles (1990), suggested that the term congenital word blindness was misleading, since there was no blindness (for words or otherwise) in the ordinary sense of the word. Instead he proposed the term *"strephosymbolia"* (twisting of symbols) and believed that this was a hereditary condition, often associated with unusual patterns of 'handedness' and 'eyedness'. He also talked about 'letter reversals' (e.g. confusing b and d) and 'kinetic reversals' (e.g. reading 'was' as 'saw'). Letter reversals are now recognised as classic signs of dyslexia and the link between these and unusual 'eyedness' patterns has since been further explored (see section 3.1 for further discussion).

What is now usually called 'developmental dyslexia' is now recognised as the most common of the developmental disorders, thought to affect around 5% of the population (e.g. Jorm, Share, Maclean and Matthews, 1986). The World Federation of Neurology (1968) define dyslexia as:

"a disorder in children who despite conventional classroom experience, fail to attain the language skills of reading, writing and spelling commensurate with their intellectual abilities".

It is a specific learning difficulty usually diagnosed when there is a discrepancy of at least 18 months between reading age and chronological age, with no immediately apparent cause in terms of deprivation, emotional difficulties or general low intelligence. Typical manifestations include bizarre spelling, reading problems, reversal of letters, confusion between left and right, problems in recognising words that are already known and difficulty in simple copying. However, neither the general definition nor the definition used for diagnosis includes such manifestations, and the pattern of difficulties actually extends much further than this (see e.g. Miles, 1983; Fawcett and Nicolson, 1994).

Dyslexia is most commonly considered as a failure in reading and writing in relation to *intellectual level*. Children are excluded if they have low IQ, socioeconomic disadvantages, emotional problems, sensory deficits or neurological damage. Using intellect in this way results in excluding those who may have reading difficulties, but who also have below average intellect; this prevents them from getting the specialist help that they need. It also excludes those with emotional difficulties or social deprivation *in addition* to dyslexia and those who have emotional difficulties *because* of dyslexia. Furthermore, and perhaps most importantly, it relies on waiting for children for dyslexia to fail before offering help! Those who achieve an average or above average level of performance through hard work are therefore also excluded because although it is likely that they never reach their full potential, they may also never fail. Thus, exclusionary definitions of dyslexia have led to much controversy and dispute (e.g. Applebee, 1971; Stanovich, 1991;1996; Siegel, 1992; Miles, 1996; Siegel and Himel, 1998).

Unfortunately, at present the criteria themselves are mostly exclusionary. However, in recognition of the impact which the phonological deficit hypothesis (see below) has had on dyslexia research and remediation, the Orton society definition now includes reference to the phonological difficulties experienced by many dyslexic children.

Dyslexia is a neurologically-based, often familial, disorder which interferes with the acquisition and processing of language. Varying in degrees of severity, it is manifested by difficulties in receptive and expressive language, including phonological processing, in reading, writing, spelling, handwriting and sometimes in arithmetic.

(Orton Society, 1995)

One reason for over-reliance on exclusionary definitions is that it seems likely that there are *several* causes and consequences of dyslexia. For dyslexia to be a syndrome, people diagnosed as dyslexic should have much in common with regard to their abilities and deficits and relatively few differences; they should form a homogenous group (or at least homogenous subgroups). Miles (1994) offers a useful discussion about 'lumping' people together on certain characteristics and 'splitting' them on the basis of others. Once either lumped or split, similarities within groups, but differences between groups, tend to be emphasised. Miles suggests the analogy of applying pressure to a rock in order to break it into smaller pieces; if pressure is applied along a fault line the resultant chunks are meaningful to study, else pressure in other places will result in disconnected fragments. He suggests that the concept of poor reading would be drawing the boundaries in the wrong place, with several poor readers not being dyslexic (see e.g. Stanovich, 1988) and others displaying other symptoms associated with dyslexia but having adequate reading. In addition, poor reading would not fully characterise the full range of difficulties experienced by people with dyslexia. Furthermore, dyslexia should be seen as a persistent condition (whether compensated for and partially overcome or not) and one would expect the cause of the problems (whether known or not) to be similar in each case. However, children with dyslexia often do not conform to such criteria. Nonetheless, distinctive profiles have been found in many dyslexic children in comparison to their controls, most notably the consistent findings of phonological deficits. Using such positive signs of dyslexia might therefore be a more effective and accurate way of identifying the syndrome in comparison with exclusionary definitions. This would not be to say however, that such positive signs are necessarily causal of dyslexia.

However, until research does provide valid, reliable and accepted positive inclusionary signs of dyslexia, more attention could be given to the idea of looking for a discrepancy between listening and reading comprehension (as did for example Spring and French, 1990). In addition to Applebee's (1971) criticisms of placing too much emphasis on potential variables which may impact on achievement, such as intelligence and social class, Siegel (1989) criticises all IQ-achievement discrepancy definitions on the basis of four main assumptions that they make. These are; that IQ measures intelligence, that intelligence and achievement are independent, that reading is usually predicted by IQ scores, and that dyslexic children with discrepant IQ scores will have different skills from those who are just not very good at reading because they have low IQ scores. Stanovich (1996) argues along similar lines, noting that, "the poor reading of low IQ individuals is not explained by their low IQ" and that "we need a specific processing explanation" (see also Miles, 1996; Siegel and Himel, 1998). Looking for a discrepancy between listening and reading comprehension would therefore appear to have more face validity. Indeed, listening comprehension does correlate more highly with reading comprehension than (even verbal) IQ (Caroll, 1977 cited in Stanovich, 1991). However, Nicolson (1996) argues that anti-discrepancy theorists conflate research on: the cause of poor reading vs. research on the cause of dyslexia, phonological deficit as cause of dyslexia vs. symptom of dyslexia, and research on causes of dyslexia vs. research on diagnosis and remediation of dyslexia. With regard to the question of IQ, Nicolson argues that just because phonological deficits are associated with poor reading in both dyslexic children and in children with low IQ it does not mean that the initial cause of the phonological deficits are the same.

In adults, the diagnosis of dyslexia *cannot* rely on a reading age discrepancy and so is yet more complicated. Reading and spelling ages using standard normed tests do not go beyond around 17 years, and thus a discrepancy definition in terms of years is usually inappropriate. For example, if a thirty year old has a spelling age of 16 or 17 years (for example 9 errors out of 50 words on the WORD test), rather than '17 years and above' (up to 8 errors on the WORD test), is this a discrepancy worthy of attention? Moreover, many adults may have reached this level of performance through a great deal of hard work, but still have difficulties learning to spell new words together with fluency deficits spelling words correctly under time pressure. McLoughlin (1994, p19) discusses the use of discrepancy definitions in adulthood and list some typical adult dyslexic characteristics which may be more informative than looking for a discrepancy.

Definitions and Diagnosis of participants in this thesis

All the dyslexic participants used in this thesis (both children and adults) were diagnosed as dyslexic by an appropriately qualified psychologist. For the children used in Chapters 2, 4, 5 and 6 this meant that their IQ was 90 or above (on the WISC-III, Wechsler, 1976) and their reading age (Wechsler Objective Reading Dimension single word reading) at least 18 months behind their chronological age at time of diagnosis. In addition, they showed no signs of emotional, behavioural or socio-economic problems or Attention Deficit Hyperactivity Disorders on the DSMIIIR (American Psychiatric Association, 1987), where a score of at least 8 out of 14 markers of the disorder is required for clinical diagnosis. This is important if investigating pure dyslexia, unconfounded by other factors. These participants' reading and spelling ages (WORD tests) were retested usually at the time of the experiment, or else had been tested within the previous 3 months. In most cases, IQ scores, because of the time and expertise involved in obtaining them, were taken from the last update of diagnosis.

For the dyslexic participants used in Chapter 3, students were diagnosed by an appropriately qualified psychologist using the method described by Fawcett and Nicolson (1998a) for adult diagnosis. This system uses spelling age, nonsense word reading and 'ACID profile' measures as well as taking into account any previous diagnosis of dyslexia. Spelling age is tested using the WORD test of spelling (as in children), with spelling ages below 16 years being evidence of dyslexia. A nonsense word passage presents difficulties for many dyslexic adults who have managed to overcome and compensate for their difficulties in other areas. It taps phonological skills in particular. The nonsense word passage used is that of Finucci, Guthrie, Childs, Abbey and Childs (1976). They suggested a nonsense word 'Jabberwock' reading passage to be of use in assessing adults, finding that on their passage, adults with dyslexia tended to make more than 7 errors and took longer than 59 seconds to read the passage, whereas controls managed to read the passage both faster and more accurately. The third component of the method is the ACID profile. This describes a pattern on the IQ test where the Arithmetic, Coding, Information and Digit Span subtests are significantly lowered (more than 3 scaled score points) in comparison to the other subtests. This pattern is thought to reflect memory difficulties and has been found to exist in dyslexic children and children with ADHD (e.g. Ackerman, Dykman and Peters, 1976). Assuming IQ stays constant throughout the lifespan, then the ACID profile can also be used as an adult indicator of the presence of dyslexia using the WAIS-R IQ test. Previous diagnosis of dyslexia is considered because more severe cases of dyslexia would be expected to have been noticed before reaching adulthood (although it is noted that this is not necessarily the case for mature students). In addition, it is considered that somebody who has already been diagnosed as dyslexic once will remain dyslexic even if, through hard work, they will show no signs of difficulty in their current functioning. There are therefore some complications in reaching a consensus on the best methods of diagnosis for dyslexia. This is, in part, because the cause of dyslexia is not agreed upon. As with most biological syndromes, knowing the cause would allow development of an objective and accurate test, most probably related to a biological marker.

Cause of dyslexia

This thesis concentrates on investigating the most likely cause of dyslexia. This chapter introduces six major theories of dyslexia, outlining the current evidence supporting the theories as well as evidence which they have difficulty accounting for. The foci of the experimental chapters are then presented together with a table

indicating for which theories the experiments may have implications. Frith (1997) postulates that a truly causal explanation of dyslexia should be able to link observed behaviour to cognitive anomalies and then back to neurology. A causal theory of dyslexia should therefore be able to provide (i) neurophysiological evidence for the deficit (e.g. from brain imaging techniques or ERP), (ii) a coherent link between the deficit and the manifest symptoms, (iii) behavioural evidence of the deficit, (iv) evidence that the deficit is a precursor to the behavioural signs and (v) an explanation of how previously unrelated phenomenon actually derive from a common underlying source. A true causal theory should explain not only the symptoms, but also the underlying cause of those symptoms. As already discussed, Nicolson (1996) argues that some researchers conflate research on the cause of poor reading versus research on the cause of dyslexia and phonological deficit as cause of dyslexia versus symptom of dyslexia. A true causal theory should link biological explanation with cognitive deficits and then to behaviour. Below the major current theories of dyslexia are outlined (in order of their main development periods) and their abilities to account for the different levels of Frith's framework discussed.

1.2 Visual Deficit Hypothesis: Oculomotor deficit

Dyslexia was originally conceived of as a visual problem and anecdotal descriptions of symptoms often included visual difficulties (such as letters floating above the page, letter reversals and letters moving over one another). Hinshelwood (1895) first suggested that dysfunction of the visual system could lead to dyslexia and as early as 1943 it was suggested that a disturbance of binocular vision could be influential in causing reading disability (Park and Burri, 1943).

Various aspects of vision in dyslexia have now been investigated (see e.g. Evans, 1998; Evans and Drasdo, 1990; Evans, Drasdo and Richards, 1996; Kulp and Schmidt, 1996a; Lennerstrand and Ygge, 1992; and Simons and Grisham, 1987 for reviews), although results have often been inconsistent. For example, as discussed further in Section 1.4, research relating to dyslexia and differing 'visible persistence' (as measured by the time interval between two stimuli necessary to detect a gap) was originally refuted due to seemingly contradictory findings and failures to find differences. However, this was later claimed to be due to important differences in apparatus. Investigations of oculomotor ability in dyslexia have suffered similar problems of replication and methodology. For example, occasionally oculomotor performance has only been measured monocularly (e.g. Pavlidis, 1980), or sometimes rather subjective methods have been used (e.g.

Bedwell, Grant and McKeown, 1980). Such difficulties have considerably weakened arguments for a visual deficit and have given yet further strength to phonological theories of dyslexia where deficits appear to be predictive, persistent and consistent. However, a plethora of research examining visual aspects of dyslexia does exist, from sources including ophthalmic, education and psychology journals [for example, Evans' review (1998), targeted at the practising optometrist, and Kulp and Schmidt's review (1996a) both cite over 120 references, many of which are different]. Furthermore, differences have been found in many areas including accuracy of saccades, vergence control and general binocular coordination. Stein and colleagues in particular have found a great deal of evidence for a deficit in binocular vergence control (see e.g. Stein, 1994), as discussed further in Chapter 3. A visual deficit may therefore be able to account for some, if not all, of the symptoms shown in dyslexia. Crucially, it fails to account for phonological, speed or motor skill deficits. In terms of the cause of any visual deficit, Stein and Walsh (1997) suggest the posterior parietal cortex (PPC) as a potential site to account for both temporal processing (section 1.4) and eye movement abnormalities, although little neurophysiological evidence for this exists. It is now generally accepted that a visual deficit alone cannot account for the pattern of difficulties exhibited in dyslexia.

1.3 The Phonological Deficit Theory

The phonological deficit theory (e.g. Snowling, 1987; Stanovich, 1988; Vellutino, 1979), suggests that dyslexic children have difficulties in reading and spelling due to impairment of phonological processing abilities (e.g. their knowledge of rhyme, alliteration, and grapheme-phoneme correspondences; Bradley and Bryant, 1978). It became the most prominent theory of dyslexia (in preference to visual deficit explanations) and was supported by deficits in skills such as naming speed (e.g. Denckla and Rudel, 1976) and non-word repetition (e.g. Snowling et al, 1986). Furthermore, Ellis (1981) showed directly that dyslexic reading problems cannot be attributed to visual difficulties (or at least not visual difficulties alone) in his experiment which compared dyslexic and control children's speed of same/ different letter matching judgements. Ellis found that dyslexic children were slower at matching pairs which required name encoding (e.g. Aa and Bb), but not visually identical pairs (e.g. AA and bb).

Although an association between reading ability and phonological skill has been generally accepted, in the past there has been some doubt over any causal relationship, or whether a common third factor is involved. A lack of reading ability could cause a decreased phonological knowledge (or vice versa) or an unrelated factor could affect both skills independently. However, Bradley and Bryant (1985) found that ability on a sound categorisation task at four years old (before learning to read) accounted for 4-10% of the variance in reading ability and 6-10% of the variance in spelling ability four years later (after controlling for background factors). Children given training in sound categorisation (in the same study) also performed better in later reading tests, suggesting a causal role. Rack (1994), however, offers criticism of this study because the sound categorisation training was only successful when integrated with letter knowledge training. One could argue therefore that in effect the children were being taught to read. Furthermore, this categorisation task has been criticised for being a better measure of memory than of phonological skills (Wagner and Torgesen, 1987). However, they are known to covary; Jorm, Share, Maclean and Matthews (1984) for example found that the memory abilities of 5-year-olds were predictive of later success in reading.

Lundberg, Frost and Peterson's (1988) study offers stronger support for a causal role of phonological skill on reading ability. In this study, the children in kindergarten trained in pure phonological skills performed significantly better in reading and spelling in later schooling. Moreover, since the group given no training scored higher in maths tests, the extra training was obviously specific to reading and did not cause a global effect. The study does not show that the reverse causal relationship cannot also hold true, however, (i.e. that improvement in reading improves in particular phonemic awareness) and there is some evidence suggesting that this could also be the case (see Morais, 1991). Further support for the centrality of the phonological deficit in dyslexia, comes from Fawcett and Nicolson (1995a) who find that such deficits persist in older dyslexic children, at least up until the age of seventeen. All three dyslexic groups (aged 8, 13 and 17 years) performed less well on tests of sound categorisation and phoneme deletion than both their chronological and their reading age controls.

The phonological theory of dyslexia explains short-term memory deficits observed in dyslexia as being a result of inefficient phonological coding. Baddeley (1966) found that recall from short term memory decreased with phonologically confusable (rhyming) letters in comparison with non-rhyming letters, suggesting that phonological information is used in short term memory tasks. Shankweiler, Liberman, Mark, Fowler and Fischer (1979) found that this effect was less strong in dyslexic readers, possibly suggesting less use of phonological coding.

9

Furthermore, Nicolson (1981) found that the observed improvement in children's memory span with age could be explained completely by the increase in processing and articulation speed. However, Hulme (1997) found that reading rate had a strong relationship with recall (similar to the relationship found by Baddeley, Thompson and Buchanan, 1975), although the relationship was different for words and non-words; fewer non-words were recalled even when reading rate was equal. Hulme (1997) suggested that it is because memory and reading both depend on access to phonological representations and noted that the speed element alone cannot fully account for the data. Similarly, long term difficulties with memory are explained as a consequence of the short-term memory difficulties. Bauer and Emhert (1984) found a reduced primacy effect in dyslexic children, suggesting less efficient transfer from short- to long-term memory rather than a long-term memory deficit *per se*.

Thus, once again it can be seen that memory and phonological skill have a strong relationship, and whereas proponents of the phonological deficit theory would claim that short term memory deficits result from phonological coding inefficiencies (e.g. Done and Miles, 1978; Ellis and Miles, 1978; Hulme, 1997), others might argue that difficulties on phonological tasks such as non-word repetition result from memory difficulties (e.g. Torgesen, 1978). Torgesen (1979) offers a brief review of the demands reading places on various aspects of short term and working memory and discusses the possible relationships between memory and reading. LaBerge and Samuels (1974) suggest that differences in memory skills may underlie individual differences in automatising visual recognition of words and thus postulate that some aspect of memory is essential to reading. The other alternative is that another factor may lead to both memory and reading difficulties. One possibility for such a factor is processing efficiency (however, this may include, but is not necessarily or specifically, phonological coding efficiency; and thus phonological difficulties may lead to both memory and reading difficulties). However, Senf and Freundl (1972) (cited in Torgesen, 1979) suggest that attention might account for the memory differences obtained in their study and LaBerge and Samuels emphasise the importance of attention in the early (pre-automatic) stages of learning to read. Similarly (as discussed in more detail in Chapter 4), some researchers have used measures of attention which might more generally be considered as measures of memory (e.g. Tarver and Hallahan, 1974)! Torgesen (1979) goes on to criticise memory measures which require organisation through verbal labels (e.g. Ritchie and Aten, 1976) and those which fail to consider differences between recall and recognition (e.g. Noelker and Schumsky, 1973) or fail to satisfactorily equate general task difficulty (e.g. Mason, Katz and Wicklund, 1975). Torgesen also presents evidence suggesting that successful encouragement of verbal rehearsal in dyslexic children eliminates group differences (Torgesen and Goldman, 1977), that organisation and 'metamemory' may have a large role to play in differences (Torgesen, 1979) and that memory strategies which are used may be related to naming speed. Miles and Wheeler (1974) also contend that the main difficulty in dyslexia is "*an inability to retain <u>complex</u> information over time*" (cited in Thomson and Wilsher, 1978: my underlining). In summary, the thrust of the argument suggests that rather than a deficit in memory capacity, dyslexic children may suffer from a deficit in strategy, which in turn may be related to other factors. With respect to the question of an attentional deficit, this will be considered further in Chapter 4. However, if such a deficit would be considered to encompass an 'attention management' deficit (Perfetti and Goldman, 1976) and attention is equated with processing activity (e.g. Kahneman, 1973) then attention might be considered a contender!

More recently, evidence for the phonological deficit theory has occasionally made attempts to relate phonological deficits to brain function. Ackerman, Dykman and Oglesby (1994) examined visual event-related potentials of dyslexic, attention deficit disorder and reading age control children to rhyming and non-rhyming stimuli. In a similar experiment, Rugg (1984a, 1984b) showed that rhyming and non-rhyming trials differed in adults on the N450 brain wave (a negative component occurring 450 msecs after the stimuli). Rugg suggested that the N450 may be similar to the N400, commonly associated with surprise/ incongruous information and that its presence in non-rhyming trials may be because the first stimulus 'primes' candidate rhymes. Thus, Ackerman et al (1994) hypothesised that the dyslexic group would not show rhyme/ non-rhyme ERP modulation effects as strong as the other groups. This was confirmed, with dyslexic groups showing non-significant enhancement of the N450 wave under non-rhyming conditions. together with the finding of a more pronounced P500 component (positive wave 50msecs later). This positive wave was thought to reflect either further processing, or to be larger because the preceding negative wave was smaller. In addition, functional imaging studies have shown differences between dyslexic and control brain activation (in numerous areas) during various phonological and/or language tasks (e.g. Paulesu, Frith, Snowling, Gallagher, Morton, Frackowiak and Frith, 1996; Hagman, Wood, Buchsbaum, Tallal, Flowers, Katz, 1992; Gross-Glenn, Duara, Barker, Loewenstein, Chang, Yoshii, Apicella, Pascal, Boothe, Sevush, Jallad, Novoa and Lubs, 1991). However, it seems that such findings are unlikely

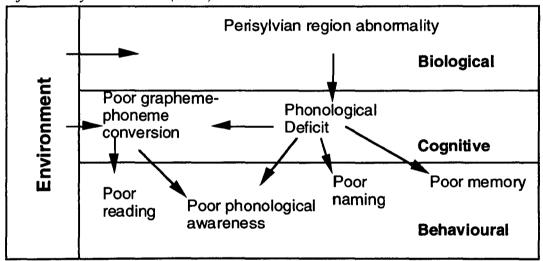
to reflect the cause of the deficit, but rather are a consequence of it. Furthermore, additional methodological difficulties occur when participants perform with different degrees of accuracy on the task itself, because one might then plausibly expect differential brain activation for success and failure whether or not anatomical differences or processing related differences are present.

Convincing evidence for an anatomical *cause* of verbal deficits of some kind comes from direct investigation of dyslexic and control brains. Neuroanatomical findings have included absence of the usual asymmetry in the planum-temporale (part of the temporal lobe thought to make up a portion of Wernicke's speech area) and minor malformations in and around Broca's area and the perisylvian fissure; e.g. Galaburda, Sherman, Rosen, Aboitiz and Geschwind, 1985; Humphreys, Kaufman and Galaburda, 1990; Galaburda, 1989. Such findings have also been confirmed using Magnetic Resonance Imaging (MRI); Hynd, Semrud-Clikeman, Lorys, Novey and Eliopolus, 1990; Larsen, Höien, Lundberg and Ödegaard, 1990. However, the precise site for a phonological processing deficit in the brain is still far from clear, and until potential sites are specified at least in terms of whether they should relate to motor, sensory or association processes of language, progress is likely to be slow.

Figure 1.3.1 outlines the proposed cause of dyslexia according to the proponents of phonological deficit theory, as illustrated by Frith (1997). An abnormality in the perisylvian region is the most probable initial cause at the biological level (e.g. Galaburda, 1989; Paulesu, Frith, Snowling, Gallagher, Morton, Frackowiak and Frith, 1996). This abnormality is then said to cause the phonological deficit (cognitive level), although the environment may protect against this. The phonological deficit leads to poor grapheme-phoneme conversion (cognitive level). This poor grapheme-phoneme conversion leads to poor reading and (together with the phonological deficit) poor phonological awareness. The phonological deficit is also said to result in poor naming skills and poor memory (behavioural level).

There is therefore an abundance of evidence supporting the phonological deficit theory, although the exact role and mechanisms of phonological processes in reading development in people with and without reading difficulties is not fully understood. There is evidence suggesting that lack of phonological awareness may be a necessary but not necessarily a sufficient, or an independent, criterion for dyslexia. Lovegrove (see 1992), for example, has found visual deficits even with stimuli requiring no verbal processing at all (see section 1.4). Nicolson and Fawcett (1995) tested dyslexic and matched control children on a battery of 'primitive skills' tests and found deficits in most skills, including motor, phonological, and balance tasks (see section 1.5). Smith-Spark (1997) found memory deficits in dyslexic adults even on a non-verbal task. Furthermore, the PDH predicts difficulties only with phonological aspects of spelling, although other aspects are impaired, and does not account for handwriting problems at all. Moreover, there is some debate over whether phonological deficits are specific to dyslexic children. Slow learners have been found to show very similar difficulties (see e.g. Ellis, McDougall and Monk, 1996a; Stanovich, Siegel and Gottardo, 1997). Furthermore the theory fails to provide a well-specified *cause* for the phonological deficit, with the biological level of explanation having been somewhat neglected over the years.

Figure 1.3.1 Causal diagram of reading problems according to the phonological deficit theory. From Frith (1997)



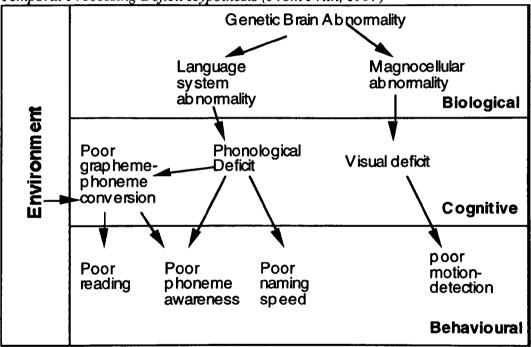
In summary, the phonological deficit theory of dyslexia is the most widely accepted and most widely investigated theory of dyslexia. It offers neurophysiological (and also some anatomical) evidence of a phonological deficit. Furthermore, phonological deficits are both consistently found in dyslexia and do appear to predate reading difficulties. The theory is also able to account for memory difficulties shown, however it fails to explain the full range of deficits exhibited by children with dyslexia, particularly with motor skills and speeded performance¹. The Rapid Temporal Processing Deficit Hypothesis can account for some, but not all, of these shortcomings.

¹Although Stanovich (1986; 1988) suggested that poor readers may be victims of a 'Matthew Effect' (...from him that hath not shall be taken away...) in that an initial processing difficulty results in difficulties in other areas.

1.4 Rapid Temporal Processing Deficit Hypothesis

The Rapid Temporal Processing Deficit Hypothesis (RTPDH) postulates that children with dyslexia have difficulty processing anything quickly. The first evidence which suggested difficulty in rapid performance generally (for production) came from Denckla and Rudel (1976). They found that children with dyslexia had difficulty in 'rapid automatised naming' of stimuli (even non-word stimuli). Further evidence from Nicolson and Fawcett (1994) found speed deficits in a choice reaction time paradigm. However, findings of these broad difficulties in rapid performance were preceded by deficits in rapid (perceptual) processing in visual and auditory modalities. Frith (1997) provides the causal diagram of reading difficulties according to the RTPDH see in Figure 1.4.1.

Figure 1.4.1. Causal diagram of reading difficulties according to the Rapid Temporal Processing Deficit Hypothesis (From Frith, 1997)



Visual modality: transient system deficit hypothesis

The visual deficit hypothesis of dyslexia was first refuted because of; consistent failures to find differences on a wide range of tasks, seemingly contradictory findings, and non-correlation between severity of dyslexia and several visual tasks (Lovegrove, Martin and Slaghuis, 1986). It was therefore assumed that the principal cause of dyslexia was *not* a visual one. The rise of the phonological

deficit theory was rapidly accepted by researchers and clinicians as the most useful and most valid theory.

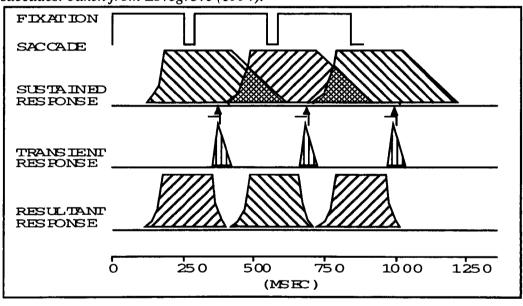
Tolhurst (1975) suggested a possible mechanism to explain earlier inconsistent findings and a framework for future research. He suggested two types of visual channels in human vision; the sustained and the transient. Subsequent research suggested that the sustained system signals colour and detail but has little sense of timing of stimuli. In addition, it is less sensitive to contrast, more sensitive to high spatial but low temporal frequencies, has slow transmission times, responds throughout the stimulus presentation and predominates in central vision. It is thought that it may inhibit the transient system (Breitmeyer and Ganz, 1976). In contrast, the transient system signals the timing of events and basic spatial information. It has been shown to be highly sensitive to contrast, low spatial and high temporal frequencies, to have fast transmission times, respond only at stimulus onset and offset and predominate in peripheral vision.

The concept of sustained and transient systems offers a possible explanation of why visual deficits were originally not consistently found in dyslexic children. Research has indicated that dyslexic children may have a deficit only on the transient channel. Thus, differences between dyslexic and control children are likely to be found only when stimuli have properties principally detected by the transient system; low spatial frequency, high temporal frequency and so on. However, original inconsistencies in research were also present even when spatial and temporal frequencies were taken into account.

Howell, Smith and Stanley (1981), like Lovegrove, Heddle and Slaghuis (1980) used visual persistence durations (with different spatial frequencies) as an indication of the activity of the transient system. Visual persistence is a measure of how long an image is still seen by the individual after it has been removed (not the same as iconic memory or afterimages: Di Lollo, Clark and Hogben, 1988). It can be measured by rapidly presenting two identical stimuli with a blank stimulus between them. If the blank is not seen by the participant, the previous stimulus must have 'persisted', or lasted, up until the final stimulus was presented. Carrying this procedure out at different speeds can establish the length of any individual's 'visual persistence'. The length of persistence is assumed to be determined by the speed of the transient system, since it is this system that probably inhibits the sustained system (which responds to details of the stimulus).

Unlike Lovegrove and his colleagues, Howell et al (1981) found no differences in visual persistence between control and dyslexic children. Howell et al (1981) proposed that the discrepancy between the two sets of results may have been due to differences in apparatus, Lovegrove (1980) using a tachistoscope (and thereby creating flicker) and Howell et al (1981), using an oscilloscope. The discrepancy could have been due to the role of flicker in the transient system. Physical flicker from the tachistoscope (as the slides went round) may have increased the involvement of the transient system, which is more effective and efficient in control children (particularly at low spatial frequencies), which may therefore have led to exaggerated differences between dyslexic and control children.

Figure 1.4.2. A hypothetical response sequence of sustained and transient channels during three 250 millisecond fixation intervals separated by 25 millisecond saccades. Taken from Lovegrove (1994).



Assuming that the transient system does indeed suppress the sustained system during eye movements, a deficit may impair reading fluency by superimposing words onto one another. Figure 1.4.2 shows a hypothetical and schematic diagram of how the sustained and transient channels might be expected to work together. The sustained system examines the high spatial frequency characteristics of each word and is activated during fixation periods. The transient system is activated following saccades and is thought to inhibit the sustained system. In a well functioning system, the resultant response is clearly separated periods of being able to see the detail of each word in turn. A finely tuned system is therefore required to use to the transient system to find the next word and then to engage the sustained system for brief periods in order to actually examine the (high spatial frequency) letters. Fixation time on each word is estimated to be around 250msec with saccades taking around 25msec (Lovegrove, 1994). The strongest interaction effects between detection of different spatial frequencies and group (dyslexic or control) are at around 300msec; therefore very close to the duration of fixation during reading. The transient system is necessary to (a) find the next word, (b) allow the sustained system to examine the detail of the word, (c) inhibit the sustained system and (d) move onto the next word. It is stage (c) that appears to be the crucial one in dyslexia. If the transient system is not rapidly or sufficiently strongly enough activated to inhibit the sustained system, then the detail of the first word may be superimposed onto the next. This would result in a jumble of unrecognisable characters, similar to reported symptoms of some children with dyslexia² (see e.g. Lovegrove, 1994; Cornelissen, Evangelinou, Hansen and Stein, 1997 and Figure 1.4.3).

Figure 1.4.3. What might happen if the transient system does not inhibit the sustained system properly during saccades. Taken from Lovegrove (1994).

Normal VIBIONNAL SVII INTERNATI AND INTERNOCIASTIC	(THREE FIXATIONS)
Normal Vision Nermanon des Iconoclastic	(TWO FIXATIONS)
Normal Vision Is Iconoclastic	(ONE FIXATION)

Owing to the way that the transient system is assumed to operate during reading by this theory, Lovegrove and colleagues (see Lovegrove, 1994), predicted that dyslexic children would experience fewer problems reading if one word was presented at a time, as opposed to continuous lines of text. Lovegrove found that dyslexic children did have a distinct 'one-word at a time advantage'. In the condition where words were presented one-by-one in the centre of the screen (so that participants did not have to move their eyes), and in the condition where the words were presented one-by-one successively to the right of the previous ones, dyslexic children made fewer errors than reading-age matched controls.

²Marr and Poggio (1979) first recognised the role of the transient system in the control of eye movements, especially when looking at near objects requiring convergence of the two eyes. Patients with lesions of the right posterior parietal cortex (PPC), where the transient system is thought to project to, exhibit unusual eye movements and experience difficulties in fusing together images from the two eyes. Livingstone and Hubel (1987) point out the role the transient system plays in depth perception and stereovision in this way. Chapter 3 examines a general visual deficit hypothesis of dyslexia in a novel yet objective way.

A transient system deficit can therefore account for findings in the dyslexia and vision literature. Furthermore, there is neuroanatomical evidence for differences in the magnocellular layer of the lateral geniculate nucleus in dyslexic brains (Livingstone, Rosen, Drislane and Galaburda, 1991). This is the system which (in primates) conducts fast, low contrast information. There is also evidence from fMRI that activation in the magnocellular system in dyslexia produces a different amount of activation during processing of such information (Eden, VanMeter, Rumsey, Maisog, Woods and Zeffiro, 1996) and that magnocellular function may predict patterns of reading errors (Cornelissen, Evangelinou, Hansen and Stein, 1997). However, the theory has not been without criticism. In particular, there has been some inconsistencies between techniques designed to measure the same thing: transient system function (e.g. DiLollo, Hansen and McIntyre, 1983; Arnett and Di Lollo, 1979; Hogben, Rodino, Clark and Pratt, 1995) as well as difficulty replicating results (e.g. Hayduk, Bruck and Cavanagh, 1996; Johannes, Kussmaul, Münte and Mangun, 1996; Gross-Glenn, Skottun, Glenn, Kushch, Lingua, Dunbar, Jallad, Lubs, Levin, Rabin, Parke and Duara, 1995). Furthermore, although high spatial frequency stimuli are generally shown to have greater visible persistence than low spatial frequency stimuli, dyslexic children show longer visible persistence with low spatial and shorter visible persistence with high spatial frequencies (compared with their controls). Because writing is high spatial frequency material, this finding makes little intuitive or logical sense with respect to the theory of how a transient system deficit actually impacts on reading: it is the control children who would be more likely to experience visible persistence and therefore confusion with high spatial frequency letters. The RTPDH in the auditory modalities has suffered criticism too.

Auditory Processing

The proposed rapid processing deficit in dyslexic children may not only affect the visual system. Research carried out principally by Tallal and her colleagues suggests that a pan sensory and possibly cross modal basic temporal processing deficit could exist (see e.g. Tallal, Miller and Fitch, 1993). Tallal carried out much of her research on language impaired children (many of whom who were also dyslexic) but points out that much of the data between them and dyslexic children is convergent (Tallal, Sainburg and Jernigan, 1991)

Tallal and Piercy (1973a and 1973b) found that language impaired (dysphasic) children found it more difficult than controls to discriminate and respond accurately to two tones of different frequencies if presented in rapid succession. The accuracy

of the dysphasic children also decreased more than that of their controls when more than two tones were used. Rather than a 'hearing' problem as such, the evidence pointed towards a higher level auditory processing deficit, involving problems with auditory sequencing and/or auditory memory. Because the dysphasic children's performance decreased with shorter interstimulus intervals, an auditory sequencing problem appeared more primary, with confusion between which stimulus appeared first (when both stimuli appear within a short time frame). However, since the deficit did not completely disappear with longer interstimulus intervals when more than three tones were used there may be a concomitant serial memory deficit.

After testing children with and without delayed reading development, Tallal (1980) found a high positive correlation (r=0.81) between scores on nonsense word tests and rapid auditory perception. She concluded that the deficiency in rapid auditory temporal perception "may be related to difficulty in learning the phoneme-grapheme correspondences involved in learning phonics rules". This tied in with the principles of the phonological deficit theory and added a deeper explanation for the phonological deficit, attributable to neurological mechanisms.

Tallal and Stark (1982), however, found few differences between purely reading impaired (no language impairment) and control children on auditory processing tasks and tests of phonics skills. Reading impaired children did perform worse on serial memory, visual scanning and sequencing tasks. Tallal and colleagues (see Tallal et al, 1991) have also compared dyslexic children with and without a concomitant language impairment on phonological coding and temporal processing tasks. They found whereas those with language problems had phonological coding deficits (reading nonsense words) and temporal processing problems, those with normal language scores had neither. Existing problems were also found to be highly correlated (r=0.81). This questioned details of the phonological deficit theory because dyslexic children without a language impairment showed no deficit. Tallal concluded that the temporal processing deficit may interfere with phoneme analysis resulting in language deficits, subsequent decreases in phonological awareness and only sometimes problems in learning to read. For this reason, she suggests, dyslexic children without a language impairment may benefit from different approaches from those with one.

Similar results have been found with tactile stimuli and even using two different modalities (Tallal, Stark and Mellitis, 1985). Tallal and colleagues (see Tallal et al, 1981), however, have also found that performance for dysphasics of 7-8 year olds

was twice as poor on auditory as on visual tasks, whereas there was a general deficit in the younger group (5-6 year olds). This could be due to different processing parameters in the two systems, or because only the more seriously impaired children continue to be language impaired when they are older. It has also been found that dyslexic children experience problems in producing rapid, sequential motor actions (see e.g. Tallal et al, 1981 and also Wolff, Michel, Ovrut and Drake, 1990).

Other, more recent, evidence for the possibility of a rapid temporal processing deficit hypothesis comes from PET studies of normals which suggest that it is possible to have such a selective deficit. Fiez, Raichle, Miezin and Petersen (1995) have shown that increases in rCBF are found in the frontal opercular areas for auditory detection tasks involving rapid temporal changes as opposed to steady state vowels. They have not yet examined the performance of LI or dyslexic children under these same conditions. This is the obvious next step.

The hypothesis of an auditory RTPD therefore initially seems to have much support. However, as well as lack of replication outside of Tallal's own lab, the theory, and in particular Tallal's inferences from her evidence, have been heavily criticised. Studdert-Kennedy and Mody (1995) and Mody, Studdert-Kennedy and Brady (1997) provide a comprehensive catalogue of the problems. They object in particular to the apparent confusion between 'perception of rate' (which Tallal implies) and 'rate of perception' and also to the claim that the deficit shown is anything but speech specific. In addition, they criticise Tallal for being inconsistent in her claims. The evidence presented here for the RTPDH has not been described in sufficient detail to warrant listing the difficulties. However, in brief, they conclude that the deficit in dyslexic children reflects difficulty in stimulus identification when presented rapidly, rather than any difficulty of temporal order as claimed. Furthermore, they suggest that the deficit is specific to stimulus identification and is therefore not a difficulty in rate of perception either. Moreover, they suggest that the claim that the difficulty is a general auditory deficit rather than a speech specific deficit is unsubstantiated.

These problems appear to have arisen principally from unwarranted inferences from speech to non-speech tasks (and vice versa) and a failure to account for the fact that the reading impaired children's performance on tone temporal order judgement was not significantly worse than their performance on tone discrimination alone. Furthermore, although Tallal and Piercy (1975) concluded that '...*it is the brevity, not the transitional character*' of the stimuli which causes difficulties, the difficulty in identifying /ba/ and /da/ (the starting point for the whole hypothesis) is attributed to difficulty in 'the analysis of rapidly changing acoustic information'. Such inconsistencies together with the sheer volume of papers which Tallal has published on this same subject is enough to convince me to leave that particular argument here! In any case, although the theory may be able to account for discrimination and some visual difficulties in dyslexic children, it cannot account for the difficulties which dyslexic children have with rhyme perception (which requires slow processing). Furthermore, although training studies have shown that training on temporal processing skills improves performance on temporal processing tasks in language impaired children (Tallal, Miller, Bedi, Dyma, Wang, Nagarajan, Schreiner, Jenkins and Merzenich 1996), this has not been extended to either reading or dyslexic children, has failed to include a control group and is not entirely surprising in terms of practice effects.

Thus, in summary, the RTPDH is able to provide neurophysiological evidence for a rapid temporal processing deficit, and can explain discrimination deficits via this mechanism. Behavioural evidence has also been found indicating such a deficit and it seems that the deficit does predate the behavioural signs. The theory is also able to explain various previously unrelated speed, visual and phonological problems. However, crucially, it fails to account for difficulties with writing, spelling and rhyming and has proved difficult to replicate.

1.5 Dyslexia Automatisation Deficit Hypothesis

The Dyslexia Automatisation Deficit hypothesis (DAD) (Nicolson and Fawcett, 1990) suggests that, rather than specific phonological (e.g. Bryant and Bradley, 1985), motor (e.g. Miles, 1983; Augur, 1985; Rudel, 1985) or visual (e.g. Lovegrove: as described above) deficits, dyslexic children suffer from a more general problem in the automatisation of skills. Both Norman (1982) and Anderson (1982) emphasise the importance of automatisation in their models of the acquisition of skills and expertise. Reading is a complex skill which requires automatisation for speed and fluency, and in particular to be able to read and comprehend at the same time (see LaBerge and Samuels, 1974). Shiffrin and Schneider (1977) describe the difference between automatic and controlled processing and their differential demands on attention, with automatic processes making few demands on attention in comparison with controlled processes, such as

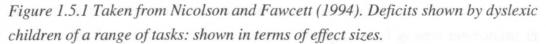
comprehension. The DAD proposes that children with dyslexia have difficulty making all skills automatic.

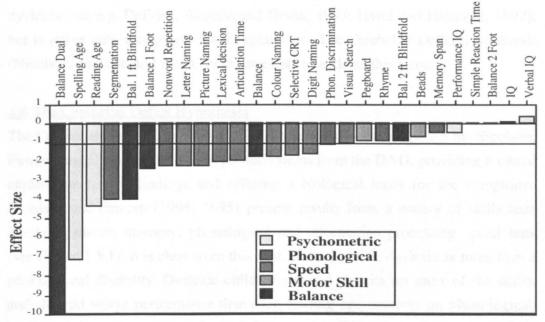
An accepted procedure for establishing whether a task is being performed automatically or not is to give the person an additional task in order to determine whether the first deteriorates under pressure. If the first is automatic, it should be taking up little attentional capacity, and there should be resources left to do a simple additional task. If the first task requires controlled processing, however, there will be few remaining resources to perform a further task. Thus, strong support for the DAD comes from Nicolson and Fawcett (1990), who tested dyslexic and control children's balance with and without an additional task. They found that dyslexic children balanced equally as well as controls when that was all they had to do (using conscious compensation: see below). However, when an additional task was added the dyslexic children tended to wobble and fall, whereas the controls (balancing automatically) did not. This was the case even with the difficulty of the additional task (counting backwards) calibrated to each child individually, ensuring that the results were not due to the dyslexic children simply finding the additional task more difficult than the controls (see Fawcett, 1990 for a review). This finding was later replicated by Yap and van der Leij (1994a). No other theories of dyslexia would predict such difficulties with balance. Further experimentation using a different paradigm showed that the deficit was not one of learning: dyslexic children, although slower, learnt to find their way through a 'pacman' type maze at the same rate as controls (Nicolson and Fawcett, 1993c)

The DAD posits that phonological difficulties (and hence short term memory deficits) arise from non-automatisation of phonological abilities, rather than inability in phonological tasks *per se*. It therefore happily accounts for naming speed deficits found in dyslexic children. Denckla and Rudel (1976) found naming speed deficits in children with dyslexia even in comparison to non-dyslexic children suffering from minimal brain dysfunction (MBD), although the children with MBD made more errors (thought to be visual errors). The naming deficit in dyslexia was therefore concluded to be only one of speed, with accuracy within the normal range.

The DAD can therefore explain the difficulties of dyslexic children in reading fluently. However, as such a general hypothesis, it also predicts deficits on other skills. In recognition of this Nicolson and Fawcett (1995) tested 66 children on a large battery of primitive skills (e.g. naming speed, bead threading, balance and

phonology) and found no evidence for subtypes of dyslexia, but more importantly that dyslexic children showed deficits on most of the skills, with all children showing deficits on at least two skill modalities. The results are shown in Figure 1.5.1. This diversity of deficits is not well explained by any, more specific, theory of dyslexia.





Accompanying the DAD is the Conscious Compensation (CC) hypothesis (Nicolson and Fawcett, 1990). CC is postulated to account for dyslexic children's apparently normal performance on many tasks apart from reading, which presumably also involve the automatisation of a skill. The hypothesis proposes that providing remaining resources are present, children with dyslexia will use those resources to enable them to do a task using controlled rather than automatic processing. Dyslexic children are therefore more likely to consume more resources in order to do the same task. Anecdotal evidence for such behaviour comes from the phenomenon that dyslexic children seem to suffer from excessive tiredness after performing relatively routine operations (e.g. Augur, 1985; Miles, 1983). The DAD states that they have to concentrate more and expend more effort on tasks which others perform automatically (and therefore with virtually no effort at all).

Methodological difficulties in investigating the DAD arise because the automatisation problems only appear at high levels of skill, or when several skills need to be performed together. Furthermore, although the concept of conscious compensation accounts for the observed tiredness of dyslexics after task performance, it is not exactly clear just how far they can compensate or at what point they start. This is generally true of research in this area, so whilst making it a strong theory, it also makes it difficult to falsify³. However, although the DAD provides a good explanation of the wide range of deficits in dyslexic children, it does not propose any causal mechanism and is perhaps rather *too* general. In addition, it provides no anatomical substrate for the deficits observed despite evidence for the operation of some kind of biological and genetic mechanism in dyslexia (see e.g. DeFries, Alarcón and Olson, 1997; Hynd and Hiemenz, 1997), but is rather only a cognitive level explanation. The Cerebellar Deficit Hypothesis (Nicolson, Fawcett and Dean, 1995) attempts to address this issue.

1.6 The Cerebellar Deficit Hypothesis

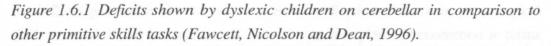
The Cerebellar Deficit Hypothesis (CDH) of dyslexia was proposed by Nicolson, Fawcett and Dean (1995). The hypothesis stems from the DAD, providing a causal explanation for the findings and offering a biological basis for the symptoms. Nicolson and Fawcett (1994; 1995) present results from a battery of skills tests involving motor, memory, phonological and information processing speed tests (see Figure 1.5.1). It is clear from this that, as they claim, dyslexia is more than a phonological disability. Dyslexic children showed deficits on most of the skills, and showed worse performance than even reading age controls on phonological, bead threading, naming speed and balance tasks. Nicolson and Fawcett (1995) view the DAD as a theoretical 'half-way house', describing rather than explaining many of the symptoms, and being still too specific to explain others.

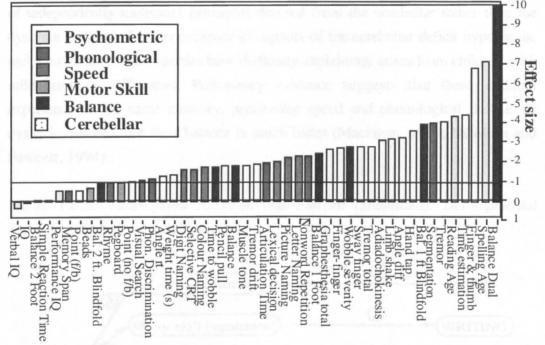
The cerebellum is generally considered to be a motor area, particularly involved in the acquisition of skill (e.g. Marr, 1969; Krupa, Thompson and Thompson, 1993). By way of brain imaging techniques Jenkins, Brooks, Nixon, Frackowiak and Passingham (1993) demonstrated the role of the cerebellum in both new learning and automatic sequential movement. The CDH can therefore account for motor skill

³ An approach to such methodological difficulties which I have always admired, particularly given such a wide range of deficits in children with dyslexia, is that of manipulating paradigms in an attempt to make apparent 'deficits' advantageous. This is obviously not always possible. However, the technique has been used in investigations of the RTPDH of dyslexia (see section 1.4). Hogben, Rodino, Clark and Pratt (1995) investigated the hypothesis of increased visible persistence in dyslexic children causing reading problems. They used a paradigm where visible persistence would be advantageous in order to identify a missing part of a pattern presented on two separate but rapidly sequential slides (separate without visible persistence, but expected to be seen together with visible persistence). Children with dyslexia were expected to (but did not) perform better on this task if they experienced visible persistence. In Chapter 5 I attempt a similar manipulation in order to investigate the DAD. Finding dissociations between tasks is also important (attempted in Chapter 4 and work which followed from Chapter 6).

and automaticity deficits in children with dyslexia (e.g. Rudel, 1985; Augur, 1985; Wolff, Michel, Ovrut and Drake, 1990; Nicolson and Fawcett, 1995; Fawcett and Nicolson, 1995b). However, until relatively recently, the cerebellum has not generally been considered as a serious contender in dyslexia research since it was thought to be involved only in motor skills. However, with the advent of new technology (functional imaging) and increasing interest in the area, the cerebellum has been suggested as having a role in many cognitive activities in addition to motor skills. Neuroimaging studies show the cerebellum to be activated in tasks involving memory (Grasby, Frith, Friston, Bench, Frackowiak and Dolan, 1993), and word processing (Petersen and Fiez, 1993). Neuropsychological studies on cerebellar patients show deficits in attention shifting (Akshoomoff and Courchesne, 1994) and memory (Lazareff and Castro-Sierra, 1996). Evolutionarily new links have also been found between the cerebellum and Broca's language areas (Leiner, Leiner and Dow, 1993). The CDH of dyslexia therefore now presents a plausible argument. However, despite the cerebellum only recently being regarded as any more than a motor area, it has been implicated in dyslexia before. Controversially, Levinson (1988; Frank and Levinson, 1973) proposed a cerebellar deficit in dyslexia (on the basis of eye movement data).

Nicolson, Fawcett and Dean (1995) first found indirect evidence of cerebellar involvement in dyslexia using a time estimation task on which cerebellar patients have been found by Ivry and Keele (1989) to be less accurate than normals. They found that, similar to cerebellar patients, dyslexic children were impaired on a time estimation, but not a loudness estimation, task. Furthermore, Jueptner, Rijntjes, Weiller, Faiss, Timmann, Mueller and Diener (1995) showed in a PET study that cerebellar activation did occur in the time, but not the loudness, estimation task in controls. It therefore seems plausible that a mild cerebellar impairment could explain many of the observed deficits in children with dyslexia. However, although cerebellar patients show the same time/ loudness estimation dissociation, time estimation is by no means a direct measure of cerebellar function. Thus, additional strong support for the CDH came from Fawcett, Nicolson and Dean (1996) using a battery of clinical cerebellar tests (from Dow and Moruzzi, 1958). They found that dyslexic children were impaired on 13 out of the 14 tests. The dyslexic children were impaired in comparison with both age- and reading- matched controls and were equivalent only on a finger-to-finger pointing test. Figure 1.6.1 shows the effect sizes of the cerebellar, in comparison to other, deficits already shown in Figure 1.5.1. More recently, in a replication of Jenkins et al (1994), Nicolson, Fawcett, Berry, Jenkins, Dean and Brooks (1999) have found significantly reduced cerebellar activation for dyslexic adults during pre-learned ('automatic') and novel sequences of finger movement tasks. This is evidence that the cerebellar of dyslexic children may not be fully contributing to the acquisition of motor skill (novel sequences) or the performance of learned tasks. Furthermore, Finch (1998) has found neuroanatomical differences in dyslexic and control cerebella, although age differences between the brains do somewhat limit inferences which can be made at this stage.

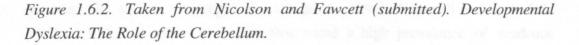


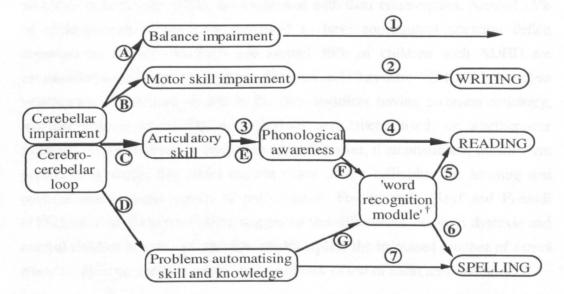


A cerebellar impairment therefore provides an anatomical substrate for phonological, motor and automatisation difficulties exhibited by children with dyslexia. Learning to speak (and articulate) is probably one of the most complex fine muscular skills which we ever learn. It is accepted that articulatory skills play a key role in language development, and, in particular, the development of phonemic awareness (Locke, 1983). A mild cerebellar deficit would be likely to impair this fine motor performance, thereby delaying speech and possibly the understanding of its structure (Fawcett, Nicolson and Dean, 1996). This idea is similar to the motor theory of speech perception (that we 'know' phonemes by how they are made, not how they sound: see e.g. Liberman and Mattingly, 1985), although in weaker form. Similarly, handwriting is likely to require more effort and therefore not be as tidy; a characteristic of dyslexia which is not addressed by many other theories. The CDH explains short term memory deficits in a similar way to the phonological

theory of dyslexia; that articulation speed is slower and so less information can be stored in the phonological loop of working memory.

Nicolson and Fawcett (submitted) present a figure illustrating the hypothetical causal chain linking cerebellar problems, phonological difficulties and eventual reading problems (see Figure 1.6.2 below). The theory therefore provides neurophysiological evidence for a cerebellar anomaly, a coherent link between cerebellar roles and manifest symptoms, behavioural evidence of some classic cerebellar signs and is able to explain otherwise disparate deficits in phonological skill, motor skill and automatisation. In addition, it explains phenomenon in terms of independently motivated principles derived from the cerebellar rather than the dyslexia literature. Further evidence in support of the cerebellar deficit hypothesis, and which many other theories have difficulty explaining, arises from children with mild learning difficulties. Preliminary evidence suggests that these children experience all the same memory, processing speed and phonological deficits as dyslexic children, but their balance is much better (Maclagan, 1999; Nicolson and Fawcett, 1994).





1.7 Attentional Deficits?

Learning anything would appear to require attention, so an attention deficit would be expected to result in many of the deficits exhibited by children with dyslexia. Whyte (1994) argues that in this context attention must be seen as being "operationally linked with long-term memory; both at the initial stages, when LTM guides the efforts that are directed towards focusing, and at the later stages, when integration and transformation of the stimulus is taking place". She cites Craik and Lockheart (1972) as evidence showing how the effectiveness of such procedures are related to attentional and perceptual processes at the time of learning. Furthermore, LaBerge and Samuels (1974) emphasise the role of attention in automatising skills. According to their model of reading, automaticity in reading is dependent on reorganising components of reading (e.g. graphemes, phonemes, whole words and so on) into larger and larger units until whole groups of words can be processed. They state that attention is crucial for such reorganisation (see p315), but that accuracy may have to be sacrificed for automaticity at the early stages of reading. Attention deficits in dyslexia may therefore be able to offer an explanation of automaticity deficits in reading and possibly other skills.

Attentional problems have been linked to dyslexia for many years. It is a common clinical observation that dyslexia and attentional disorders frequently co-occur and that children with dyslexia can have difficulty 'keeping on track' (Augur, 1985). Unfortunately, however, within the clinical literature, the assessment of children is dominated by the use of rating scales (Halperin, 1996). Cantwell and Satterfield (1978) (cited in Light et al, 1995) first noted a high prevalence of academic problems in boys with ADHD in comparison with their counterparts. Around 15% of children with dyslexia are estimated to have concomitant attention deficit hyperactivity disorder (ADHD) and around 36% of children with ADHD are estimated to have dyslexia (Shaywitz, Fletcher and Shaywitz, 1994). It is not clear whether the co-morbidity is due to the two disorders having common aetiology, whether there is an overlap in definitions and criteria used, or whether one syndrome has a tendency to lead to another. However, if an attentional deficit were present in dyslexia, this could explain many of the difficulties in learning and perhaps also in some aspects of performance. For example, Senf and Freundl (1972), cited in Torgesen (1979), suggested that differences between dyslexic and control children in terms of attention might explain the increased number of errors made by dyslexic children on the digit span task (a test of short term memory).

Hynd, Semrud-Clikeman, Lorys, Novey and Eliopulos (1990) found neuroanatomical links between ADHD and dyslexia, but also differences between ADHD and dyslexic brains, with a distinct symmetry or even reversed asymmetry of the planum temporale unique to children with dyslexia. Light, Pennington, Gilger and DeFries (1995), in a twin study, found that heritable variation accounted for around 70% of the observed covariance between the reading and hyperactivity measures used. This suggested common genetic influences of concomitant reading disability and ADHD, as opposed to one syndrome having 'knock-on' effects upon the other. Stevenson, Pennington, Gilger, DeFries and Gillis (1993) also estimated that around 75% of the co-occurrence of the two conditions was due to shared genetic influences.

However, further studies have compared and contrasted cognitive deficits in ADHD and reading disability. Pennington, Groisser and Welsh (1993), for example, examined phonological abilities and executive functions in children with reading disabilities, ADHD, or both. They postulate a 'phenocopy hypothesis' which suggests that rather than both disorders having a common aetiology (e.g. Light et al. 1995), or one disorder causing another in its full form, one disorder may lead to the symptoms of another but the deeper characteristics of the secondary deficit will not be present. They found that in children with comorbid reading disability and ADHD, there was no executive function deficit (where a deficit was present in children with pure ADHD). This was assessed by performance on a composite score created from the Tower of Hanoi task, the matching familiar figures test, the WCST⁴ and the Continuous Performance Test. However, deficits in phonological abilities were found in both groups. This is taken as evidence that ADHD symptoms in children with dyslexia are secondary to reading disability; the two syndromes are not fully exhibited in full in terms of their deeper characteristics.

In contrast, Korkman and Pesonen (1994), assessed children with pure dyslexia, pure ADHD and both syndromes on a 'comprehensive set of neuropsychological measures' (the Neuropsychological Assessment of Children; Korkman 1988). They found that children with specific learning disorder were impaired in phonological awareness, verbal memory span, verbal IQ and storytelling. Children with ADHD were impaired in the control and inhibition of impulses. Children with both ADHD and specific learning disorder showed *all* of these deficiencies and all groups exhibited deficits in visual motor precision and name retrieval. Similarly, in a partial replication of Pennington et al (1993), using different measures of the supposed 'core deficits' of reading disabled and ADHD children (rapid automatised naming in the case of RD and the WCST in the case of ADHD), Närhi and Ahonen (1995) found no support for the phenocopy hypothesis. As hypothesised, RD and RD+ADHD groups were deficient in their naming speed in comparison to the

⁴Winconsin Card Sorting Test

ADHD group. The ADHD group (in comparison to the others), however, were not significantly different on executive function measures, although a trend towards the comorbid RD+ADHD group having worse performance than the others did occur. Executive function deficit was therefore found not to be specific to ADHD. There were, however, several problems with both studies. Närhi and Ahonen's groups, were not matched for age: ADHD groups were younger. Instead, age was controlled by using normative data to which the test results were compared. However, since the influences of maturation on ADHD and RD are not known, inferences are limited by this design. If, for example, the executive function deficit in children with ADHD (Pennington et al, 1993) was a developmental lag of some sort, then the control comparison data of younger children would not necessarily be expected to elicit differences. Moreover, in both studies, the IO criterion for inclusion was fairly low. Pennington et al (1993) required a FSIQ of 80 or above, Närhi and Ahonen's criterion was lower, with either verbal or performance IO of 80 or above acceptable. Although this is an acceptable criterion is the US, in the UK the standard criterion is a full scale IQ of 90 or above. Närhi and Ahonen's mean IO was 90.6. This is below average, though in the average band (which is acceptable). However, it is highly likely that many of Närhi and Ahonen's participants had full scale IQs much lower than 90. Meanwhile, Pennington et al's composite score of executive function tasks appears to be rather complex and not in all cases representative of the data on the individual tests. It is interesting to note that, looking at the scores on the individual tests, there are no significant differences between groups on any measures on the WCST, the very task used by Närhi and Ahonen. Närhi and Ahonen's failure to replicate the executive function deficit found by Pennington et al (1993) in children with ADHD is therefore not entirely surprising.

There is therefore evidence for links between ADHD and dyslexia. It is possible that dyslexia actually leads to some attentional deficits, although other evidence suggests that dyslexia and ADHD symptomatology frequently co-occur due to a common aetiology in terms of genetics and/ or brain dysfunction. Unfortunately, relatively little information exists on the exact nature of the attentional disorder in dyslexia and also whether attentional difficulties genuinely exist for those dyslexic children without ADHD. One possibility is that all dyslexic children have some degree of ADHD, with a continuum of difficulties actually exhibited; if there is a common aetiology between the two disorders then this is at least a plausible description. However, Whyte (1994) comments on how teachers ratings of attention are usually related to cognitive performance so teachers conceptions of attention may be more closely related to school learning than attention as defined by researchers.

1.8 Scope of this thesis

The main theories of dyslexia are therefore the phonological deficit theory, the dyslexia automatisation deficit hypothesis, the cerebellar deficit hypothesis, the rapid temporal processing deficit hypothesis, the attention deficit hypothesis and the visual deficit hypothesis. Although experiments presented in this thesis each have implications for these theories (even if only to say that the theory is not broad enough to encompass the wide range of deficits shown), not all the theories are explicitly investigated.

Chapter 2 investigates the CDH by using a prism adaptation paradigm, comparing dyslexic and control children on their speed of adaptation. The CDH is a relatively recent hypothesis, which encompasses and is able to account for many previous research findings. However, as yet, there is relatively little explicit evidence for this hypothesis (in comparison to the phonological deficit hypothesis, for example, which has stood both the test of time and of several investigations). The time estimation task (Nicolson, Fawcett and Dean, 1996), clinical cerebellar tasks (Fawcett, Nicolson and Dean, 1996) and the PET study showing reduced cerebellar activation in dyslexic children evidence (Nicolson, Fawcett, Berry, Jenkins, Dean and Brooks, 1999) are currently the most important published pieces of evidence. The cerebellum is thought to be crucial in adaptation and therefore a stringent test of the CDH is the ability to adapt.

Chapter 3 involved taking an exciting opportunity to further investigate the hypothesis of a visual deficit in dyslexia. Thus, rather than focusing on any current major theory of dyslexia, it (rather opportunistically) takes advantage of a novel technique in the vision literature in order to take a further look at a less popular, but still controversial, theory. The sequential stereopsis paradigm (Enright, 1996) allows examination of the ability to control vergence across saccades. This is undoubtedly a necessary skill in fluent reading, but one which has not been previously examined in an objective and scientific manner. As well as the main advantage of the method, that it allows dynamic investigation of vergence control *across* saccades, it is also interesting because it encompasses saccadic accuracy, vergence control and stereovision. These are all functions which have been recently implicated in dyslexia (see e.g. Stein 1994; Pavlidis 1980-1985, Bedwell, Grant and McKeown, 1980). In addition, it affords the ability to investigate posterior

parietal cortex function in dyslexic students, an area which Stein (1994) has suggested as a possibility for dyslexia research and to which the transient system projects. Moreover, previous studies have elicited inconsistent results when investigating either one function or another. Thus, in line with Simons and Grisham's (1987) suggestion to examine the relationship of several binocular functions in the same study, Chapter 3 does exactly that. In addition it examines the automaticity of the binocular control by adding a secondary task (and therefore has implications for the DAD), as well as the effects of fatigue on visual function, thought to be important by clinicians generally (see e.g. Watten, 1994, for an overview).

Chapter 4 takes another different tack in order to investigate reported attentional difficulties in dyslexia, an area which has been given little attention in the literature (despite anecdotal evidence from clinicians), but which presents an important practical issue. The paradigm used systematically investigates the ability to focus, shift and sustain attention in dyslexic children. It also examines the time necessary to reorient attention. It therefore has implications for the RTPDH and, because cerebellar patients have been shown to have difficulty rapidly shifting their attention, the CDH. Furthermore, it is a possibility that dual task deficits in dyslexic children actually represent a difficulty in rapidly shifting attention between two tasks, so a deficit in rapidly shifting attention would cast doubt on the interpretation of this evidence.

Chapter 5 further examines results obtained in Chapter 4. It focuses in particular on two possible explanations of the results, namely a general resources deficit and an automatisation deficit. Much of the evidence which supports the DAD could also be accounted for by a general resources deficit in children with dyslexia. The two alternatives therefore have important theoretical implications for the DAD. An explanation of children with dyslexia having fewer resources overall, rather than an automaticity deficit, is equally plausible but has not yet been examined. The paradigm used also provides an opportunity to replicate results found in Chapter 4.

Chapter 6 examines the hypothesis of a sustained attention deficit in children with dyslexia again, but this time in terms of attentional 'lapses' over a period of seconds, rather than concentration over tens of minutes. The possibility of attentional lapses is interesting and may characterise the actual form of any attention deficit better than standard tests of attention. It may also be able to account for

some of the reported behavioural patterns exhibited and reported by dyslexic children and adults (such as walking into a room and then forgetting why!).

The scope of this thesis is therefore perhaps wider than most. Having started examining the CDH quite explicitly, I go on to look at visual and then attentional factors in dyslexia.

The structure of the thesis is actually a simple chronological one; each chapter presented in the order in which the studies were done. Having enjoyed the challenge of designing a test of prism adaptation (but obtaining slightly disappointing results), subjective observation led me to consider a visual deficit, particularly since the opportunity arose to investigate this hypothesis in such a novel way. The interest in attention came mainly from the scarcity of literature in the area involving well conducted, empirical investigation. Furthermore, it presented the opportunity to examine a function which would have potential implications for the RTPDH, the CDH and also possibly the DAD (since an inability to rapidly shift attention might also be able to account for dual task difficulties). In the finest tradition, none of the predictions were confirmed! However, results were interesting, and on this occasion led to two further investigations presented in subsequent chapters. Research following results from the final chapter has also been conducted.

			Theoretical Approaches					
			Phonological	DAD	CDH	Visual	Rapid	Attention
			Deficit			Deficit	Processing	
Ch	apter/							
Stu	dy no.	Topic						
	la	Prism	Х	X	control	Х	x	x
2	1b	Adaptation	X	X	~	X	X	X
	2a	Sequential	Х	x	X	~	x	x
3	2b	Stereopsis	X	~	X	~	X	X
	3	Attention	Х	~	~	Х	v	v
4		Shifting						
	4	Resources	Х	~	x	Х	Х	X
5								
	5	Sustained	Х	X	X	Х	X	~
6		Attention						

Table 1.6.1 Table showing theories specifically investigated in each chapter

Chapter 2

Prism Adaptation in Dyslexia

Investigating the Cerebellar Deficit Hypothesis

Summary:

This chapter reports a novel and stringent investigation of the cerebellar deficit hypothesis of dyslexia (Nicolson, Fawcett and Dean, 1995). Since prism adaptation is often associated with the cerebellum (and cerebellar lesions appear to impair adaptation to prisms), this study examined the speed and type of prism adaptation exhibited by children with dyslexia and their controls. First, a control study is presented, in which susceptibility to the 'straight ahead shift' on its own is investigated. As predicted, this study highlighted the need to perform prism adaptation experiments in darkness (or an unstructured environment), since the prisms appeared to have little effect on participants' judgement of straight ahead in an illuminated room. No group differences were found. The main study compared the same groups on their speed and type of adaptation to 12 diopter fresnel prisms in a darkened room. Groups were compared on several measures, including initial pointing accuracy, speed of adaptation, intermanual transfer and negative after effect. No significant group differences were found on any of these measures, although (even for the control group) results were generally rather noisy and difficult to interpret. It is concluded that prism adaptation is not a satisfactory experimental measure for the purposes of investigating the cerebellar deficit hypothesis.

2.1 Prism adaptation

2.1.1. Prism Adaptation and the Cerebellum

A classical and much-investigated function of the cerebellum was chosen as a stringent test for the cerebellar deficit hypothesis of dyslexia. The cerebellar deficit hypothesis and the evidence supporting it is detailed in Chapter 1 (section 1.6).

Using a similar argument to the Nicolson, Fawcett and Dean (1995) and Fawcett and Nicolson (1996) studies, if children with dyslexia have mild cerebellar abnormalities, they might be expected to show deficits in functions normally attributed to the cerebellum.

Prisms placed in front of the eyes make various distortions to vision (most commonly displacements to either side) by refracting the light entering the eye. This refraction has the effect of changing the position of the visual field, so that if one is apparently 'looking' straight ahead, the field of view will actually be to either side. The amount of change is related to the angle of the prism.

Experimentally, prisms can be used in order to investigate adaptation ability. If a substantial, or even a small, change is made to the field of view, this requires correction in order to be able to continue with life as normal. This correction can occur in different ways and at different rates. Hess (1956) (cited in Gross, 1990) found that after fitting chickens with prisms which shifted their field of view to the left, they always pecked to the left of the grain. Unfortunately, no matter how many times they pecked (and missed), they never adapted to the prisms and never changed their behaviour to allow them to eat normally. Humans and monkeys *can* adapt to prisms. However, adaptation to displacing prisms has been shown to be less, or impossible, in both macaque monkeys with cerebellar lesions (Baizer and Glickstein, 1994) and also in patients with cerebellar damage (Weiner, Hallett and Funkenstein, 1983). Thus, in a stringent investigation of the cerebellar deficit hypothesis of dyslexia, this experiment compared ability to adapt to prisms in children with dyslexia and their controls.

2.1.2. Components of adaptation

Prism adaptation is more complicated than it may first appear; the process usually consisting of more than one component, with each component not necessarily independent. Researchers dispute the finer points of the exact components, their categories, and their names, but there are three basic categories of components involved:

i) sensory components:

Sensory adaptation is usually proprioceptive. Since vision tends to be the dominant sense in humans, the felt position of the arm tends to adapt in order to become congruent with vision. Harris (1963) has shown how, having seen their stationary

right hand through right displacing prisms, participants felt their hands to be further apart (at a given distance) than participants who had not seen their hands. This suggested that the felt position of the hands had altered in order to become congruent with vision.

In addition to proprioceptive adaptation, visual adaptation can occur. However, some researchers would claim that visual adaptation is mostly *not* a component of sensory adaptation at all, but is rather related to a cognitive reappraisal of the task (see following sections). One measure of visual adaptation and/or visual shift is the pre-post shift in apparent visual straight ahead (which can also change due to an adaptation in terms of head position). True visual shift is correlated with a change in the felt direction of gaze, or felt relation of head to body.

ii) the straight-ahead shift

The straight-ahead shift (Harris, 1974) or immediate correction effect (Rock, Goldberg and Mack, 1966) is often not considered to be a measure of true adaptation. The name of the effect refers to the fact that cues from the experimental environment (in particular the direction of 'straight-ahead') can make it very apparent that the prisms are rotating and displacing vision, thereby making compensation for the displacement rapid and easy. Compensation might take 2 forms: a *rapid* visual adaptation/ immediate correction (as above), or, depending on both the task and the environment, a cognitive reappraisal of the true meaning of the task. These two components appear to be interrelated. For example, if a participant is asked to point (or position a target) straight ahead, having viewed the room *before* prism displacement (and therefore knowing that they are actually facing a wall perpendicularly) then a prism rotation is quite obviously a visual distortion. Thus, in this situation, visual adaptation is likely to occur quickly. The cognitive reappraisal component of the adaptation is such a situation is the driving force, but is not suspected to be a cerebellar function.

Further evidence against the notion that this shift in vision is a true component of adaptation, Harris (1974) explains how simply standing askew in a corridor (with no visual distortions or displacements being involved) and being asked to point straight ahead, could result in differential interpretations of the task. He maintains that instead of pointing in his or her median plane, a participant may point towards the end of the corridor; the environment may therefore change interpretations of straight-ahead, as opposed to any true visual adaptations of any kind taking place.

A similar phenomenon can occur whilst wearing prism goggles; rather than any true visual adaptation taking place to the judgement of straight ahead, the response is determined by the experimental environment. Indeed, it can be shown that no real adaptation has occurred by subsequently asking the participant to point at a previously unseen target, or, more convincingly, by asking them to point with an unseen hand at the target that they have just judged as straight ahead of them [the participant will still make an error concordant with the expected size of the prism displacement (Harris, 1974, p464) and hence no visual adaptation has occurred].

Harris does argue, however, that the straight-ahead shift [defined by him as "a change in the egocentric spatial direction that is treated as 'straight ahead' by a participant" (p.464)] ought to combine algebraically with other components of adaptation. Harris cites Harris and Gibson (1967 - unpublished) and Wilkinson (1971) as support for this conclusion. It seems therefore that a straight-ahead shift is likely to speed true adaptation, but is not in itself a cerebellar function.

iii) Behavioural Compensation/Assimilative Corrective Response (Welch, 1974). The adaptation component named 'behavioural compensation' results from participants' conscious strategy. Such strategy can be elicited by participants' explicit knowledge of the prisms' effects, or alternatively from use of feedback to calculate what the prisms are likely be doing (and adjusting the responses accordingly). Part of this component may be an 'assimilative corrective response' (Welch, 1974), occurring from repeated practice (until automatic) of the error-corrective response, but often without consciously or explicitly realising what is

happening

Effects of an assimilative corrective response are almost impossible to separate from other sensory components, since they are all reflected by target pointing behaviour. Support for this component's existence arises from Welch and Goldstein (1972) (cited in Welch, 1974). They have consistently found that the negative aftereffect when pointing *exceeds* the amount of proprioceptive shift (as measured by the participants setting a luminous target above the felt position of the right index finger) even using methodology *previously* found to induce little or no visual shift. Furthermore, Welch and Rhoades (1969) found a significant correlation between the negative aftereffect and proprioceptive shift when the exposure period involved no target pointing (but still terminal exposure), but not when target pointing was involved. Although the cerebellum may have a role to

play in any automatisation of a response, it is not suspected to be involved in any initial behavioural compensation component of adaptation.

Proprioceptive adaptation is therefore the main component of adaptation that is suspected to be a function of the cerebellum. Proprioceptive adaptation and other types of adaptation are likely to be negatively correlated in amount, so that together they will produce the full amount of adaptation. Thus, if more of one type of adaptation occurs, then less of the other is necessary. Furthermore, if behavioural compensation is introduced into the equation, the magnitude of the negative correlation between proprioceptive and visual adaptation will be reduced (Welch, 1974, p 453). Proprioceptive adaptation is the least disputed component.

2.2 Individual Differences in Adaptation

It is thought that the characteristics of an individual can affect how much of each component of adaptation occurs. Warren and Platt (1974) review Hamilton and Bossom's (1964) study of the decay of the prism after-effect with reafferent vs. non-reafferent experience (receiving visual feedback of one's own movements or not) and conclude that the non-significant result is unsurprising in view of the high variability between participants. They maintain that these differences should not simply be regarded as 'error nuisance', but rather investigated in their own right.

Early authors on prism adaptation investigated whether it was the felt arm position *or* the visual system that adapted (e.g. Helmholtz 1866). However, Warren and Platt (1974) found that proprioceptive adaptation correlated positively with individual eye fixation accuracy (thus suggesting that the more accurate the visual system, the more likely adaptation will occur elsewhere). Similarly, Kahane and Auerbach (1973) found that professional dancers experienced less proprioceptive shift than non-dancers, lending further support to Canon's (1970) (cited in Canon, 1971) *attentional* theory of which components adapt. In Canon's theory, dancers would be less likely to experience proprioceptive shift due to their increased attention to body positions. On the same theme, Luria, McKay and Ferris (1973) found that adaptation to visual distortions of size and distance underwater were handled differently by those with and without diving experience, and also by left-and right-handers. In addition, only the right-handers showed negative correlations between adaptation to size and adaptation to distance. However, greater correlations were found when after effect measures, rather than compensation

measures, were analysed, again illustrating the importance of distinguishing between behavioural compensation and true sensory adaptation.

2.3 Rationale of Experiments

Previous research has therefore shown that prism adaptation is a complex process. Adaptation can consist of more than one component and each component is highly dependent on the exact methodology, apparatus and instructions given. Speed of adaptation, intermanual transfer, type of adaptation and negative after effects are all components which can be measured either directly or indirectly in such experiments. Pilot work for the studies reported here, together with previous research, also suggests substantial individual differences, thereby further complicating the disentanglement of the processes.

Because of the evidence on individual differences, it was important to ensure in this experiment that all the possible components of adaptation were measured (either directly or indirectly). This would then ensure that any difference between the groups was not merely a result of a different *type* of adaptation (as opposed to a different amount). Hence, it was not sufficient to compare dyslexic and control groups on only one measure and make conclusions about ability to adapt. Both handedness and visual differences have been found to be associated with dyslexia. Thus, if only one measure of adaptation were taken, then it could be possible that adaptation was occurring in a different modality rather than differences reflecting either group's ability to adapt *per se*.

Researchers have investigated prism adaptation using several different paradigms and on several species and populations. Weiner, Hallet and Funkenstein (1983) required human participants to point to a vertical line with their finger from below a two-way opaque mirror (so that they could not see their hand). When a light was lit below the mirror, it became opaque, so that the participants could see their pointing position. Initial accuracy was measured first, followed by an adaptation period to (20 diopter base left fresnel) prisms and a measurement of negative after effect when the prisms were removed¹⁶. The negative after effect measure is thought by many to be the measure of true adaptation and in Weiner et al's experiment "*is attributed to persisting visual adaptation and should not be influenced by cognitive*

¹⁶The negative after effect is recognised by a mispointing in the opposite direction to the prisms' displacement after they have been removed; it is thought to be evidence of either motor learning of new responses, or visual changes (adaptation).

*correction*¹⁷" (p766). Weiner et al compared 25 normal adult volunteers with 59 patients with lesions of the central nervous system. The patients were divided into six different groups, patients with; cerebellar dysfunction, Parkinson's disease, left cerebral hemisphere lesions, right cerebral hemisphere lesions, Korsakoff's syndrome and Alzheimer's disease. Weiner et al found one significant result; a Student's t test showed that the cerebellar group produced less negative after effect than the controls on the first trial after the prisms had been removed (p<0.01). Therefore, a stringent and intriguing test of the cerebellar deficit hypothesis in children with dyslexia was to investigate various aspects of their prism adaptation.

Two studies are presented here. The control study measured responses of groups with dyslexia and matched controls in an experiment measuring extent of straight ahead shift. Re-analysis of some unpublished research (Meakin, 1995) found indirect evidence that groups with dyslexia and control groups may be differentially affected by the straight ahead shift. In a prism adaptation experiment conducted in full illumination, analysis restricted to the matched older groups of children with dyslexia and their controls found that the groups with dyslexia were initially significantly more affected by the prisms than their controls (p<0.01, see Appendix 2.3.1). Furthermore, rapid adaptation was seen over a period of only three pointing trials in both groups, suggesting that a rapid visual adaptation component was affecting the results. The control study presented was therefore important and sought to measure the effect directly. If groups were differentially affected by the shift, since it is thought to combine with other types of adaptation, the proprioceptive and visual adaptation which I intended to measure may have been unduly affected by the shift.

The main study compared the same participants on several different measures in a task which involved pointing at targets; initial accuracy without prisms, adaptation to prisms of the preferred hand (with feedback), intermanual transfer of any adaptation, and negative after-effects on both hands. Results of the two experiments can be interpreted together. If both experiments find significant group differences, then it is possible that the difference in the straight ahead shift was affecting the results of the main experiment (although the possibility of this occurring was avoided as far as possible by performing the main experiment in darkness). If differences were found *only* in the straight ahead shift experiment, this may suggest further lines of enquiry. If differences were found *only* in the

¹⁷[since participants are aware that the prisms have been removed]

adaptation experiment, then this is strong evidence for the cerebellar deficit hypothesis from a most surprising source.

Experiment 1 - Straight ahead shift : A control

AIMS: A control study for Experiment 2. An investigation and comparison of susceptibility to the 'straight-ahead shift' and initial effects of prisms on dyslexic and control groups of two different age groups.

Experiment 2 - Prism adaptation study: The main study

AIMS: This study involved participants pointing at targets with either the left or the right hand and with and without prism goggles. The aim was to compare dyslexic and control children of two age groups on speed, amount and type of adaptation.

Issue 1: Assuming that there is a mild cerebellar deficit in dyslexic children, and that prism adaptation is dependent on the cerebellum, children with dyslexia of both age groups should adapt more slowly than the control children.

Issue 2: Again assuming a mild cerebellar deficit in dyslexic children, one might expect dyslexic children of both age groups to produce less negative after effect than control children. Negative after effect is often assumed to be the true measure of adaptation, unaffected by conscious correction and cognitive components.

2.4 Experiment 1 - Straight ahead Shift Experiment (a control study)

Method

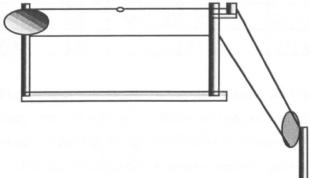
Design

The experiment used a mixed measures design. There were four groups containing 28 participants in total. Independent variables were age (12 or 16 years) and group (dyslexic/control). Dependent variables were the mean and standard deviations of the measurements taken from participants' judgements of straight ahead in each condition. Groups were compared across the repeated measures variable of condition. Measurements were taken in six conditions *in the following order*: (i) in the light with clear goggles, (ii) in the dark with clear goggles, (iii) in the dark with prism goggles, (iv) in the light with prism goggles, (v) in the dark with prism goggles (2) and (vi) in the dark with clear goggles.

Apparatus

A structure was built from 'climpexTM' to enable a luminous bead to be moved along on string by a wheel to the side of the participant (see Figure 2.4.1)¹⁸. The bead was marked with a line down the centre to encourage greater accuracy by the participants and to enable accurate measurements using a scale positioned directly underneath the bead (not shown and scale not visible to the participant). The string was approximately 57cm away from the participant's eyes when they were biting on a bite bar.

Figure 2.4.1. Schematic diagram of the apparatus used for the straight ahead shift experiment (shown from the participants point of view). The participant is asked to move the luminous bead along the string using the wheel to the side of them so that it appears to be straight ahead of them under current conditions.



Other apparatus included the bite bar, prism goggles with 12 diopter fresnel prisms (with removable 'flip up' covers), clear goggles with covers¹⁹ (similar to prism goggles: 'placebo goggles') and a small, dimmed torch.

Note 1:

Twelve diopter prisms (producing a 6° distortion) would be expected to produce a deviation of 6cm at a distance of 57cm. Each prism diopter produces a linear apparent displacement of 1cm at 1m or an angle of apparent deviation of 0.5° . Thus 12 dioptres =6° and:

$$\tan 6^\circ = \frac{x}{57} \qquad \therefore x \approx 6 cm$$

¹⁸Thanks to Phil Duke for his help in the design and building of this equipment

¹⁹The goggles were adapted from standard DIY goggles. Note that the larger type had to be used to enable the wearing of goggles *over* any spectacles required by participants.

Participants

Psychometric data (means and ranges) for the four groups of participants are shown in Table 2.4.1. Individual participant data is given in Appendix 2.4.3. All participants were drawn from the dyslexia panels in the Department of Psychology, University of Sheffield and had taken part in several previous experiments.

Table 2.4.1. Mean psychometric data for the four groups used (range shown in parentheses).

Group	n	Chronological Age	Reading Age	IQ
D12	7	12.5 (11.2-13.5)	10.5 (8.0-13.3)	108.9 (90-126)
C12	7	13.3 (12.3-13.9)	15.2 (12.0-17.0)	114.3 (101-124)
D16	6	16.6 (15.0-18.1)	11.9 (8.9-14.0)	118.5 (101-131)
C16	8	16.7 (15.5-17.2)	>17 (17.0-17+)	116.9 (96-130)

Participants with dyslexia had been diagnosed by a full psychometric assessment. They were of normal or above normal intelligence [operationalised as IQ of 90 or more on the full scale WISC-III (Wechsler, 1976)] and without known primary emotional, behavioural or socio-economic problems. Each participant's reading age or spelling age was at least 18 months behind his or her chronological age. Two age groups were used with mean ages 12.5 and 16.6 years (D12 and D16).

Normally achieving control participants had also been given a short-form psychometric assessment and obtained normal or above normal IQ and reading and spelling ages in line with or above their chronological age. Two age groups were used, approximately matched for chronological age with the group with dyslexia (C12 and C16). Full details of each participant are given in Appendix 2.4.3.

Procedure

Participants Initial Instructions:

The participants were shown that when they turned the wheel, the luminous bead moved from side to side. They were told that during the experiment they were to keep their heads still using the bite bar and that on each trial they were to position the bead so that it appeared to be straight ahead of them. It was explained that they would be wearing goggles on each trial and that occasionally the goggles would be exchanged for a different pair, but that they should continue to keep their heads still (using the bite bar) whilst this was done for them. During trials in darkness, they were asked to replace the flaps on the goggles after each trial (so that they could not see the experimenter using the torch to note their judgement). The participants were briefly reminded of the instructions before the start of each separate condition.

The Conditions:

The clear goggles were positioned first. Participants were asked to bite on the bar and hold their head still from that point on, using the wheel to position the bead so that it was straight ahead of them. After each trial, the bead was moved and participants were asked to repeat the process. This procedure was repeated 4 times for each condition, the bead being moved to quasi-random positions on alternate sides of the apparatus between trials. During the trials in darkness, the participant was reminded to replace the flaps on the goggles between each trial. Measurements were taken (to the nearest mm using the ruler directly beneath the string) in the following conditions:

(i) in the light with clear goggles
(ii) in the dark with clear goggles
(iii) in the dark with prism goggles
(iv) in the light with prism goggles
(v) in the dark with prism goggles (2) and
(vi) in the dark with clear goggles.

Results

For each condition, participants positioned the bead four times. The means and standard deviations of judgements in each set of four trials were calculated. Each mean was then calculated as a 'displacement score' in relation to each individual's mean judgement in the dark with clear goggles on (presumed to be the 'purest' judgement)²⁰. Effects of interest could then be calculated using these measurements for each individual participant (see below and Table 2.4.2).

Three-factor ANOVAs were performed on the calculated data. Factors investigated included age, group and condition. The factor of condition had two levels in each case: the condition under investigation and the baseline measurement of the dark

²⁰The measurement for the dark condition with the clear goggles on was therefore zero in every case. This was important because 'straight ahead' would be slightly different for each participant anyway depending on the exact position of their head.

clear condition (zero). This was in order to test whether or not the condition had elicited a significant effect.

	Effect (please refer to text)					
	a (dark clear prisms)	b (prisms light-dark)	c (light clear-dark clear)	d (light prisms-light clear)	e (negative after effect)	f (dark prisms 2nd-1st)
D12	-6.6 (4.3)	6.8 (4.5)	-1.2 (2.7)		1.5 (5.1)	4.8 (6.7)
C12	-4.6 (1.2)	4.6 (1.7)	-1.1 (1.4)	1.2 (1.2)	2.0 (1.8)	3.7 (2.0)
D16	-6.9 (2.9)	6.2 (3.1)	-0.4 (2.4)	-0.2 (2.4)	1.1 (3.1)	3.3 (3.6)
C16	-6.4 (4.3)	7.0 (5.1)	0.4 (2.9)	0.2 (2.5)	1.4 (1.2)	4.9 (5.2)

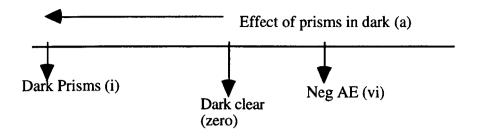
Table 2.4.2. Mean size of different effects (refer to text) for the four groups. Standard deviation shown in parentheses.

Effects investigated

ANOVA tables of all results are shown in Appendix 2.4.1

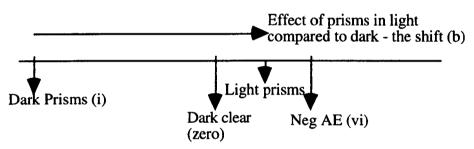
a) Initial effect of prisms in the dark (the dark measurement made with prisms on minus the dark measurement with clear goggles).

A pure measure of the effect of the prisms. As would be expected, this was generally around 6cm to the experimenters left (participants' right): indicated by a negative number. This effect was highly significant ($F_{1,24}$ =88.49, p<0.0001). It can be seen from the means that the young control group seem to be least affected by the prisms at this stage. However, there were no other main effects or interactions (all F's<1).



b) The effect of the prisms in the light, compared to their effect in the dark (a subtraction of the measurement taken in the dark with the prisms from the measurement taken in the light with the prisms).

A positive number, as in this case, indicates that the prisms had less effect in the light than in the dark: i.e. a shift of that amount *back* to the right when the light is turned on. This effect was significant ($F_{1,24}=71.03$, p<0.0001) and as predicted by the straight ahead shift theory. There were no other significant effects (see Appendix 2.4.1).



c) The effect of the light on the measurement with clear goggles (compared with the measurement in the dark i.e. light clear, minus dark clear).

Again, a positive measurement indicates a slight pull to the right, a negative measurement to the left. It can be seen that the effect is mostly small and that the light pulls values slightly to the left in most cases. The effect that the light had on the judgement of straight ahead did not have a significant effect. The judgements made in the illuminated room were therefore not significantly different from those made in the dark. No other effects were significant.

d) The extra effect of the prisms in the light (the measurement taken with prisms on in the light minus the measurement with the clear goggles on in the light).

The real effect of the prisms in the light therefore. A positive value (as in this case) indicates a shift to the right: the opposite direction to that which would be expected using these particular prisms i.e. prisms not causing displacement. The effects are

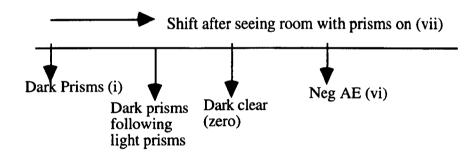
small in all cases. The effect that the prisms had in the light, adjusted for the effect that the light had on the judgement anyway, was not significantly different from zero. No effects were significant.

e) The negative after effect (after removing the prism goggles at the end).

Since the prisms displaced vision left, a negative after effect is indicated by a positive number (a shift to the right). Small negative after effects were obtained for all groups. Overall the effect was significant ($F_{1,24}$ =6.87, p<0.05). No other effects were significant.

f) The second measurement in the dark with the prisms on minus the first.

Essentially a measure of the shift caused by seeing the room with the prisms on; be it due to memory or adaptation. As expected, seeing the illuminated room in between the first and second measurements taken in the dark with the prisms on had a significant effect on judgements ($F_{1,24}=22.62$, p<0.0001). No other effects were significant.



Standard Deviations

In order to investigate the consistency (accuracy) of judgements in the different groups and across the conditions, a three-factor ANOVA was conducted which considered the effects of age, group and condition on each individual's standard deviation of measurements for that condition. The mean standard deviations for each group are shown in Table 2.4.3. The ANOVA table is shown in Appendix 2.4.2. The effect of condition was highly significant ($F_{5,120} = 13.69 \text{ p} < 0.0001$). Judgements in the light tended to have higher consistency than those in the dark. There were no main group or age effects ($F_{1,24} = 2.86$ and $F_{1,24} = 2.47$ respectively).

Table 2.4.3. Mean size (over groups) of the standard deviation of the four judgements made by each person in each condition. Standard deviation between those people also shown It can be seen that judgements in the light were more accurate than those made in the dark and that dyslexic and control groups differ most in their accuracy on judgements made in the dark (standard deviation of the standard deviations shown in brackets).

	light clear	dark clear	dark prisms 1	light prisms	dark prisms 2	negative ae
D12	1.06 (0.50)	3.47 (2.02)	4.73 (2.43)	1.59 (0.75)	2.37 (0.87)	2.99 (2.01)
C12	1.27 (0.79)	3.36 (1.02)	2.68 (1.35)	1.28 (0.73)	2.60 (1.91)	2.79 (1.01)
D16	1.52 (0.71)	3.13 (1.58)	3.51 (1.38)	1.92 (1.49)	2.02 (0.96)	2.06 (1.53)
C16	1.44 (1.05)	2.81 (0.72)	2.48 (0.93)	1.06 (0.57)	1.87 (1.18)	1.85 (1.18)

To further analyse the effect of condition on the standard deviations of each condition, two-factor ANOVAs were performed on each condition individually. These resulted in a main group effect ($F_{1,24} = 6.80$, p<0.05) only on condition (iii: dark prisms for the first time), and no other significant effects on any condition. This showed that children with dyslexia tended to be less consistent the first time the prisms were put on in the dark.

Summary

No significant group or age differences were found between the means of the various conditions. Although the prisms had a significant effect in the dark, and produced a significant negative after effect, the effect of the prisms in an illuminated room was not significantly different from zero. As expected, the effect on viewing an illuminated room on judgements of straight ahead whilst wearing prisms in the dark was highly significant. Analysis of the consistency of the set of four judgements showed that children with dyslexia tended to be less consistent the first time the prisms were put on in the dark.

Discussion of Experiment 1

Results of Experiment 1 do not allow us to reject the null hypothesis. There was no evidence for a group difference in susceptibility to the SAS. It should be noted, however, that CDH would not predict a difference on the straight ahead shift, since it is not considered to be true adaptation, and that this experiment was principally a precautionary measure used in order to reduce the possibility of potentially confounding variables. However, the experiment illustrated the existence of the straight ahead shift, and in particular the role which it can play in adaptation (by the existence of a significant negative after effect at the end of the experiment). In order to be able to accurately measure speed of adaptation therefore, cues for adaptation need to be limited, or else adaptation is likely to occur too quickly. A significant group difference emerged in the condition where the prisms were positioned in darkness for the first time (p<0.05). The groups with dyslexia were more inconsistent in their judgements. This could therefore represent lower sensitivity in the groups with dyslexia or simply more confusion in the different conditions. It is unlikely to represent strategic behaviour in lit conditions because dyslexic and control groups showed equal consistency in the dark clear goggles conditions.

2.5 Experiment 2: Prism Adaptation

Experiment 1 found no significant group differences in susceptibility to the straight ahead shift (SAS). All four groups performed the task within the expected range. These findings therefore confirmed that the dyslexic group do not show differences in the (non-cerebellar) straight-ahead shift component of adaptation. If differences had been found, then these differences may have been able to influence the results of the main prism adaptation study. With no differences found, the main study set out to investigate speed and type of (other components of) adaptation in children with dyslexia and matched controls.

Method

Design

The experiment used a mixed measures design. Four groups (28 participants) separated by two independent variables (age and group) took part. One participant was excluded due to inability to complete the task (a participant from the young dyslexic group²¹). The dependent variable was the displacement which the prisms elicited as measured by the pointing position (in millimetres) relative to the target. Four parts to the study compared (i) initial accuracy, (ii) speed of adaptation, (iii) amount of intermanual transfer and (iv) degree of negative after-effect in either hand. The design is similar to that of many studies in the area (e.g. Weiner et al, 1983), checking initial accuracy to ensure that the prisms have a significant effect

²¹This participant showed extreme left-right confusion rendering his ability to point at lights in the dark completely useless. He also reported double vision and being unable to see the lights at all.

and then measuring speed of adaptation. Negative after effect is measured because it is thought to be the true measure of adaptation. It was considered interesting to examine intermanual transfer of any adaptation in the light of evidence suggesting individual differences in types of adaptation. Behavioural/ cognitive compensation and proprioceptive components other than arm position (such as adaptation of the head position) were controlled as closely as possible by using a bite-bar and also by asking the participants to point where they *saw* the light (as opposed to where it *is*).

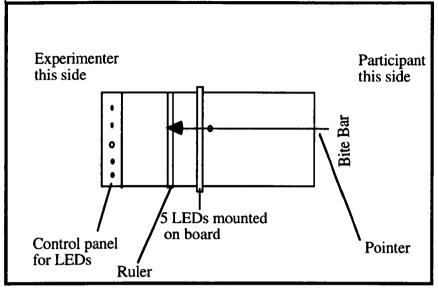
Apparatus

Custom built apparatus was employed in this experiment²²:

Briefly, the task involved pointing at various LEDs in the dark either with or without prisms on, with feedback provided by another LED at the end of each trial. Figure 2.5.1 illustrates a schematic view of the apparatus from above. Five red, dimmed LEDs were mounted on a black wooden board facing the participant, approximately 57cm away from the participants' eyes when they were biting on the bite bar. The five mounted LEDs were controlled from the experimenter's side. A pointer ran underneath this board onto which were attached two further LEDs, one on each side of the board. These LEDs could be lit by a button on the participants' side of the pointer giving a controlled amount of delay (Kitazawa et al, 1995). This mechanism also signalled to the experimenter that the pointer was in the desired finishing position. The pointer continued through to the experimenter's side, pointing to a ruler from which error measurements could be calculated.

²²Thanks to Mr Andy Ham for his technical assistance in making this equipment.

Figure 2.5.1. Schematic diagram of the apparatus used in the present experiment (shown from above). The participant sits on the right hand side and points at the mounted LEDs (lit in turn by the experimenter) using the pointer. Feedback is provided by a second LED, positioned on the pointer close to the mounted LEDs



Further apparatus included a bite bar, a small dimmed torch to enable the experimenter to take measurements and prism goggles with 12 diopter fresnel prisms (as before). The prism goggles were standard safety goggles (which could be worn over spectacles) with prisms attached and painted around the edges in order to obscure any vision other than through the prisms. The prisms were positioned base left, so that they displaced vision to the right. Liberal amounts of black cloth prevented any light (other than that from the LEDs) reaching the participant and allowing them to see either the room or their arm.

Participants

The same participants were used as in Experiment 1. Psychometric data are presented in Table 2.4.1. Experiment 2 followed Experiment 1 after a short break.

Procedure

The participants were asked to sit down and bite on the bite bar in order to keep their heads as still as possible. They were familiarised with the equipment in full illumination (shown the LEDs and the pointer). Participants were asked to use the pointer on each trial to point to where they saw the lit red light. They were told to press the button on the end of their pointer when they thought that it was pointing as accurately as possible to the red light. It was explained that this would light up the light on the end of the pointer and therefore signal the position of the pointer relative to the target. After each trial participants were asked to release the pointer because it would be moved in between each trial. They were told that on some of the trials they would be wearing goggles which the experimenter would position for them, and that the whole experiment would be carried out in darkness. Since participants were aware that there were 2 possible sets of goggles (from the previous experiment), they could not know for certain the effect of the goggles. The room lights were then switched off, and the participants were asked to try pressing the button on the end of their pointer, whilst moving it around, in order to ascertain the effect of hand position on light position. They were asked to do this with both hands. They were then shown the five mounted LEDs. The following measurements were obtained:

i) Measurements on initial accuracy.

Measurements for each five targets were taken three times each for both hands. Participants pressed the button on the end of the pointer for feedback.

ii) Measurements on speed of adaptation with the preferred (usually the right) hand.

The prism goggles were positioned for the participants (firstly with their covers down in order to prevent any immediate realisation of the prism displacement). The covers were then removed in the dark. The participants were asked to use their preferred hand to point to the LEDs, which were lit in a pseudo random order (as shown on the data sheet - see Appendix 2.5.1). Only the central three LEDs were used for this part of the experiment in order to leave room for error. During these twelve trials, participants pressed their button to receive feedback.

- iii) Measurement of intermanual transfer of any adaptation that had occurred.
 Six measurements were taken with participants using their non-preferred hand, without feedback, to check for amount of intermanual transfer of adaptation.
- iv) A further six trials were completed with the preferred hand and with feedback.
- v) The goggles were removed and negative after-effect was then measured in both hands without feedback for 9 trials per hand: the preferred/ trained hand was tested first, followed by the non-preferred/ non-trained hand.

See Appendix 2.5.1 for example data sheet.

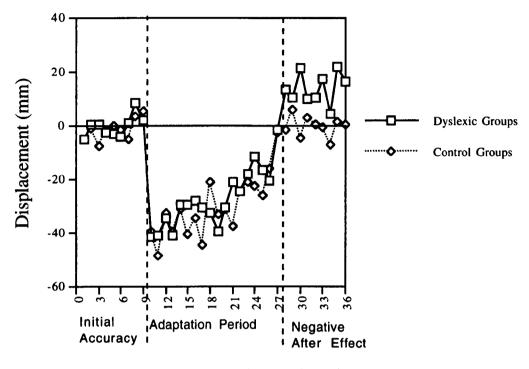
Results

The raw data

The data consisted of displacement from target measurements (positive and negative for left and right respectively) in millimetres for each participant on each trial. Susceptibility to the prism displacement caused negative errors. Negative after effect produced positive errors. The first trial after the prisms were positioned can be taken as an approximate measure of the prisms initial effect.

Groups were compared on initial accuracy, adaptation (expected to be slower in dyslexic groups from the experimental hypothesis), intermanual transfer and negative aftereffect (expected to be less in dyslexic groups from the experimental hypothesis) for each hand. Figure 2.5.2 shows the mean displacement values of dyslexic and control participants (collapsed over age groups) in the three main conditions (initial accuracy, adaptation and negative after effect) for the preferred hand. It can be seen that initial accuracy is reasonable, that the prisms produced a displacement in the expected direction and that there was a small negative after effect in the dyslexic groups. Gradual, although slightly noisy, adaptation can be seen in the adaptation period.

Figure 2.5.2. Graph of the mean displacement values of the dyslexic and control participants (collapsed over age group) per trial in the three main conditions: initial accuracy, adaptation and negative after effect.



Trial Number/ Experimental Period

i) Initial accuracy

Mean results for each of the four groups in terms of initial accuracy are shown in Table 2.5.1 (standard deviations shown in parentheses). Table 2.5.1 shows that there are no obvious differences between groups. The mean absolute errors show that errors had an average of around 12mm deviation from the correct position each time. The standard deviation can be converted to indicate that 95% of the responses would be within around 3.5cm of the correct position if the responses are distributed normally. Thus accuracy was reasonable, but not particularly good.

	Initial accuracy (both hands)					
	signed error/ bias (mm)	absolute error (mm)				
D12	0.49 (17.4)	12.82 (11.7)				
C12	-2.10 (15.7)	12.33 (9.9)				
D16	1.14 (16.0)	12.65 (9.8)				
C16	-0.62 (14.9)	11.31 (9.7)				

Table 2.5.1. Initial accuracy with both hands (standard deviations in parentheses)

Initial accuracy was compared between groups in terms of the 3 different target positions used in later conditions. It is important to consider target position, since accuracy to certain targets might be greater than accuracy to others. In addition, both real/ signed errors and absolute errors were used for both the preferred (usually right) and non-preferred (usually left) hands²³. Multi-factorial ANOVAs were used to examine effects of: group, age, target position, trial number and hand used on both absolute and signed errors (See Appendix 2.5.2 for full ANOVA tables). However, because of the reoccurring effects of hand used in the five factor ANOVAs, four factor ANOVAs were performed on results from each hand separately. For brevity, only these will be reported here.

For the preferred hand, in terms of signed errors, there were no significant effects of age or group (F's<1). Various interactions between individual targets and group were significant, or showed trends (see Appendix 2.5.2). There was a significant effect of target position ($F_{2,48}$ =4.55, p<0.05). The interaction diagram of target position and age ($F_{2,48}$ =8.13, p<0.001) showed that in general, younger groups showed less bias overall. Groups appeared to be equally biased on target two (straight ahead), whereas on targets one and three (to the right and the left respectively) the older groups appeared more biased (to the right and the left respectively). A target-by-group-by-age trend also emerged (F_{48} =2.88, p<0.1). The interaction diagram showed again that generally a negative (right hand) bias was found for target one (right), but a left hand bias for target three (left), and that this was particularly apparent in the older groups, but was not the case in the younger dyslexic group. It also shows that the young controls were the least biased

²³It is interesting to look at both absolute and signed errors, because signed errors may offer indications of biases towards certain directions for groups, whereas absolute errors reflect only actual accuracy, regardless of bias.

group, on average, and for all three targets. For the preferred hand absolute errors, no effects reached significance.

On the non-preferred hand, in terms of signed errors, there were no significant main effects of age or group ($F_{1,24} = 1.04$ and $F_{1,24} = 0.66$ respectively). However, there were significant effects of target position (as before) and a target-position-by-group interaction ($F_{2,48}=9.48$, p<0.0005 and $F_{2,48}=4.18$, p<0.05 respectively). Means showed equal bias on targets one and two for both groups, but an increased positive (left) bias on target three (left) for the dyslexic groups. This result is interesting and seems to tie in with the similar result of most groups getting a negative (right) bias for target one with the right hand. Again, the young dyslexic group did not display this latter effect. In terms of absolute errors for the non-preferred hand, only a target-by-trial-by-group-by-age interaction was significant (p<0.05): this was interpreted as 'noise'.

In summary, there were no significant effects of group on initial accuracy, although target position showed a strong effect.

ii) Adaptation

Several different analyses were performed in order to compare speed of adaptation between dyslexic and control participants. ANOVA tables are shown in Appendix 2.5.2.

Firstly, a regression line was calculated for each individual participant for the 18 preferred hand adaptation trials. The mean coefficients are shown in Table 2.5.2. Adaptation is not generally considered to be particularly linear, although this was thought to be the most objective way to investigate this issue. A transformation for the data was considered, but the individual adaptation graphs did not look especially curved either.

Individual correlation coefficients (r) for the line of best fit calculated ranged from only 0.02 up to 0.61. Some of the individual participant's slopes were actually negative. The mean values are presented in Table 2.5.2. The regression lines did therefore not, in many cases, characterise the actual form of adaptation satisfactorily.

The slope coefficients for each participant were examined in a two-factor ANOVA (age and group) with slope as the dependent variable. The main effects of age and group were not significant ($F_{1,24} = 1.90$ and $F_{1,24} = 0.09$ respectively). No significant age or group differences or interactions were found. It can be seen from the means that the younger groups had slightly steeper slopes (on average) than the older groups. The intercept coefficients were also analysed, but again no significant main effects of age or group were found ($F_{1,24} = 2.03$ and $F_{1,24} = 0.01$ respectively). Younger groups had slightly higher intercept coefficients (less initial displacement) than older groups, particularly amongst the dyslexic groups.

Table 2.5.2. Mean linear regression slope coefficients and correlation coefficients for the four groups. The mean adjusted slope coefficients have been forced to have an intercept of -60mm (the expected prism displacement initially).

	mean slope coefficient	mean r	mean adjusted slope
D12	1.54	0.38	4.28
C12	1.60	0.31	3.31
D16	1.30	0.25	1.47
C16	0.72	0.17	1.75

Following the first regression line analysis, it was considered that groups may have been (and seemed to be) affected differentially by the prisms to begin with. Since this would have an effect on how large their slope coefficients *could* be, slope coefficients were also calculated for each participant forcing the regression line to go through the specific intercept of a displacement of -60 mm (the amount the prisms would be expected to displace vision at this range). Using this method obviously decreased the correlation coefficients, but it had the advantage of making the slope coefficients easier to interpret and more objective, since all groups then had the same initial displacement.

Mean slope coefficients when forced through a -60mm intercept are presented in Table 2.5.2 (adjusted slopes) and were analysed with a two-factor ANOVA. No significant group effect was found ($F_{1,24} = 0.14$), but there was a significant effect of age ($F_{1,24} = 8.21$, p<0.01). The means show that the younger groups appear to be adapting more quickly than the older groups. An analysis (signed values) of the first trial (the prisms initial effect), can explain this difference in part. A significant

age effect was found ($F_{1,24} = 7.51$, p<0.05) in the prisms initial effect, with the older groups being affected by the expected amount (-54mm), but the younger groups being affected less (-27mm). There was no similar effect for group ($F_{1,24} = 0.04$). Forcing the regression line through -60mm therefore, when the younger groups actually started at -27mm is likely to have caused a steeper regression line slope.

As a third type of analysis, since the first analyses were not wholly satisfactory and due to the consistently significant effects of target position found for initial accuracy of both hands, slope coefficients and intercepts for individual target positions were calculated and analysed. Again, this method was not altogether satisfactory either. Results are shown in Appendix 2.5.2. There were no significant effects of age or group ($F_{1,24} = 0.98$ and $F_{1,24} = 0.11$ respectively). Target position did have a significant effect ($F_{2,24} = 3.50$, p<0.05), suggesting that the method was justified. Means showed that target one (right) had the flattest slope (i.e. slowest adaptation) and target three (left) the steepest.

iii) Intermanual transfer of adaptation

No main group, age or interaction effects were found for amount of intermanual transfer of the adaptation (all F's<1). This was as calculated by the displacement of the prisms for the nonpreferred hand after some adaptation of the preferred hand. Trends towards significant intermanual transfer did occur as calculated by a comparison of the initial displacement of the prisms compared with mean values for displacement with each of the three targets in turn. See Appendix 2.5.2 for further details.

iv) Negative after effects

Since in a multi-factorial ANOVA (examining effects of group, age, hand, target number and trial) significant effects of hand used were found ($F_{1,24} = 8.84$, p<0.01), negative after effects were further analysed using two four-factor ANOVAs for each hand separately. Mean negative after effects for groups for both hands are shown in Table 2.5.3²⁴.

For the preferred hand negative after effect, the ANOVA showed a trend ($F_{1,24} = 3.31$, p<0.1) towards a group effect, with the dyslexic groups having larger

²⁴There were several other significant effects in the multi-factorial ANOVA (mostly involving the factor of hand used). These will not be discussed here since they will mostly be reflected by results of the two-factor ANOVAs below (refer to Appendix 2.5.2 for details).

negative after effects than the control groups. This is contrary to predictions of the experimental hypothesis. However, the means show that this effect is mainly due to the older control group having a *negative* negative after effect; the other groups all produce small negative after effects in the expected direction. No other effects were significant. A negative after effect adjusted to account for the amount of adaptation at the end of the adaptation period was also considered (see Appendix 2.5.2), but no main effects were found.

	Preferred Hand	Non-Preferred Hand
D12	15.32 (26.7)	24.67 (25.8)
C12	10.30 (24.1)	18.33 (28.6)
D16	12.3 (27.7)	13.89 (28.8)
C16	-11.21 (22.8)	5.68 (27.3)

Table 2.5.3. Mean negative after effects produced by each group and for each hand (millimetres).

For the non-preferred hand it can be seen that the negative after effect is actually slightly larger than for the preferred hand. This difference was significant ($F_{1,24} = 8.84$, p<0.01). No effects of age or group were found ($F_{1,24} = 2.68$ and $F_{1,24}=1.03$ respectively) and no age-by-group interaction (F<1). There was a highly significant effect of target position ($F_{1,24} = 14.12$, p<0.0001), showing least negative after effect to target one (right) and most to target three (left). A trial-by-group interaction also emerged ($F_{1,24}=5.15$, p<0.01). This indicated that across all the target positions, whereas the dyslexic groups' negative after effect had virtually disappeared. There was also a target position-by-trial interaction which indicated that target two (the central target) was most likely to lose its negative after effect by trial 3, whereas target one (right) was most likely never to have had one. Other results are presented in Appendix 2.5.2

v) Summary of results

No significant group differences were found. A significant age effect was found when slope coefficients were compared for the adaptation period (p<0.01), but only when the regression line was forced to pass through a specified point for each participant. Age and group trends (p<0.1) were found for the preferred hand negative after effect, with children with dyslexia and younger participants getting larger negative after effects than controls. The larger negative after effect in children with dyslexia is contrary to the original hypothesis. However, it was caused mostly by the older control groups' *negative* negative after effect rather than a greater negative after effect in the children with dyslexia. The non-preferred hand negative after effect elicited a similar age trend (p<0.1), but no group trend. Again, the group effect was due mainly to the older controls having no (for the preferred hand) or only small (for the non-preferred hand) negative after effects. Target position had a significant effect in many of the analyses. This reflected the tendency for people to be more accurate to the central target and point too far towards the left on the left hand target and towards the right on the right hand target. Younger and dyslexic groups showed this tendency less. A surprising result was that the nonpreferred hand produced a larger negative after effect than the preferred hand (p<0.01).

Discussion

Main results showed no support for the experimental hypothesis in Experiment 2, although no differences in straight ahead shift (Experiment 1) would be predicted by the cerebellar deficit hypothesis. Some evidence emerged for age related differences in adaptation, but there was little suggestion of any group differences. In addition, those trends which did emerge appear more likely to be related to cognitive, rather than anatomical, mechanisms and were in the opposite direction to that predicted because the controls failed to show an effect, thereby (assuming the controls are normal!) discrediting the idea that the effect is caused solely by the cerebellum. However, the data were generally rather noisy and difficult to interpret (despite several changes to the design of the equipment and the apparatus in order to try to avoid this) and some of the results were rather surprising. In particular, the aberrant performance of the control group for the negative after effect throws doubt on comparisons between groups.

A first look

An initial assessment of the paradigm used showed that the basic shape of the graph for the preferred hand looked as expected (see Figure 2.5.2). Similar graphs produced for each individual group showed similar patterns, with the exception of the older control group, who (as a group) did not produce a negative after effect at all and even seemed to be shifting in the opposite direction. Adaptation was obviously not complete in many cases and this may, in part, account for the rather small negative after effects. In addition, graphs for the non-preferred hand showed that initial accuracy was not as good as that for the preferred hand, but that negative after effects were larger. This finding was not expected or considered in the design

of the experiment, particularly since the non-preferred hand's effect was measured last of all.

However, despite a reasonable general pattern of results, adaptation curves for the groups were far from smooth. Individual adaptation graphs were even less convincing. However, it is interesting to note that other studies often present only group means: sometimes also averaged over several trials. Therefore, is not clear whether or not this pattern of results is usual.

The experiment therefore did not work as well or as 'cleanly' as expected and results were difficult to interpret, largely due to both noise and the large and apparently systematic effect which the position of the target had on the pointing errors. Additionally, adaptation was in many cases not complete after the 18 trials and the negative after effect produced was smaller than expected. Nevertheless, the paradigm did produce the majority of the expected patterns. Thus, it was concluded that the groups could still be compared, but with reservations.

Potential Improvements

Despite extensive pilot work, it is apparent from the data that several improvements could be made to the design of this experiment. The large effect of target position was the most obvious pattern of results that was not initially accounted for. Although all targets were within easy reach for all participants, some targets are clearly easier to reach than others. Because which targets are easier to reach varies depending on which hand is used to point to them, and more importantly because the distortion of the prisms also affects the pointing position in just one particular direction, the effect of target position may have influenced many of the results.

One simple improvement which could be made to the study would therefore be to alter the direction of the prism displacement for half the participants. This would ensure that if a greater negative after effect were found for the 'non-preferred' hand, one could be more confident that this was due to the non-preferred hand genuinely having a greater negative after effect, rather than it being due to the effects of the direction of the prism displacement. With hindsight, it would also be sensible to alter and counterbalance the order in which the negative after effect for each hand is measured so that effects have not had time to wear off (or increase) at all by the time the non-preferred hand is tested (as may have happened to some extent in this experiment). In addition, the data may have been clearer if only righthanded participants were used, or if right- and left-handed participants were analysed separately (although there was only actually one left-handed participant in this experiment).

Another oversight in the design of this experiment was the notion that cognitive compensation components had been removed. The participants were asked to point where they *saw* the target (as opposed to where it was). However, on at least one occasion, it was obvious that conscious correction was taking place. The participant was obviously pointing to where he saw the light and then moving it a clear and determined 6cm. The removal of this component is therefore difficult, particularly with children anxious to perform 'well' and enjoying being able to point near to the target 'against all odds'! The children were unaware that the experimenter could see well enough to see such actions (as far as they were concerned, the room was completely dark). One way around this would be to use variable prisms so that the child comes to realise that a conscious technique is not successful and so that (s)he is not aware of the prisms displacement at any given time anyway.

Interpretation of findings

There are therefore many improvements which could be made to the study together with various associated difficulties in interpreting the existing results. Main findings to explain include the group trend (in the opposite direction to that predicted) for the negative after effect on the preferred hand, age trends for both adaptation speed and negative after effects for both hands, and a larger negative after effect for the untrained hand.

To account for the fact that the nontrained hand produced a negative after effect, one might expect visual adaptation (and therefore intermanual transfer) when exposure is terminal rather than continuous (as in this case). A visual shift is plausible in terms of the possible suppression of the usual domination of vision over other senses because of the lack of continuous visual stimulation (see Welch, 1974). Similarly, Canon (1971) suggested that adaptation occurs in the modality to which least attention is paid. Consistent with this position certainty related explanation (with more uncertainty because of terminal exposure), a trend (p<0.1) emerged towards the non-preferred hand being less accurate in terms of absolute errors made. However, since the nontrained hand had a *larger* negative after effect than the trained hand, confounding effects of target position and hand used are

perhaps a more likely explanation. Furthermore, the non-preferred hand was not deliberately adapted *at all*, so the theory of position certainty was not really designed to account for such situations.

The age trends found in this experiment are far less unexpected. Winn, Gilmartin, Sculfor and Bamford's (1994) found that in participants from 19 to 85 years, the magnitude of adaptation to prisms declined significantly as a function of increasing age. However, the age differences between the groups used in this experiment are relatively small, and adaptation is generally thought to decline in old age, rather than with age from childhood. For this experiment, this finding is perhaps more likely due to position certainty (that younger people are not as aware of their position as older people).

The main result with regard to the original hypothesis was that children with dyslexia produced slightly larger negative after effects. This was contrary to predictions and cannot therefore be directly accounted for by the cerebellar deficit hypothesis. However, Nicolson and Fawcett (1995) show that children with dyslexia have poorer motor skills than control children and it has often been remarked that dyslexic children have tendencies towards clumsiness. It is therefore possible that children with dyslexia have less position certainty and so actually adapt *more* easily. However, cerebellar patients would certainly be expected to have poor motor skills too, but in Weiner et al's (1983) study they showed less adaptation. Thus, perhaps a more likely explanation is that some of the older control group were aware that they may get a negative after effect and compensated accordingly. Alternatively, they may have been performing the task via behavioural compensation rather than any true adaptation taking place (despite instructions implying not to).

Even if the larger negative after effect shown by dyslexic groups is attributed to methodological error, however, both groups still adapted with equal speed in the adaptation period. This was also contrary to prediction. However, the unnatural conditions may have made it possible that neither group were given opportunity to adapt quickly, or more importantly take advantage of all the reported components of adaptation. Equal adaptation may have occurred for this reason. If assimilated corrective responses had been given more opportunity to arise, for example, results might have been different. For example, the cerebellum is thought to be involved in the automatisation of behaviour (see section 1.5), as well as in prism adaptation. It

has been shown that minor alterations in the design of prism adaptation experiments can have major effects on the balance of components of adaptation (see e.g. review by Welch, 1974). It seems that Weiner et al's (1983) experiment was may have been an ideal paradigm in order to elicit an assimilated corrective response (i.e. automatic responding). This was principally due to the fact that the target was always in the same place each time, and that each participant carried out many trials. The present experiment varied on several points which make an automatised response to pointing towards the targets less likely²⁵. Nicolson and Fawcett have found that children with dyslexia have problems automatising skills, so an exact replication of Weiner et al (1983) could potentially elicit results in the expected direction, although not for the originally hypothesised reasons.

It is also possible that although the cerebellum plays some role in prism adaptation, in the cerebellar patients it is not this deficit specifically that is leading to the difference. Subsequent to the present study, Clower, Hoffman, Votaw, Faber, Woods and Alexander (1997) in a PET study on prism adaptation found posterior parietal cortex activation but no cerebellar activation. They postulated that cerebellar participation may be limited to the process of error correction that typically accompanies prism adaptation, a mechanism that could be anatomically as well as functionally distinct from the coordinative remapping between visual and proprioceptive representations.

Welch and Goldstein (1972) compared brain-damaged patients with frontal and non-frontal (general mixture of deficits) lesions with psychiatric patients and found that the brain damaged patients revealed less adaptation than the psychiatric

these differences combined could potentially account for the non-significant results.

²⁵Firstly, there were five targets in all, at least three of which were used in any one condition. Repeated pointing in the same place did therefore not occur. Secondly, participants were (implicitly) asked not to behaviourally compensate for any distortions and to point to where they saw the target. Thirdly, since participants were not always accurate initially, and since this inaccuracy was brought to their attention via the feedback, the extra inaccuracy caused by the prisms (around 6 centimetres) was less likely to be behaviourally compensated for. The fourth difference was the use of the pointer: used in order to obtain more accurate and objective results. In order to prevent bias and additional cues, the pointer was started in a different 'pseudorandom' position each time [It was considered particularly important to vary the starting position and the direction that the participant had to move the pointer because of evidence on dyslexia and past pointing - another similarity to cerebellar patients found by Fawcett and Nicolson 1996. A large amount of adaptation could have probably been simulated by positioning the pointer on the opposite side of the target to which an effect was 'required'. A large prism effect, for example, may have been possible to simulate by starting the pointer towards the left for a target on the right if the prisms were displacing vision to the right. A large negative after effect could be simulated by starting the pointer on the other side). However, precisely because the pointer was started in a different position each time, automatised behaviour (such as that described by Welch, 1974) was again less likely to occur. All

patients, a finding again revealed only on the negative after effect measure. An analysis of the error reduction (ignoring the direction of the error) also found group differences, suggesting that the brain damaged participants were less accurate in their attempts to consciously correct for their errors. It may therefore be the case that any type of brain damage causes problems with adaptation (due to the cognitive component) and the effect is not specific to the cerebellum. Since the cognitive component in this study was removed as far as possible, if brain damage in general (as opposed to the damage to the cerebellum) is the key factor in adaptational differences (and these differences are indeed related to the cognitive component) then differences would not be expected in the present study because the cognitive component had been removed. However, whether Welch and Goldstein's brain damaged patients had damage to the cerebellum as well as the other damaged areas mentioned in the paper cannot be easily ascertained.

A further, perhaps more obvious, explanation for the lack of differences between groups in the present experiment is that any cerebellar deficit in children with dyslexia which may exist is not severe enough to cause a significant deficit in prism adaptation, such as that found in cerebellar patients. Moreover, one cannot necessarily compare adults with acquired cerebellar lesions and children with a developmental disorder. This is particularly true because it seems that younger people are better at adapting anyway (e.g. Winn et al 1993); this simple task may therefore not show up adaptational differences in children.

Yet another alternative is that the cerebellar deficit in children with dyslexia is not in the *area* of the cerebellum necessary for prism adaptation. Baizer and Glickstein (1973) found that only one of five monkeys with cerebellar lesions lost the ability to adapt to displacing prisms. In 1994, Baizer and Glickstein explain these same results as being due to more caudal areas of the cerebellum now being known to be the major target of visual information and lesions elsewhere having little effect on adaptation. It is therefore quite plausible that children with dyslexia do have a mild cerebellar deficit, but not in the area responsible for prism adaptation. Since no area has yet been specified for children with dyslexia, this is obviously something which needs to be worked towards, particularly as the cerebellum is now postulated to be involved in so many different functions and is one of the largest structures in the brain in terms of the number of neurons it contains (Williams and Herrup, 1988).

In conclusion, this paradigm is certainly not showing interpretable differences between dyslexic and control children; reasons as to why (or why not) are difficult to decide between with the available data. Aberrant performance of the control group for the negative after effect makes it difficult to infer anything from between group comparisons. Support for the cerebellar deficit hypothesis was not obtained. One slightly unexpected result was found in the control study: the mean effect of the prisms in the light was virtually nil for the task of positioning the bead straight ahead. This is not particularly surprising in view of the phenomenon of the straight ahead shift or indeed the immediate correction effect. However, it is intriguing that research into prism adaptation that has been performed in a well lit room, (such as, but not exclusively, Weiner et al's experiment with cerebellar patients) has not suffered at all from this phenomenon. Even though most of the research mainly involves pointing as opposed to positioning something, since such a shift is thought to combine algebraically and therefore speed other types of adaptation, the fact that participants do not adapt almost immediately is difficult to reconcile with the data presented here. This is particularly true in situations where participants are aware that what they are supposed to be pointing at is straight ahead of them (such as in Weiner et al's experiment). Differences between the design of the main study presented here and Weiner et al's study on cerebellar patients may be able to account for the lack of support for the cerebellar deficit hypothesis. Furthermore, the age of participants may be important for adaptation studies and mild cerebellar abnormalities may still be present in children with dyslexia, but in a different area of the cerebellum.

Chapter 3

Visual Deficits in Dyslexia?

An indirect investigation of vergence control across saccades in adults with dyslexia

Summary:

Since the prism adaptation line of investigation in Chapter 2 was not highly successful, a new line of investigation was sought. The difference between groups in terms of variability of responses on the straight-ahead shift experiment (Chapter 2) was suggestive of possible visual differences in dyslexia. Simons and Grisham (1987) concluded that there is general support for a relationship between binocular anomalies and reading problems. However, they point out numerous methodological difficulties together with failure to examine the effect of compensatory mechanisms, the susceptibility of the visual system to fatigue and the relationship of several binocular functions in the same study. Experiments reported in this chapter therefore attempt to further investigate the visual deficit hypothesis of dyslexia, addressing issues raised by Simons and Grisham. Furthermore, these experiments examine an ability in adults with dyslexia that has not previously been considered using a novel 'sequential stereopsis' paradigm: ability to control vergence across saccades. The first experiment found no difference in the ability to perform sequential stereopsis between adults with dyslexia and their controls. However, on a control 'simultaneous' stereopsis task, experience on the sequential task improved stereoacuity thresholds for the control group, whereas the thresholds of the group with dyslexia became worse. This finding, together with the large variance in the group with dyslexia suggested the possibility of 'Conscious Compensation' operating. In the second experiment a dual task condition was used to investigate the possibility of Conscious Compensation, but still no differences between groups were found. It was concluded that adults with dyslexia can show normal vergence control across saccades.

3.1 Introduction

As discussed in section 1.2, dyslexia was originally conceived of as a visual problem and anecdotal descriptions of symptoms often included visual difficulties. As early as 1943 it was suggested that a disturbance of binocular vision could be influential in causing reading disability (Park and Burri, 1943). A plethora of research examining visual aspects of dyslexia now exists, from sources including ophthalmic, education and psychology journals [for example, Evans' review (1998), targeted at the practising optometrist, and Kulp and Schmidt's review (1996a) both cite over 120 references, many of which are different]. However, often studies have measured oculomotor performance monocularly, or used rather subjective methods. In their review, Simons and Grisham (1987) conclude that there is general support for a relationship between binocular anomalies and reading problems, but the susceptibility of the visual system to fatigue and the relationship of several binocular functions in the same study is often overlooked. The current study offers comparison of dyslexic and control performance on a task thought to require vergence control across saccades, therefore examining the dynamic operation of two functions together. Thus, some of the more widely cited (and more controversial) papers relevant to these functions will be discussed and some of the various difficulties highlighted. Subsections on poor binocular control, binocular instability and saccadic control (at the risk of ignoring investigations into numerous other factors including refractive error and accommodation) have been included.

Poor binocular control?

One of the most crucial skills in fluent reading is the ability to coordinate the two eyes. It is surprising therefore that many studies concentrate on either stationary (as opposed to dynamic) visual functions, or measure the movement of only one eye. However, at least two studies have focused on the dynamic binocular coordination abilities of poor readers; Birnbaum and Birnbaum (1968) and Bedwell, Grant and McKeown (1980).

Birnbaum and Birnbaum (1968) conducted a study to investigate whether binocular co-ordination difficulties could be a factor in reading achievement. Using binocular and monocular reading conditions, they concluded, on the basis of improvement in monocular conditions, that there are a significant number of children whose reduced reading ability is related to binocular inefficiency. However, large order effects emerged, indicating that the majority of children read less well the first time that they were tested, regardless of condition. This seriously weakened Birnbaum and Birnbaum's argument for binocular coordination difficulties (despite the fact that some counterbalancing did occur). Furthermore, they state that, "It should be noted that the highest percentage of binocular inefficiency problems was found in the best reading groups.". This statement is more troublesome, since it seems to imply that the same basic hypothesis, examined with the same data, but by a different method would elicit vastly different conclusions.

Bedwell, Grant and McKeown (1980) also concluded that problems of dynamic binocular vision and control contribute to poor reading, although their results are probably less convincing. They used a variety of tests of visual function, and found differences both in acuity of stereopsis and in several ratings of video recordings of 'dynamic binocular behaviour' during reading. These recordings were rated by 'observers experienced in this type of work'. On variables such as instability in co-ordinating the eyes on looking from left to right, unequal eye movements, over-convergence and indecision as to the controlling eye, poor readers elicited higher 'presence' ratings than good readers. However, details of the basis and scale on which the ratings were made are far from clear, and therefore appear to be highly subjective (although a reasonable inter-rater reliability coefficient was obtained, suggesting some validity). Furthermore, the study was not performed on children with dyslexia specifically, but also 'garden-variety' (Stanovich, 1988) poor readers. Moreover, the study used reading itself to assess visual differences, which meant that cognitive aspects of the task were likely to be interfering with the eye movements.

Thus, these studies have obvious methodological problems. However, despite this they illustrate that examination of the behaviour of both eyes together may be able to offer additional insights into cases of poor reading, given more objective and controlled methodology.

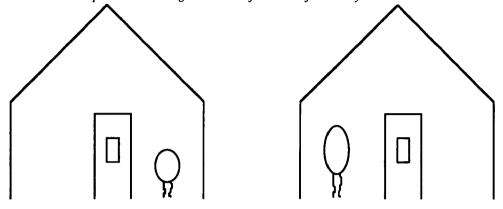
Binocular Instability?

A second visual factor which has been suggested as being influential in dyslexia is binocular stability. Binocular stability refers to whether or not the dominant (or reference) eye remains constant. In order to see in three dimensions, the two eyes receive slightly different images of the same object. One of these images has to be accepted as correct, whilst the other plays more of a 'supporting role' for stereo vision. Thus, the dominant eye is said to provide important directional cues during vergence (Ogle, 1962). Dunlop (1972) states that, "Using lateralised symbols for communication, modern man must accept one image as the standard correct orientation and reject the other as incorrect. Thus, it is essential for one eye to become the master or reference eye in the central binocular region when it is used for interpreting symbols.". There is some evidence that children with dyslexia have an unstable (inconsistent) dominant eye. However, again methodological criticisms have been made, in particular against the Dunlop Test (see e.g. Dunlop, Dunlop and Fenelon, 1973; Dunlop, 1976) which has been used in the majority of investigations of binocular stability and eye dominance.

The Dunlop Test is therefore particularly controversial and has generated much debate over the years (see e.g. Bishop 1989a; 1989b). It involves participants viewing two slightly different slides (see Figure 3.1.1) in each field of a synoptophore. The fields are then gradually pulled apart, forcing the eyes to diverge in order to maintain fusion. Ogle (1962) noted that when the eyes are made to converge or diverge, fused stationary images may appear to move prior to fusion breaking. Stein (1994, p332) states that normal children can diverge their eyes up to five degrees, but then one eye (the 'dominant' or 'reference' eye) continues to track the picture it sees, whilst the other stops tracking its picture and follows that of the dominant eye. Part of the picture followed by the non-dominant eye (the part that is different from the other picture) appears to move as the two images separate. One of Stein's (1994, p332) examples uses two slides of a house, one with a small tree on one side of the door, the other with a large tree on the other side of the door (see Figure 3.1.1). Observers report which tree moves. The test therefore claims to be an indirect measure eye dominance, since eye dominance can be inferred from which part of the picture is reported to move when fusion of the two pictures is broken.

Dunlop et al (1973) first reported that dyslexia was associated with 'crossed reference'; the dominant eye being on the opposite side to the dominant hand. More recent research suggests that lack of a stable and consistent reference eye over several trials of the test, rather than crossed reference, characterises children with dyslexia (e.g. Fowler and Stein, 1980). This finding is now interpreted as reflecting poor binocular stability and vergence dysfunction (e.g. Stein, Riddell and Fowler, 1987, 1988), although this relationship has also been disputed (e.g. Evans, Drasdo and Richards, 1994: see below). Lack of a consistent reference eye is inferred as a possible cause of letter reversals and confusions between letters such as 'b' and 'd' in children with dyslexia. Stein, Riddell and Fowler (1986) confirmed an association between unstable Dunlop test responses and poor reading in primary school children.

Figure 3.1.1: Adapted from Stein (1994). An example of pictures that could be presented in each field of a synoptophore. The pictures are identical except for the trees on either side of the door. When the pictures are fused, a house with trees on both sides of the door is seen. As each field of the synoptophore is gradually pulled apart, fusion breaks and one of the trees appears to move. Whichever tree is reported as moving is said to reflect the reference eye.



Bishop (e.g. 1989b) has been a particularly influential critic of reported findings from the Dunlop Test. In particular, she has criticised Stein et al's (1986) conclusions from the Dunlop test, and studies of monocular occlusion in relation to reading. Criticisms included the failure to consider the influence of IQ (which Bishop, Jancey and Steel, 1979 found to be associated with inconsistent performance on the Dunlop Test); the failure to explain how 24% of good readers could have an unfixed reference eye on the Dunlop Test, and a number of other methodological and statistical flaws. Furthermore, the Dunlop Test itself has been criticised on the basis of its artificiality and complexity for children to perform, and on the high number of false positives it generates (e.g. Stein, Riddell and Fowler, 1986;1987). Moreover, it has since been found, using arguably more reliable methods, that children with dyslexia have lower vergence amplitudes (e.g. Buzzelli, 1991; Eden, Stein, Wood and Wood, 1994; Evans, Drasdo and Richards, 1994) potentially confounding the results. However, not all studies agree (e.g. Ygge, Lennerstrand, Rydberg, Wijecoon and Pettersson, 1993; Bedwell et al, 1980) and some show inconclusive results (e.g. Goulandris, McIntyre, Snowling, Bethel and Lee, 1998).

A lower vergence amplitude means that the eyes can converge and diverge less before fusion breaks. Evans, Drasdo and Richards (1993) helpfully describe some other common synonyms of this term: relative vergences, fusional reserves, fusion amplitudes, vergence reserves and prism vergences. Decreased vergence amplitude would mean that fusion breaks sooner on the Dunlop test, resulting in a decreased amount of time to judge the movements of the pictures. Less time would be available for the judgement whether or not vergence control more generally, or the consistency of the 'dominant eye', influences outcome. Atzmon (1985) found that improving vergence reserves (via orthoptic exercises) in children with reading problems improved their reading. Atzmon failed, however, to consider the possibility of 'Hawthorne Effects' (improvement via experimenter attention) and had no control group of any description. She also relied on self-report of improvement and had follow up times ranging from 6 weeks to a year. However, it is, of course, possible that *both* dominant eye and vergence amplitude factors play a role in the (albeit inconsistent) results found for children with dyslexia on the Dunlop test.

Different methods of measuring binocular stability have also led to some null results. Evans, Drasdo and Richards (1994) found no significant differences between children with dyslexia and their controls in stability of motor ocular dominance on their modified version of the Dunlop test (although there was a tendency for the group with dyslexia to be slightly less consistent). In addition, in contradiction to Stein and colleagues (and the ideas discussed above), stability on the Dunlop test correlated with neither vergence stability nor amplitude. Moreover, the distribution of people with 'fixed' or 'unfixed' reference eyes did not differ between the groups. Using reading-age matched groups, and a monocular verses binocular simulated reading visual search task, they concluded that any ocular motor correlates of dyslexia which they did find were in most cases not causal factors in the reading difficulty.

Similarly, Bigelow and McKenzie (1985) did find an association between reading ability and stability of ocular dominance. However, in contradiction to the idea of an inconsistent reference eye leading to problems of orientation, this instability led neither to more errors nor longer decision times for distinguishing left-right mirrorimage figures. Nevertheless, Stein, Riddell and Fowler (1988, or see 1987) have detected subtle differences in the vergence control of children with dyslexia who have unstable Dunlop test responses (around 64%) using *direct* measures of eye movements in a synoptophore.

It would seem therefore, that although the Dunlop test itself is controversial, it has been beneficial in generating research interest and may also be useful in detecting binocular instability or deficits in vergence control. In addition to Bishop's (1989b) previous criticisms, however, she also argues that binocular instability is not necessarily the cause of the poor reading, but vice versa.

Supporting the case for binocular instability being a causal factor in reading difficulty, Cornelissen, Bradley, Fowler and Stein's (1991) find that an increased print size for those children with dyslexia with binocular instability (as shown by the Dunlop test) alleviates non-word reading errors to a degree. A similar increase in size for those children with dyslexia with stable responses on the Dunlop Test does not help to the same extent. Furthermore, Cornelissen, Bradley, Fowler and Stein (1992), investigated monocular and binocular non-word reading errors in reading-age and IQ matched groups of children with stable and unstable responses on the Dunlop test. A significant viewing condition by Dunlop test interaction was found, with the 'unstable' group making fewer errors in the monocular condition, but the stable group making fewer errors in the binocular condition (p<0.01). It would therefore appear that differences in binocular instability can play a causal role in at least some cases of reading difficulty. Differences between results of different research groups may in part reflect a degree of referral bias, such that, for example, Stein's group appear to have a higher percentage of children with visual deficits in their groups, whereas Snowling's group (who concentrate on phonological deficits) may have fewer.

Saccadic Control

A third important visual factor related to reading, is the ability to make accurate saccades. This factor has also received both much attention and much criticism. Pavlidis (e.g. 1980, 1981, 1985a, 1985b, 1985c), for example, has examined oculomotor abilities of children with dyslexia in his specialised 'ophthalmokinesis laboratory'. In asking participants to follow a light spot target in steps from left to right (e.g. 1981), he found an increased number of saccades in total, and in particular more corrective eye movements and a higher number of regressions back to previous lights in children with dyslexia. The children with dyslexia were unable to accurately follow the sequentially illuminated LEDs and, rather impressively, there was no overlap between the groups in terms of regressive eye movements.

Pavlidis' work has also been particularly controversial. Although some have replicated his work during reading tasks (e.g. Griffin, Walton and Ives, 1974, and Zangwill and Blakemore, 1972), few have replicated it using non-reading tasks (e.g. Elterman, Abel, Daroff, Dell'Osso and Bornstein, 1980, but only in two out of five children with dyslexia in non-reading tasks, both of whom also had history

of seizure disorders) and many have failed to replicate the results at all (e.g. Brown, Haegerstrom-Portnoy, Adams, Yingling, Galin, Herron and Marcus, 1983; Olson, Kleigl and Davidson, 1983; Stanley, Smith and Howell, 1983; Black, Collins, DeRoach and Zubrick, 1984a and 1984b). Biscaldi, Fischer and Aiple (1994) compared children with dyslexia and controls on 75 eye movement parameters on 5 'Pavlidis type' tasks. Again, Pavlidis' finding of excessive regressive eye movements to previous targets in participants with dyslexia was not replicated. However, differences between children with dyslexia and controls were found, including poorer fixation quality and failure to 'hit' the target first time. It is possible that some of the differences may, in part, result from differences in reading experience.

Biscaldi et al (1994) also divided their participants with dyslexia into those with and without additional cognitive impairments, and found differences between those two groups in terms of size and frequency of saccades. Strangely, however, only movements and fixation times of the left eye appear to have been measured. Hendriks (1996) gives a brief overview of the highly disjunctive nature of eye movements during reading, and reading type tasks. Briefly, it seems that both eyes ought to be measured together to make the best use of data, and also that the direction of the saccade needs to be taken into account. Evidence from Bedwell et al (1980) also suggests a need to consider both eyes together. Enright (1984) reports that post-saccadic vergence is not symmetrical (i.e. each eye contributes a different amount to the movement) and is dependent on whether the target is underor overshot by the saccade. Enright (1984) also comments that the *dominant* eye in the people that he tested was often the one on target, with the other eye contributing most to the post-saccadic vergence. In view of these findings, together with the evidence concerning eye dominance in dyslexia, measuring both eyes (preferably working together) would be a far more reliable line of investigation if inferences are to be made regarding oculomotor control. An important part of oculomotor control is the co-ordination of both eyes and this holds true whether or not evidence concerning eye dominance differences in dyslexia are considered valid.

Fewer experiments have investigated visual abilities in *adults* with dyslexia, although Fischer, Biscaldi and Otto (1993) report differences in particular aspects of their saccades (number, reaction times, amplitudes, consistency of target acquisition, number of anticipatory responses and fixation durations) in tasks requiring sequences of saccades and single saccade tasks. These differences were found only in a group with persistent and severe difficulties, however, and those

with less severe difficulties were more similar to the control group. One explanation discussed for these differences is a deficit in attentional control over the saccadic system; Fischer et al (1993) suggest that those adults with dyslexia in the group with severe difficulties (who exhibit more anticipatory saccades and shorter fixation durations than the controls) may have an inability to engage attention (thereby leading to premature saccades), whereas the other group (who exhibit longer latencies than the controls), may have difficulty disengaging their attention. Fischer and Weber (1990) found similar differences between teenagers with dyslexia and age-matched controls.

From a review of the previous investigations into dyslexia, therefore, it is evident that, particularly in the area of oculomotor control, objective but non-complex and non-artificial tests are needed which examine the performance of both eyes working together. The task should not involve reading, although, for relevance and explanatory power, it should require at least one of the many visual skills required in reading. The studies described here attempt to investigate a task that has not yet been investigated in dyslexia, one that is thought to rely on accurate and reliable binocular control: ability to perform 'sequential stereopsis'.

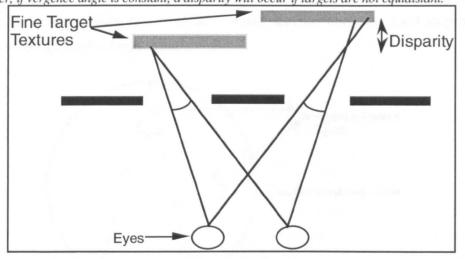
3.2 What is 'sequential stereopsis'?

Sequential stereopsis is a technique which has been used by Enright (e.g. 1991; 1996) in order to demonstrate that relative disparities are not always necessary when making judgements of equidistance. Enright (1991) replicated the finding of Ogle (1956) that better discrimination of the distance between two targets can be achieved with free fixation (looking back and forth between targets) than with fixation held on one target. Ogle had previously suggested that the increased accuracy with free fixation could result from stereopsis occurring in the midst of saccades, allowing a view of both targets together complete with their relative disparities. Enright (1991), however, showed that even when it was ensured that depth information from both targets *could never be resolved at the same time*, in the midst of a saccade or otherwise, more accurate judgements could be obtained from free, rather than held, fixation.

In 1996, Enright described a simple apparatus for investigating the underlying perceptual mechanisms of this phenomenon. The apparatus involves viewing two targets through two separate apertures (see Figure 3.2.1) positioned symmetrically about the median plane of the observer. By using fine textures for the targets,

when one target is fixated through its aperture, depth cues from the texture of the other target, seen through a separate aperture, can not be resolved because the target lies in peripheral vision. The task is to adjust the targets to the same depth from the observer.

Figure 3.2.1- Enright's proposal of how sequential stereopsis operates. As each target is fixated, depth cues from the other cannot be resolved. However, by making saccades from one target to another, if vergence angle is constant, a disparity will occur if targets are not equidistant.



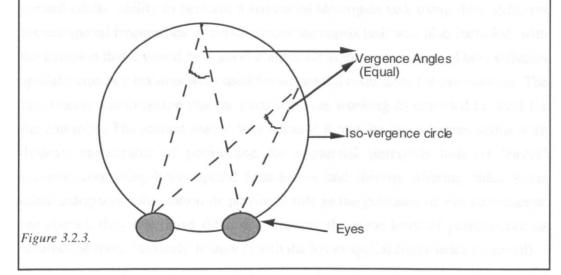
This apparatus neatly precludes the operation of what might be termed 'classical' or 'simultaneous' stereopsis based on relative disparities between simultaneously visible targets, since depth cues from the other target cannot be resolved. Instead, Enright argues that the relative distances between targets has to be judged by viewing them sequentially with saccades back and forth between them, and comparing absolute disparities obtained at each fixation (see Figure 3.2.1). This would require the capacity to make iso-vergent saccades: "Any differences between the eyes in saccadic excursion...would introduce variability into post-saccadic disparities, thereby degrading reliability of sequential comparisons "(Enright, 1991, p1559). In other words, error in vergence angle across saccades would alter the absolute disparities measured at the start of each new fixation, thereby introducing an error in judging equidistance and hence less good sequential stereoacuity thresholds.

Enright's sequential stereopsis paradigm would therefore seem to offer a convenient indirect method of investigating vergence control across saccades. Good stereoacuity seems to be possible without the presence of any relative

disparities, but this relies on the ability to make iso-vergent saccades and therefore have good control of vergence across saccades¹.

NOTE 1:

An important point, is that the technique of sequential stereopsis relies on the targets lying symmetrically about the median plane on an iso-vergence circle. An iso-vergence circle is shown in Figure 3.2.2 below. Note that the vergence angle is the same at any fixation point on the circle, hence if targets were positioned on the circle about the median plane, an iso-vergent saccade could be used to produce accurate judgements of equidistance.



Thus, two experiments are presented here in order to investigate the ability of dyslexic adults to control their vergence across saccades (a skill which is likely to be crucial for fluent reading). This was performed in an objective (although indirect) way. The first experiment investigated the ability of adults with dyslexia to perform sequential stereopsis in comparison to their controls. It also included a control simultaneous (or classical) stereopsis condition. The second experiment aimed to investigate the automaticity of this ability by using a dual task paradigm.

¹However, Frisby, Catherall, Porrill and Buckley (1997) showed that Enright (1996) may not have completely eliminated the possibility that low spatial frequencies were present in the fine sandpaper textures that he used. This may have therefore aided observers' stereoacuity based on relative disparities. A high bandpass filtered texture which eliminated low frequency components (below 16 cycles per degree) was found to elicit higher (worse) stereoacuity thresholds than the fine sandpaper texture used by Enright. Nonetheless, a reasonably good stereoacuity level was still found with these textures. Performance on the highest frequency texture was certainly far more accurate under sequential stereopsis conditions than under fixation and monocular conditions. Frisby et al (1997) concluded that Enright was correct in his conclusion that good stereoacuity can occur without the presence of any relative disparities. They suggested that performance may be worse with higher frequency textures because low spatial frequency components either play a role in guiding eye movements and /or help solve the stereo correspondence problem.

Perhaps also of interest is that Stein (1994) claims that the role of the right posterior parietal cortex would be vital in a task of this type and that this area may be implicated in dyslexia.

3.3 Experiment 1

This experiment compared the ability of adults with dyslexia and their controls to perform a sequential stereopsis task using apparatus similar to that described by Enright (1996) and Frisby et al (1997). A shortened version of this chapter has been published in Ophthalmic and Physiological Optics (Moores, Frisby, Buckley, Reynolds and Fawcett, 1998). The first experiment investigates dyslexic and control adults' ability to perform a sequential stereopsis task using three different texture spatial frequencies. A simultaneous stereopsis task was also included, with the intention that it would be a good control for static stereoacuity. Three different spatial frequency textures were used for sequential conditions for two reasons. The first reason was to ensure that the paradigm was working as expected (at least for the controls). The second reason was because it may be that whereas adults with dyslexia are capable of performing the sequential stereopsis task on 'easier' textures (containing lower spatial frequencies and thereby offering either some relative disparity information or playing a role in the guidance of eye movements: see above), they may have difficulty reaching the same level of performance as controls on more 'difficult' textures (with the lower spatial frequencies removed).

Method

Participants

Participants with dyslexia in both experiments (n=17) had been diagnosed with dyslexia (by either the author or other qualified psychologists under supervision in the department) using a full psychometric assessment.

Dyslexia in adults was defined by a combination of factors. Firstly, an IQ score (Wechsler Adult Intelligence Scale: WAIS-R, Wechsler, 1981) of 90 or greater was necessary. Following this, a number of positive signs of dyslexia had to be present from a set including specific deficits on key WAIS-R subtests (the ACID profile: Arithmetic, Coding, Information and Digit Span). Other signs included deficits on WORD spelling, nonsense word reading speed and accuracy and previous childhood diagnosis. This was performed in accordance with the method developed by Nicolson and Fawcett (1996). This method was used since the discrepancy between reading age and chronological age is of little value in

diagnosing dyslexia in adults, particularly high-achieving students, since they may well have effectively caught up with their reading over a period.

NOTE 2:

An overview of the method of diagnosis developed by Nicolson and Fawcett (1996) is that scores on the Adult Diagnostic Index (ADI) range from 0 to 4. One ADI score is given for previous diagnosis (by a full psychologist's report) of dyslexia. A second ADI score can arise from the spelling age obtained on the WORD test of spelling: half a point is awarded for a spelling age of between 16 and 17 years, a whole point for a spelling age under 16 years. The third possible ADI score is awarded on the basis of performance on a nonsense word reading passage which is timed: half a point is available if more than seven errors are made and the other half if longer than 59 seconds is needed to read the passage. The final possible ADI score can be obtained by having specific deficits (the ACID tests) on the WAIS-R IO test. On the WISC test (Wechsler Intelligence Scale for Children: Wechsler, 1992) children with dyslexia frequently exhibit normal or good scores on most of the subtests, coupled with unexpectedly low scores on two or more of the Arithmetic, Coding, Information and Digit Span subtests (e.g. Ackerman, Dykman and Peters, 1976 or see Wechsler, 1992). One ADI score can be obtained by having deficits on the WAIS-R on two or more of these tests, half an ADI score is awarded for one deficit. An ADI score of 2.5 or greater indicates dyslexia and a score between 1.5 and 2.5 borderline dyslexia.

The adults with dyslexia used in this experiment consisted only of those with an ADI score of 2.5 or more. They therefore had either a previous conventional diagnosis coupled with persisting problems in more than one domain, or a wide range of deficits typically associated with dyslexia such as persistent spelling deficits, difficulty with nonsense word reading and memory or coding difficulties, all within an average or above average IQ. A rationale for, and summary of, the method of diagnosis is presented in Appendix 3.3.1.

Control participants reported no spelling, writing or reading difficulties either presently or in the past.

Dyslexic and control participants were all university students or graduates. Unfortunately, detailed optometric data were not available on the participants so it is unclear whether they manifested any binocular vision anomalies on conventional clinical tests, although scores on the Randot stereotest showed all participants to have stereo vision.

Group with dyslexia: 3 females, 4 males; mean age = 23.8 years (18 years to 31 years), mean acuity on the Randot stereotest around 36 seconds of arc, ranging from 20 to 70 seconds of arc.

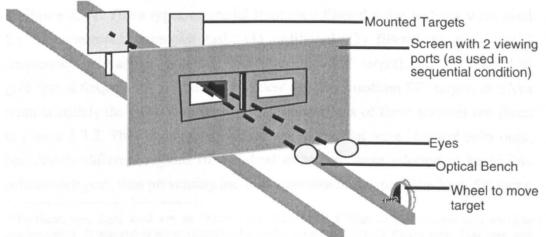
Control group: 4 females, 4 males; mean age = 21.4 years (20 years to 23 years), mean score on the Randot stereotest around 21 seconds of arc, ranging from 20 to 25 seconds of arc.

A Mann-Whitney U test showed that differences between groups in terms of level of stereoacuity on the Randot stereotest were significantly different (p<0.05), with the group with dyslexia showing worse performance.

Apparatus

A schematic diagram of the basic apparatus is shown in Figure 3.3.1. The shutter, chin rest, lighting (and extra apparatus for the dual task condition used in Experiment 2) are not shown in the diagram. The apparatus is closely modelled on that of Enright (1996) and Frisby, Catherall, Porrill and Buckley (1997).

Figure 3.3.1- A schematic diagram of the basic apparatus used in Experiments 1 & 2. Different textures could be mounted on unseen frames as targets. A wheel to move the right hand target was positioned to the right of the participant. The thicknesses of the portholes were different on each side to preclude the possibility of obtaining accurate error judgements on the basis of porthole-to-stimulus distances.



Two mounted targets were placed on optical benches, one positioned in front of each eye. The right hand target could be moved by means of a wheel positioned near the participant. The other target could be moved along the bench only by an experimenter. A combined bite bar and chin rest was positioned at one end of the two benches in order to keep the head still. An electronic shutter (operated by a switch to the side of each participant in Experiment 1, or by computer in Experiment 2) prevented view of all the apparatus when closed (whilst the experimenter was positioning the targets between trials).

The targets were equally illuminated with a fluorescent strip light and a desk top light which together created an illumination level similar to normal room lighting². There was no appreciable variation in illumination with distance of the target.

Viewing ports for both conditions were 4mm thicker (and therefore nearer to the participant) on the left hand side. This difference between the thicknesses of the sides of the viewing ports was introduced by Enright (1996) in order to check whether participants based their judgements on stimulus-to-port distances. If participants were to attempt this, they would obtain a high percentage error because their settings would be consistently too far forward or too far back.

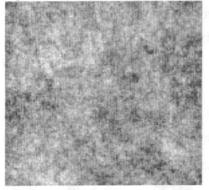
Frisby et al (1997) showed that monocular performance was very poor for the same stimuli and apparatus used in the present experiments. This is important because it illustrated that the task is not possible to perform on the basis of monocular cues alone, such as changes in the levels of illumination with depth, or patterns in the stimuli.

In the sequential stereopsis conditions each target was a printed spatial frequency filtered noise texture seen through its own aperture (40mm by 28mm) as illustrated in Figure 3.3.1. Three types of spatial frequency filtered noise textures were used for the sequential stereopsis task: (1) unfiltered, (2) filtered to give spatial frequencies only above 16 cycles/ deg (the 'high SF' target), and (3) filtered to give spatial frequencies only above 4 cycles/ deg (the 'medium SF' target) at 57cm (approximately the viewing distance used). Illustrations of these textures are given in Figure 3.3.2. The two targets used on any given trial were identical print outs, but slightly different regions of the sheet of texture were selected to be visible behind each port, thus preventing use of feature matching cues. The high SF target

²The fluorescent light used was an 'Auto Twin' light (two 8 Watt tubes) attached to a variable power supply. It was run at approximately 15 volts, creating a 59kHz flicker rate. This rate was therefore undetectable to the human eye. The desktop light was 60 Watts and was run off mains supply (50Hz). I thank the referees of the Moores et al (1998) paper for pointing out the importance of this information, as conventional fluorescent lighting may impair performance on some saccadic tasks (see e.g. Kennedy and Murray, 1991).

has been shown previously (Frisby et al, 1997) to fall below resolution at 7 degrees eccentricity (the position used³) and therefore satisfy the requirement for sequential stereopsis, i.e. elimination of all relative disparity mechanisms using simultaneously visible targets at the target separations used. Frisby et al (1997) originally estimated that the particular frequency/ contrast combination would fall below resolution threshold at this eccentricity on the basis of work by Rovamo, Virsu and Narasen (1978).

Figure 3.3.2. Unfiltered, medium and high textures: a representation.



Unfiltered

Medium SF



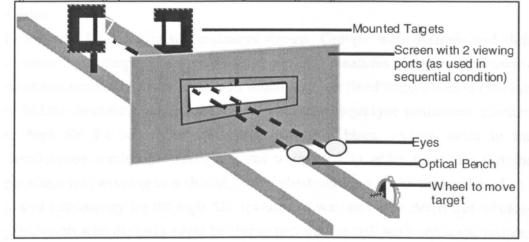


³ With respect to observer (approximately 57cm away), the two nearest edges of the target textures *that can be seen binocularly* were at an angle of approximately seven degrees apart.

In the simultaneous stereopsis condition, the two individual apertures were replaced by a viewing port with a single wide aperture (118mm by 28mm) which allowed a view of both targets together (see Figure 3.3.3). The targets were strips of black card, of slightly different widths (5mm and 6mm: in order to avoid the possibility of participants using monocular feature matching cues to do the task) mounted on unseen frames. Again, one side of the aperture was thicker than the other.

Participants could operate the shutter themselves, thereby allowing them to close the shutter when they felt that they had positioned the target as accurately as they could.

Figure 3.3.3- Schematic Diagram of Simultaneous Stereopsis Apparatus. Black strips of card are mounted on unseen frames which can be moved by the wheel to the side of the participant. A single wide viewing aperture allows a view of both targets together.



Procedure

Participants' instructions were simply to line up the targets so that they were equidistant from themselves. An example of equidistance was provided by the experimenter, using pens as targets. None of the participants had any difficulty grasping this concept. They were told that the wheel to their right could be used to move the right hand target, and that they should be as accurate as possible in their judgements. They were asked to rest their chins on the chin rest and bite on the bite bar in order to keep their heads still.

Judgements of equidistance were made in blocks of six. The right moveable target was displaced by at least 1 cm either in front or behind the fixed left hand target

during inter-trial periods when the participants' shutter obscured the participants' view and whilst they released the adjustment wheel.

The moveable target was set before each trial to random starting positions within around 8cm of equi-distance. Participants replaced the shutter themselves after each trial with a switch positioned to their right hand side. There were two blocks of six settings for each target type at both of the fixed positions for the left target. Stimuli were presented in a different pseudorandom order for each subject using the following constraints: the same fixed position did not appear more than twice in a row, the same texture did not appear more than twice in a row, and no exact combination of texture and starting position was repeated in sequence. Two of the four simultaneous stereopsis blocks were performed first as it was thought to be the easier condition (and hence useful as training), but the last two blocks were also devoted to simultaneous stereopsis to counter-balance for effects of fatigue, boredom and practice.

Design

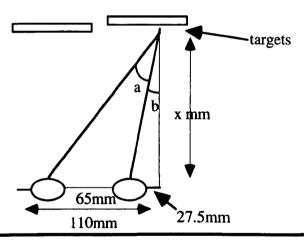
Experiment 1 used a mixed measures design. Groups with dyslexia and their controls were compared across the other repeated measures variables of: viewing condition (simultaneous or sequential stereopsis), left fixed target position (537mm or 562mm from each individual subject's eyes) and target type (unfiltered, medium or high SF for sequential stereopsis, but only black vertical strips in the simultaneous condition). Three textures were used in order to ensure that the paradigm was working as it should, with highest stereoacuity for the unfiltered and lowest stereoacuity for the high SF, textures. It was also considered that whereas participants with dyslexia might be able to perform the task with unfiltered textures (where some relative disparities are present), they might have difficulty with high SF textures.

The dependent variable was the position of the right moveable target when set to appear equidistant with the fixed target. Each block of six equi-distance settings was used to calculate a disparity threshold and an absolute percentage error of the mean setting from the distance of the stationary position from the observer's eyes.

To calculate the disparity thresholds in seconds of arc, the vergence angles adopted for fixation of each target were calculated for each trial. The disparity for each trial was then computed as the difference between these two angles. The disparity threshold for each block of six trials was then taken as the standard deviation of these disparities, based on the assumption that if this task is performed accurately and reliably, there would be only minor dispersion in the six values calculated. For each condition, the mean disparity threshold for each observer was calculated from the thresholds of the blocks of trials used.

NOTE 3:

Vergence angles adopted for fixation of each trial were calculated (using trigonometry) from an estimate of interocular distance, together with the position of the target. For example, assuming an interocular distance of 55mm, since the centres of each viewing port were 110mm apart, two triangles containing 90 degree angles could be constructed with bases of 22.5mm and 87.5mm, and length of xmm (the observed target position). The angles could then be calculated using tan $x^{\circ} = opposite \ side/ \ adjacent \ side$. The vergence angle in question could then be calculated by subtracting the smaller of the two angles opposite the base of the triangle away from the larger (e.g. a-b).



Values for the absolute percentage error of the mean setting were also calculated⁴ by expressing the absolute error as a percentage of the viewing distance to the left fixed target. This calculation used the sum of the observed values minus the correct values (ignoring signs), multiplied by: one hundred divided by the number of observations multiplied by the correct distance from the eyes:

$$Absolute\% error = \frac{|setting - correct| * 100}{(n * correct distance)}$$

Results

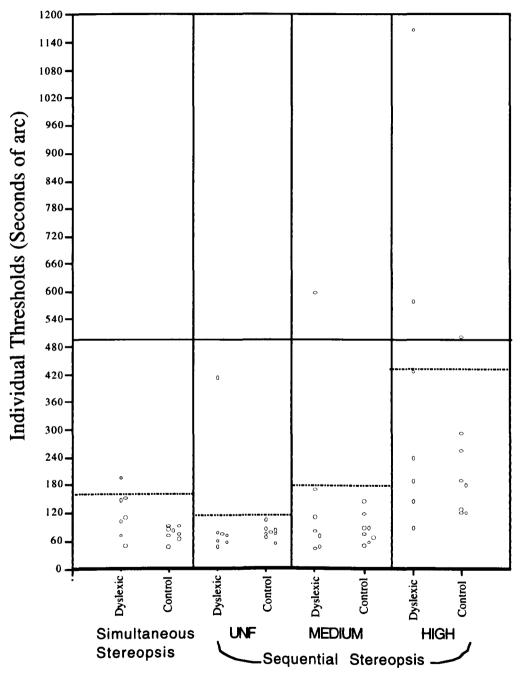
Experiment 1

Figure 3.3.3 plots the disparity thresholds of all participants for the various conditions. It can be seen that the majority of participants perform the task well, but that there is an obvious outlier with very high stereoacuity thresholds on all the sequential stereopsis conditions. It can also be seen that, as expected, thresholds are generally higher for the higher spatial frequency textures. However, there are no obvious differences evident between the groups. Various analyses will be reported that confirm this impression.

Another point concerns the bold horizontal line demarcating a threshold value of 500 seconds of arc. There are grounds for believing that thresholds above this line would reflect an inability to do the sequential stereopsis task. The reason for this is that Frisby et al (1997), using the same apparatus, found that their three practised observers obtained thresholds around 600 seconds of arc for the high SF texture when restricted to monocular vision. In that condition, the observers reported that doing the sequential task felt like guessing and the poor accuracy of their settings was consistent with those introspections. For practised observers in Frisby et al, binocular and sequential performance on the high SF condition was no higher than 400 seconds of arc. Five hundred seconds of arc was therefore taken as a midpoint value, increasing the 400 seconds of arc value for the practised observers to allow for effect of experience, but below the 600 seconds of arc point that seemed like guessing.

⁴ Unsigned error values: whether the error resulted from the target being positioned too near or too far was ignored and only the *size* of the error itself was included in the calculation. This was as in Frisby et al (1997). Signed errors can give a false impression as large error values made either side of the mean can cancel each other out leading to a small and misleading signed error. Absolute errors still reflect the true extent of the inaccuracies.

Figure 3.3.3. Individual participants' disparity thresholds. The solid black line indicates the threshold value considered as a reasonable level of performance; participants with threshold values above this line on any texture were excluded from some analyses. The dotted lines indicate the effect of the exclusions on the highest individual threshold levels on each of the textures.



Texture

In the present study, if the participants with dyslexia were seriously disadvantaged, *ex hypothesis*, by poor vergence control across saccades then it would be expected that a greater number of them would achieve thresholds higher than around 600 seconds of arc for the high SF texture. In fact, very few participants produced a level of performance above 500 seconds of arc: just 2 participants with dyslexia and 1 control participant. The data were analysed both including and excluding data from these participants, however, and the outcomes are similar in both cases. Analyses for the sequential conditions are reported here with these participants included as the exclusions had little or no bearing on the results.

Despite the careful method of calculation, some of the participants' thresholds did appear to be substantially altered by a single value. This was especially noticeable in the case of one participant from the group with dyslexia, on the medium frequency texture, where their standard deviation was increased from 48.73 to 508.2 because of one value, presumably because of some kind of lapse in concentration or mistake on the their part. The unusual value was noticed at the time as it was actually the last of the six measurements on that texture, after performance on the other five trials had been excellent. This changed AL's mean disparity threshold from 53.9 (without the single high value) to 168.7; a substantial increase. A similar pattern occurred for a few of the participants (but not in such a striking manner within such otherwise consistently accurate performance).

In Enright's (1991) first paper on sequential stereopsis, he uses a percentile calculation that excludes extremes in order to avoid a disproportionate influence of outliers. This involved taking the fifth largest value in each block of six settings from the second largest and dividing the outcome by 1.347. The divisor is taken from work by Tate and Clelland (1957), and is said to provide an unbiased estimator of the standard deviation of a normal distribution from size six samples (Enright, 1991). This method of calculation was also used for analyses, but again, the results were similar to those obtained without this equation.

Figures 3.3.4 and 3.3.5 show the group mean disparity thresholds and the absolute percentage errors for the sequential and simultaneous stereopsis conditions calculated in the same way as Frisby et al (1997) and Enright (1996). Both were analysed using a two-factor mixed measures ANOVA.

In the sequential stereopsis analysis, the main effect of texture was highly significant, as expected from Frisby et al (1997), with the 'high SF' texture leading to more inaccurate judgements ($F_{2,26}$ =16.87, p<0.0001 for disparity thresholds, $F_{2,26}$ =8.70, p<0.001 for absolute percentage errors). The independent groups factor (dyslexic vs. control) was not significant and there were no significant group by texture interactions ($F_{1,13}$ =1.34 and $F_{2,26}$ =1.67 for thresholds, $F_{1,13}$ =0.47 and $F_{2,26}$ =1.49 for absolute percentage errors). Analyses were also performed using the method described in Enright (1991) in an attempt to remove the disproportionate influence of outliers. Texture continued to have a significant effect ($F_{2,26}$ =14.9, p<0.0001) and group effects and interactions were still not significant (F<1 and $F_{2,26}$ =1.30 respectively).

Since the sequential stereopsis data may not have been normally distributed, a nonparametric test RANOVA (Random Analysis of Variance) was also conducted on the data. This test makes no assumptions about distribution. Instead, an F ratio is calculated for the observed data and then the data are randomly rearranged a given number of times (10000 in this case) and a new F value calculated enabling a distribution of F values to be constructed (the randomisation distribution). If the obtained F value appears to be an improbable value in the distribution, then the null hypothesis can be rejected. The number of F values in the randomisation distribution that equal or exceed the observed value gives an indication of the probability of obtaining the data by chance⁵. For the threshold values, the p values elicited from this test were very similar to those from the ANOVA (p<0.0005 for the texture effect and n.s. for the group effects and interactions). For the error data, the RANOVA elicited a p value of p<0.0001 for the texture effect and nonsignificant p values for the main group effect and the interaction. The original ANOVA test was therefore considered to be robust enough to cope with the majority of deviations from normality that had occurred in this data set.

⁵This method is based on one proposed by Manly (1991, p.77) and was implemented in the RANOVA program by Dr. Adrian Simpson in the Department of Psychology, University of Sheffield.

Figure 3.3.4: Bar graph of mean disparity thresholds (seconds of arc) for each group by target type. Standard error bars shown.

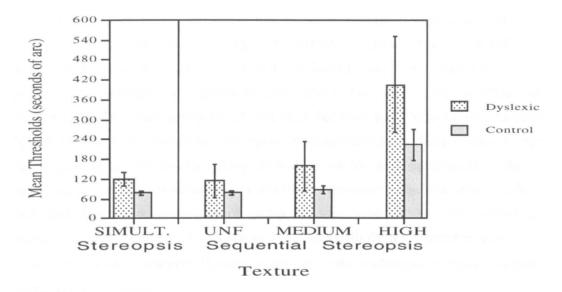
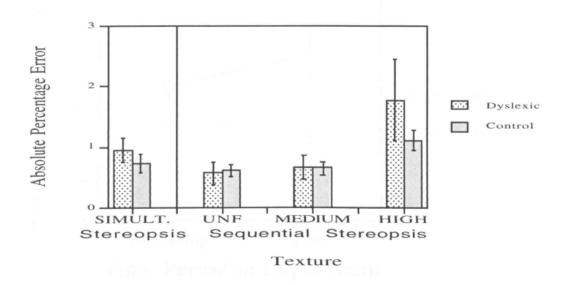


Figure 3.3.5: Bar graph of the mean absolute percentage errors of the mean setting for each group by target type. Standard error bars shown.

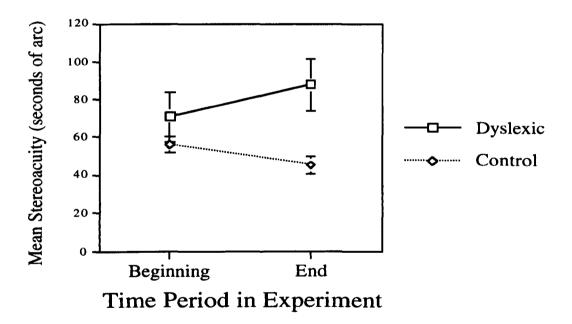


In the simultaneous stereopsis condition analyses were performed including all participants. This was because the condition was originally intended as a control for ordinary stereoacuity. Group mean disparity thresholds were slightly higher for the group with dyslexia than for the control group (117 vs. 75 seconds of arc), but differences were not statistically significant (Mann-Whitney U=13, U prime = 43, Z = -1.74; this test was used because the variances of the two groups in this condition were significantly different, F_{6,7}=11.52, p<0.01). A trend did emerge,

however (p<0.09) towards the group with dyslexia having lower stereoacuity, as might be expected from results of the Randot stereotest.

Two of the simultaneous stereopsis blocks were performed first, and two last. Therefore in order to investigate any order effects, a mixed measures RANOVA was carried out on the means of the first and last blocks. This resulted in a main group effect (p<0.05) and a group by time (first or last) interaction (p<0.05): see Figure 3.3.6. The main group effect indicated that with the additional data created by splitting the mean thresholds into those obtained before and those obtained after the sequential conditions, the group with dyslexia showed significantly reduced stereoacuity on the simultaneous condition. Moreover, the interaction effect indicated that whereas the control group became better at the task following experience on the sequential conditions, the group with dyslexia became worse.

Figure 3.3.6. Group-by-time period interaction diagram for the simultaneous stereopsis condition. Standard error bars shown.



Discussion of Experiment 1

This experiment partially replicated the results of Frisby et al (1997) in that it showed the expected pattern of thresholds for the different textures, with higher frequency textures eliciting higher disparity thresholds. However, no group differences were found on the sequential stereopsis condition, in terms of either percentage errors (see Figure 3.3.5) or thresholds (see Figure 3.3.4) and no group-by-texture interaction was found. This suggests that the ability to maintain

vergence angle across saccades is, in general, satisfactory in adults with dyslexia, and that this is not likely to be a causal factor in their reading difficulties. However, a group-by-time period interaction for the simultaneous stereopsis task was found (see Figure 3.3.6) suggesting that the visual systems of those with dyslexia may tire more easily [also see the main discussion of both experiments below (section 3.6) and the further discussion (section 3.7)].

Between group differences in variance on the simultaneous task, and groups differences in stereoacuity on the Randot stereotest are easily reconciled with results of previous researchers. Buzzelli (1991) found the same large variance amongst the dyslexic group using a Randot stereopsis test. Stein, Riddell and Fowler (1987) report that children with dyslexia *with poor binocular fixation* have significantly reduced stereoacuity than normals on the Randot test. Felmingham and Jakobson (1995) report higher (but again non-significant) stereoacuities in participants with dyslexia on both Frisby and Randot stereotests. Bedwell et al (1990) found significantly poorer stereopsis in their group of poor readers. Kulp and Schmidt (1996b) found that stereoacuity below 100 seconds of arc was predictive of reading ability in children of average intelligence. Simons and Grisham (1987) conclude that the evidence is too mixed to draw any conclusions on whether reduced stereopsis is or is not associated with poor reading ability. Evans (1998) reaches the same conclusion and suggests that it may depend on the exact type of stereotest.

However, the large variance observed in the present experiment and others could be indicative of possible subtypes of dyslexia. Boder (1973) for example, has argued that distinct subtypes of dyslexia exist, with a small percentage of people with dyslexia (around 10 percent) having visual but not phonetic analysis deficits (see e.g. Watson and Willows, 1993, for a review)⁶. An alternative explanation is that the large variance elicited by the group with dyslexia may indicate motivational differences. For example, in the case of the simultaneous stereo condition at least, the two participants with dyslexia who performed most successfully in terms of disparity threshold (JA and SB), were also those who scored least errors on the

⁶This is not to say, however, that Boder's "dyseidetic" dyslexics (who have deficits in whole word reading and rely entirely on phonetic analysis) have lower level visual deficits such as those examined here. In fact, there is some evidence to suggest that low level visual processing deficits in the *transient* visual system are common in "dysphonetic" dyslexics (deficits in phonetic analysis) but not dyseidetic dyslexics (e.g. Borsting, Ridder, Dudeck, Kelley, Matsui and Motoyama, 1996). There is also evidence to suggest a link between vergence function and transient system deficits in dyslexia; Evans, Drasdo and Richards (1996) found a significant correlation between flicker threshold and binocular instability.

nonsense word passage originally used to assess their reading difficulties, and furthermore those who took the longest time to read it (excluding JL, the obvious outlier in Figure 3.3.3, who was excluded from some statistical analyses and who read both slowly and inaccurately on the nonsense word passage). Although abilities in reading this passage obviously differ in terms of both accuracy and speed anyway, it is tentatively suggested that JA and SB may be likely to choose accuracy over speed if a trade-off is required, and this determination to do things well is reflected in their good disparity thresholds⁷. Unfortunately in this experiment, no time limit was set (although this did not appear to be a problem in most cases), and since no times were noted this explanation cannot be investigated.

The main group and group by time interaction effects elicited if simultaneous stereopsis results are divided into pre- and post-experiment measures (p<0.05 and p<0.02 respectively: see Figure 3.3.6) can also be reconciled with previous research. It is widely accepted clinically that binocular dysfunction sometimes only produces symptoms when the person is tired, or after prolonged visual tasks (e.g. Ehrlich, 1987; Yekta, Jenkins and Pickwell, 1987; or see Watten, 1994 for an overview). In addition, the Dyslexia Automatisation Deficit Hypothesis (Nicolson and Fawcett, 1990) suggests that dyslexia is an inability to become completely fluent in all cognitive and motor skills, and that equal performance to controls is sometimes achieved only through 'Conscious Compensation' (trying harder or using strategies to minimise the deficit). The concept of 'Conscious Compensation' also has the implication that people with dyslexia will become more quickly and easily tired because their performance is not automatic. This concept is therefore also able to explain the larger variance in the dyslexic group: if extra effort were needed for them to perform well, it is possible that only some of them considered it worthwhile. It was on the basis of this argument that in the second experiment a dual task was added to investigate the automaticity of the eye movements.

The second experiment therefore used a dual task, restricted time, paradigm in order to examine the automaticity of eye movements. The possibility that JA and SB were putting a great deal of effort into the task, whereas the others in the group

⁷However, when the absolute percentage error values are examined, SB *does not* appear to be doing as well on the simultaneous condition. In fact the raw data reveal that SB's values on this condition are higher than they should be, indicating that the moveable target is being positioned further back than it should be. This pattern also appears in the results of SB's other conditions. It is possible that SB was performing the task by using porthole-to-target distances as opposed to eye to target distance. A good disparity threshold with a high error value, may suggest the use of monocular cues; a consistent means of doing the task, resulting in a low standard deviation between values, but the means used being unreliable in terms of accuracy.

with dyslexia and the control group were not, would go some way to explain the large difference in variability between the groups. A dual task, would reduce the possibility of participants using 'conscious compensation' (and is a standard method of investigating the automaticity of a task: see below). Eye movements do need to be automatic in order for the reader to be able to concentrate on the text (Griffin, 1982 cited in Kulp and Schmidt). Nicolson and Fawcett's Dyslexia Automatisation Deficit (1990) and Cerebellar Deficit (Nicolson, Fawcett and Dean, 1995) Hypotheses, as outlined in the introduction, might predict a dual task performance decrement in dyslexic groups.

It was also decided to count how many glances to and from each target are required in order to perform the task. It could be that the majority of differences between groups' eye movements can be explained by differences in timing and aspects related to time. This would be in accordance with findings of the flicker contrast sensitivity differences (e.g. Lovegrove, 1994), the slower vergence facility (Buzzelli, 1991) and perhaps even the increased number of saccadic regressions (e.g. Pavlidis, 1981). The saccadic regressions observed by Pavlidis in children with dyslexia could have occurred as a result of poor timing and the children 'getting ahead of themselves'. They may have moved onto the next saccade before making full use of the first one. Alternatively, the variation in size of the saccades could be due to timing of the eye muscle activation causing under- or over-shooting of the movement. Dodgen and Pavlidis (see 1989) found inferior temporal but not spatial accuracy in eye movements in children with dyslexia when they investigated similar issues.

3.4 Experiment 2

Data were collected for this experiment in collaboration with Emma Reynolds as part of her final year project.

Introduction

In Experiment 1, no differences were found between groups, although it was noted that there appeared to be more variability within the group with dyslexia. This observation, coupled with informal observations that the group with dyslexia appeared to be putting in more effort, and the apparent fatigue effect found for the dyslexic group on simultaneous conditions, led to the idea of Experiment 2; comparing groups on a sequential stereopsis task both with and without a secondary task.

Experiment 2 used the same three textures as the first, under the same sequential conditions, but on half the trials an additional motor task was added in order to create a dual task condition and investigate the automaticity of performance. Although in Experiment 1, there were no obvious differences between the groups on the different textures, it was possible that the dual task would elicit these differences. Furthermore, use of the same textures allowed comparison between the two experiments.

As noted earlier, the addition of a second task is a standard method of investigating the automaticity of a skill; if a skill is being carried out automatically, then attentional capacity will remain in order to carry it and the additional task out simultaneously. Nicolson and Fawcett (1990) found from a series of balance experiments on children with dyslexia, that although under optimal (single task) conditions, children with dyslexia can balance as well as controls, under dual task conditions (counting backwards and balancing at the same time) they cannot. This finding was replicated with a number of different secondary tasks and suggested that the act of balancing had not been automatised in children with dyslexia. Adams (1990), concluded that, "...the most critical factor beneath fluent word reading is the ability to recognise letters, spelling patterns and whole words effortlessly, automatically and visually ... ". This experiment therefore seeks to examine the automaticity of the ability to perform sequential stereopsis in adults with dyslexia and their controls. It also looks at and compares the number of glances to and from each target that are required by each subject to do the task as part of this. This part of the investigation was mainly exploratory, with no strong hypotheses. However, if the adults with dyslexia are trying harder, then one might expect to see a greater number of eye movements being made either in order to increase accuracy or because attention cannot be fully engaged (Fischer et al, 1993), or less eye movements because eye movements cannot be as easily disengaged (Fischer et al, 1993). It is plausible that adults with dyslexia might make a greater number of saccades on each judgement of equidistance in order to be able to discount those saccades where vergence was not accurately controlled.

Method

Participants

Participants were different individuals from Experiment 1 but complied to the same criteria (total of 20 participants).

Group with dyslexia: 5 males, 5 females; mean age = 26.8 years (20 years to 41 years), mean score on the Randot around 25 seconds of arc, ranging from 20 to 30 seconds of arc.

Control group: 7 males, 3 females; mean age = 22.2 years (19 years to 40 years), mean score on the Randot around 29 seconds of arc, ranging from 20 to 40 seconds of arc.

A Mann-Whitney U test showed that differences between groups in terms of level of stereoacuity on the Randot test were not significantly different (unlike in Experiment 1, where differences in stereoacuity were significantly different)⁸.

Apparatus

For this experiment the same basic apparatus was used as in Experiment 1, except that the shutter was controlled by a computer to open for 20 seconds when triggered by the experimenter. The computer could also produce a short regular tone every 0.9 seconds for the time that the shutter was open. This facility was used for a dual task condition where participants had to trace around a figure of eight template with their left hand, once for every sound of the tone. The same target textures were used in Experiment 2 but an additional dependent variable was recorded: the number of eye movements made during equi-distance settings, counted off-line from a video recording of the participant's eyes. In order to

⁸Note that pooled Randot stereoacuity over the two experiments showed no significant differences between groups on the Mann-Whitney U test.

attempt to keep the length of the experiment reasonable, the simultaneous stereopsis condition was not used in this experiment.

Procedure

In Experiment 2, a set of pre-specified starting distances were used for the right moveable target. This was a refinement to Experiment 1 to ensure a balanced set of starting distances. For the left fixed target position of 570mm these were 510mm, 540mm, 600mm and 630mm. For the left fixed target position of 620mm they were 560mm, 590mm, 650mm and 680mm. For both the single and dual task conditions, two blocks of six settings in all were required for each texture, with one block at each of the two fixed positions of the left target.

The stimuli were presented in the same pseudo-random order for each participant, chosen using the same constraints as in Experiment 1. This had the advantage that both participants with dyslexia and their controls were subject to the same order effects, if any. The disadvantage was that if there were any order effects then inferences would be limited to the particular order chosen. As it turned out, this issue proved to be of no practical importance because analyses were conducted that showed that there were no significant order effects. In addition to the order constraints used in Experiment 1, dual and single task conditions were alternated in an attempt to prevent the dual task becoming over-practised and automatised.

Participants were asked to follow the same procedure used in Experiment 1, except they were informed that they had 20 seconds on each trial to complete the equidistance setting task before the shutter closed. This was established as an adequate time to complete the task during pilot runs. Eye movements were also videorecorded with a standard video camera in this experiment and counted off-line⁹. Nothing too elaborate was attempted with these recordings, each saccade to either the left or the right was simply counted as one movement. The necessary positioning of the video camera would not have allowed anything more ambitious and the process was merely exploratory.

Design

In Experiment 2, the same basic design was used except for the addition of the dual task and the omission of the simultaneous stereopsis task. Repeated variables were texture (unfiltered, medium or high), task (single or dual) and target position (570mm or 620mm). Dependent variables in this experiment were again the

⁹I acknowledge the hard work of Emma Reynolds in performing this tedious task.

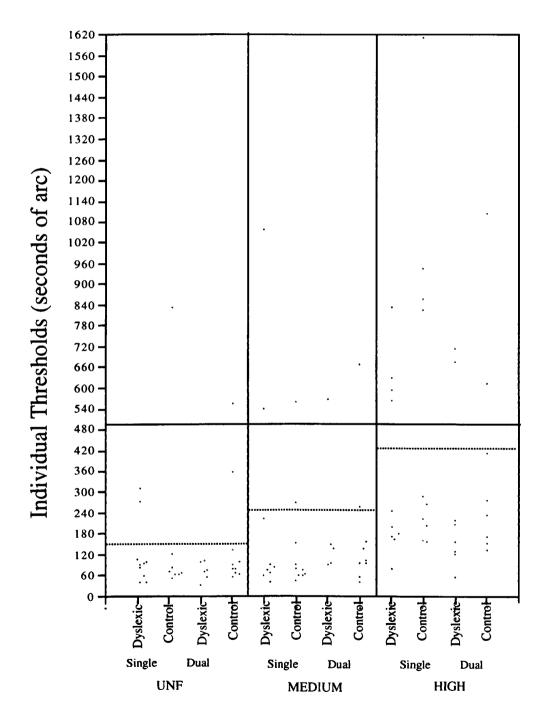
settings made [converted to disparity thresholds and error values (as in Experiment 1)] and the number of eye movements made.

Results

A plot of the individual participants' disparity thresholds for Experiment 2 is shown in Figure 3.4.1. Thresholds were generally slightly higher than in Experiment 1, but this difference was not significant (F<1). In line with the criterion used in Experiment 1, analyses were also performed with participants excluded if their thresholds exceeded 500 seconds of arc on any condition. However, if on either the single or the dual task condition their threshold was below this limit, it was concluded that they could do the task given optimal conditions (or possibly practice) and they were not excluded. This procedure led to the exclusion of 3 control and 3 participants with dyslexia, but again did not affect any of the main conclusions drawn from the experiment. It is interesting to note that the exclusion of many of the participants on the basis of their performance on the high SF condition also improved the level of performance (as determined by the worst thresholds) on the unfiltered and medium SF conditions. The dashed line on Figure 3.4.1 illustrates the point of the worst threshold after exclusions. All results are reported without exclusions as they had little or no bearing on the main results.

Mean thresholds and absolute percentage errors are shown in Figures 3.4.2 and 3.4.3. The dual task condition did not decrease sensitivity appreciably or significantly (F<1). Again, performance in both groups was good, with participants with dyslexia performing as well as controls (F<1) and there were no significant group interactions. Texture had highly significant effects in all analyses (disparity thresholds: $F_{2,36}=11.61$, p<0.0001; absolute percentage errors: $F_{2,36}=7.46$, p<0.005).

Figure 3.4.1. Individual participants' disparity thresholds in Experiment 2. The solid black line indicates the threshold value which considered a reasonable level of performance; participants with threshold values above this line on any texture were excluded from some analyses. The dotted lines indicate the effect of the exclusions on the highest individual threshold levels on each of the textures.



Texture

The problem of data deviating from a normal distribution was more difficult to deal with in Experiment 2, because the RANOVA analysis program used in Experiment 1 has thus far only been developed to cope with two factor analyses. Analysis of single and dual data was therefore carried out separately for this experiment using 10000 randomisations as before. Similar to the ANOVA programs, the RANOVA produced highly significant texture effects in all analyses and there were no significant group effects.

Figure 3.4.2. Mean disparity thresholds for groups in Experiment 2. Standard error bars shown.

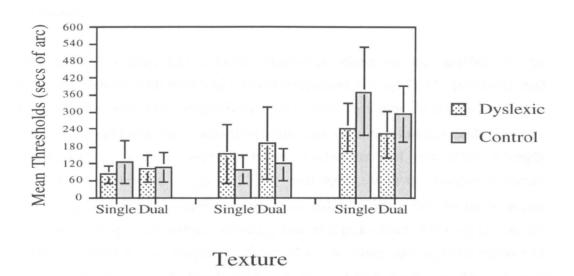


Figure 3.4.3. Mean absolute percentage errors for groups in Experiment 2. Standard error bars shown.

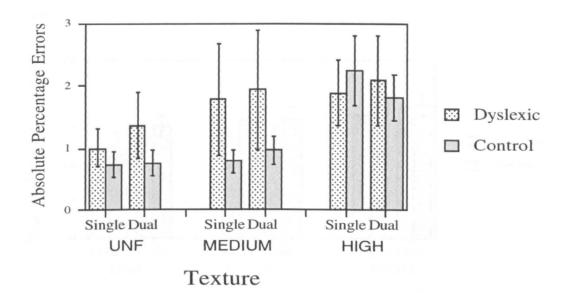
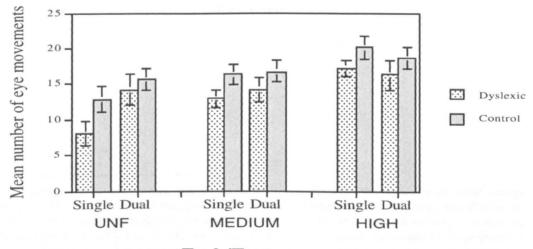


Figure 3.4.4 shows the results of the eye movement counts in Experiment 2. The control group made an average of 3 more eye movements than the dyslexic group per setting, but this difference was not large enough to reach significance ($F_{1,18}$ =1.91). However, the mean number of eye movements made by each group were significantly different after exclusion of participants ($F_{1,12}$ =6.00, p<0.05) with an average of 5 more movements being made by the control group (see Figure 3.4.4). Overall, the means before and after exclusion suggested that those participants who had been excluded were making less eye movements overall, particularly amongst the control participants (possibly indicating lack of motivation).

As expected, texture had a highly significant effect on the number of eye movements made both with and without exclusions ($F_{2,36}$ =41.15, p<0.0001 and $F_{2,24}$ =42.39, p<0.0001 respectively), with more eye movements in the higher frequency conditions. The main effect of the task condition (single/ dual) was also significant ($F_{1,18}$ =7.21, p<0.05) before exclusions and this effect strongly interacted with texture, ($F_{2,36}$ = 22.86, p<0.0001), with the unfiltered texture producing more eye movements in the dual task condition, but the medium and high spatial frequency textures showing less of a task effect. After exclusion, the main task effect was no longer significant ($F_{1,12}$ = 3.66), although the number of eye movements was still slightly higher in the dual task condition. The effect of task still interacted with texture ($F_{2,24}$ =16.15, p<0.0001).

Figure 3.4.4. Mean number of eye movements by group in Experiment 2. Standard error bars shown.

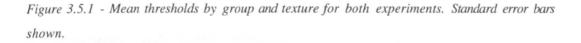


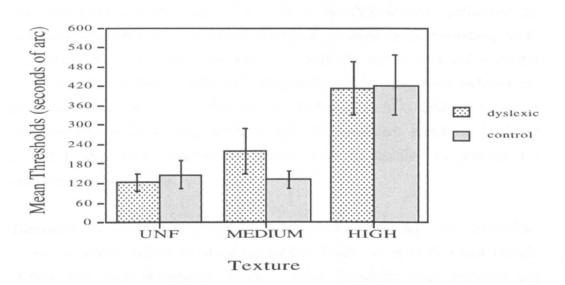
Task/Texture

3.5 Pooled Data

In order to reaffirm the findings of no significant differences between groups, single task data from the two experiments were pooled, yielding 35 participants altogether¹⁰. This was considered to be a valid action since there were no significant differences between the two experiments. Again, analyses were conducted both with and without exclusions, but since it made no difference to the results, they are reported without exclusions.

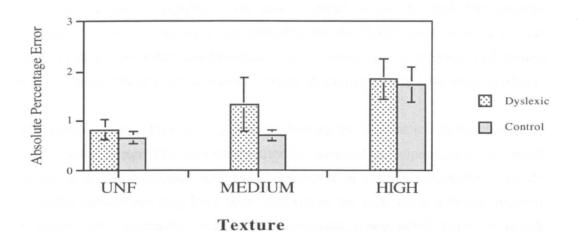
Figures 3.5.1 and 3.5.2 show group mean disparity thresholds and absolute percentage errors after pooling. Again, texture effects were highly significant ($F_{2,66} = 20.99$, p<0.0001 for thresholds; $F_{2,66} = 11.80$, p<0.0001 for absolute percentage errors). There were no significant differences between groups, and no significant interactions (all F values <1).





¹⁰Conditions for the single task conditions in both experiments were basically the same except that (a) a 20 second time limit was given in Experiment 2 whereas unlimited time was allowed in Experiment 1; (b) the moveable target was set at specific rather than random starting positions in Experiment 2; (c) only two blocks were used to generate the mean thresholds in Experiment 2 whereas four were used in Experiment 1; and (d) the same presentation order was used in each case in Experiment 2 but not Experiment 1. The participants' task was the same in each case.

Figure 3.5.2 - Absolute percentage errors by group and texture for both experiments. Standard error bars shown.



3.6 Experiment 2 and General Discussion

The main result was that adults with dyslexia and their controls performed the sequential stereopsis task at a similar level and, in most cases, reasonably well, even under dual task conditions. However, both the use of students/ graduates together with a failure to subtype the dyslexic group, may have reduced the likelihood of finding group differences. Furthermore, it is difficult firstly to ascertain whether or not the dual task exerted a significant attentional load and second to distinguish motivational effects from an inability to perform the sequential stereopsis task.

Thresholds in the sequential stereopsis task, both before and after exclusion, showed a similar pattern to those reported in Frisby et al (1997) and Enright (1996), with high SF textures eliciting higher thresholds than unfiltered and medium SF textures. Thresholds after exclusion were also of a similar level to those in Frisby et al for experienced observers. It is interesting to note that in Experiment 1 the simultaneous stereopsis condition elicited very similar thresholds to both the unfiltered and medium SF sequential stereopsis conditions (see Figures 3.3.4 and 3.3.5). This is further evidence that people do not need to use simultaneous relative disparities to compare distances accurately (cf. Enright, 1996), since the extra information provided by the relative disparities in the simultaneous stereopsis condition elicited to be the extra information provided by the relative disparities in the simultaneous stereopsis condition elicited no better thresholds.

Surprising, however, was the number of exclusions that were made in the present experiments, particularly in Experiment 2. The exclusion of six participants in Experiment 2 may either be taken as an indication that 20 seconds was not actually long enough to do the task for many people, or that, as not previously suspected, many people are not capable of the task (particularly on the high SF texture). Detailed optometric data were not available on the participants so it is unclear whether they manifested any binocular vision anomalies on conventional clinical tests (although the Randot stereotest showed all participants to have stereo vision).

The dashed lines on Figure 3.3.3 (which illustrate the level at which the thresholds could have subsequently been drawn after exclusion of participants) are very much in line with the thresholds reported in Frisby et al (1997), suggesting that the excluded participants may have been performing the task via a different method. However, when originally setting up the apparatus, it was noted that some people experienced 'stereo capture' (where the texture appears to lie in the plane of the ports) with circular viewing ports similar to those used by Enright. This happened particularly on the high SF texture. Rectangular ports appeared to diminish this problem with the original observers used; see Frisby et al (1997) for a review and possible explanation. It is possible therefore, that this problem has not been completely removed for all observers and that it had some bearing on results reported here. Nevertheless, in the case of these experiments, the number of dyslexic and control participants that were excluded were approximately equal and performance of groups was similar whether or not exclusions were made.

Thresholds may have been expected to be *slightly* higher in this experiment than in Frisby et al (1997) because mean thresholds were generated from fewer blocks of settings in the current experiment, thereby allowing both less practice over the experiment and a larger influence of less accurate settings. Motivation and experimental expertise of individual participants could also have increased thresholds, especially since this experiment, unlike Frisby et al's, and many vision experiments, did not use anyone familiar with the techniques of visual experiments.

Our failure to subtype dyslexia may have reduced the probability of identifying a vergence control difficulty. The issue of subtypes has already been briefly outlined in the discussion of section 3.3 (Experiment 1). Not all children with dyslexia have been found to have any weakness in vergence control. However, Stein, Riddell and Fowler (1988) found that 64% of their sample were unable to make proper

vergence movements to small targets, and in a preliminary small sample study, Hung (1989) found that two (out of two) adults with dyslexia showed reduced vergence velocities in comparison to controls. Furthermore, the exact categories of dyslexia subtypes are far from decided upon, and the area remains controversial. However, it should be noted that if only a small subgroup of people with dyslexia do have a visual deficit of the type investigated here, then this study is unlikely to have detected it due to the relatively small numbers of participants used.

Since dyslexia is a developmental disorder, it is also possible that whereas university students and graduates can perform this task, children with dyslexia do not show this degree of control (possibly hindering their reading development), but control is gradually gained with age. This could be something worthy of further investigation. Moreover, all the adults with dyslexia studied here are not only well compensated, but also well practised in their reading ability. Fischer et al (1993) found differences on saccadic tasks only in a group of adults with severe dyslexic difficulties. It may well be that practice in reading develops in tandem with the ability to perform sequential stereopsis (or its real world equivalent). Further investigation might therefore include adults with poor literacy skills. In addition, even for the adults studied here, it could be that adults with dyslexia can exhibit good vergence control across saccades when giving their full attention to making equi-distance settings, but that this is a fragile capacity that is lost when the extra demands of reading are involved. This is a further question for future research.

Finally, as discussed earlier, it is widely accepted clinically that binocular dysfunction sometimes only produces symptoms when the person is tired or after prolonged visual tasks (e.g. Ehrlich, 1987; Yekta, Jenkins and Pickwell, 1987; or see Watten, 1994 for an overview). Wilkins (see e.g. 1995) describes how rapid pulsation from conventional fluorescent lighting as well as striped patterns (analogous to text) can also cause visual stress. Scotopic sensitivity syndrome is a sensitivity to "light source, glare, luminance, wavelength, and black/white contrast" from which many people with dyslexia are said to suffer. Meares (1980) and Irlen (see e.g. Helverston, 1990) first reported the benefits that people with 'scotopic sensitivity syndrome' experience from using coloured filters. Wilkins (1995) explains that coloured filters can reduce the impact of fluorescent lighting on the visual system and may also have other benefits. One could therefore infer that many people with dyslexia suffer from excessive stress on the visual system during reading and reading-like tasks and only during these tasks would visual anomalies be detectable. It is therefore possible that the length and nature of this

experiment, or whatever the participants were doing before they arrived to do the experiment did not result on such stress upon the visual system. Results from Experiment 1, however, on the simultaneous condition might suggest that the length of the experiment *was* long enough to bring out some difficulties.

The effects of the dual task used in Experiment 2, in an attempt to prevent any conscious compensation by the dyslexic group, are difficult to ascertain. It was hoped that the figure-of-eight tracing task would put a substantial 'attentional load' on the equi-distance setting task. However, whether tracing did in fact impose a sufficiently great attentional demand is difficult to judge. Performance on the dual task was neither monitored nor measured but this task did have an effect, that interacted with texture, on the number of eye movements made. However, the dual task did not affect thresholds or error values. On the unfiltered SF condition, more eve movements were made in the dual task condition, whereas for the other conditions it appeared to make little difference. This might be taken as indication that it did make some demands on the observers. However, it was noted during the eye movement counts that many participants tended to move their eyes in time with the tones, so it is possible that pacing from the tone frequency led to more eye movements. On the other hand, it is not obvious why pacing should have created an effect only for the unfiltered texture. Obviously, it would be easy to choose other more demanding dual tasks. Our intention was to find one that would leave controls able to do the sequential task reasonably well. The tracing task satisfied that criterion but it may nevertheless have been too easy to bring out difficulties for the adults with dyslexia. Dual tasks that have elicited deficits in performance in children with dyslexia in the past have included counting backwards whilst balancing on a beam (Nicolson and Fawcett, 1990). Counting backwards was not a suitable second task to use in this experiment because a bite bar was being used. Furthermore, a motor task was chosen in order to 'compete for attention' with the motor component of the eye movements, since researchers do not yet agree on whether there is 'modularity of resources' or some 'central attentional capacity' (see e.g. Eysenck and Keane, 1990, for a brief review).

After exclusion of the participants that were presumed not to be carrying out the sequential stereopsis task as intended, a significant group difference emerged on the number of eye movements made to each target, with more movements made by the control group. This effect is evidently very fragile, and it would be foolish to infer too much from it, particularly given the lack of difference between groups on accuracy. However, it is interesting to note that Fischer, Biscaldi and Otto (1993)

found more anticipatory saccades in an group of adults with dyslexia with more severe difficulties and longer overall saccadic reaction times to a target in a group with less severe difficulties. This finding was interpreted in terms of attentional differences, with the less severe group showing difficulty disengaging attention. Our group of participants would probably be considered to be less severe (at least on the basis of the fact that they were all University students and graduates: they all still showed deficits on the tests used for their diagnosis and very often large discrepancies in performance), so the finding of fewer eye movements is, in this sense, consistent with Fischer et al's findings. Nevertheless, even if the difficulty in disengaging attention argument holds true, the difference between groups did not adversely affect the accuracy of our group with dyslexia. The fact that the finding of more eye movements in the control group appears only after the exclusion of participants presumed not to be performing the task as intended, could suggest that these control participants were not actually trying very hard (although this was not the impression given). If this is the case, and if lack of effort is reflected by fewer eye movements, then the original question of whether adults with dyslexia need to try harder in order to achieve an equal level of performance is not supported.

The main result therefore is that this small sample of adults with dyslexia can perform the sequential stereopsis task as well as controls. If it is accepted that this task does depend upon good control of vergence across saccades, then the inference can be drawn that poor vergence control across saccades is not likely to be a factor in their current reading difficulties. However, this argument would need to be validated with a larger population and cannot necessarily be extended to children.

3.7 Does Sequential Stereopsis Depend on Good Control of Vergence Across Saccades? A further examination of Enright's Theory

The question of the method by which stereopsis is improved by allowing free fixation has only recently been 'resurrected' by Enright. Enright (1991) proposed that sequential stereopsis operates as described in the introduction and shown in Figure 3.2.1; absolute disparities are compared at the end of each saccade/ at each fixation and that this requires the capacity to make iso-vergent saccades. However, the original ideas (e.g. Wright, 1951; Ogle, 1956) were very different from Enright's. Furthermore, more recent evidence raises questions about Enright's theory.

Wright (1951), cited in Enright, originally argued that improved distance discrimination with eye movements arose from information about convergence of the eyes. Ogle (e.g. 1956), on the other hand, claimed that stereopsis could take place in the midst of a saccade. Enright's (1991) data suggested that explanations offered by Wright and Ogle were not adequate. Convergence of the eyes was unlikely to be able to provide the necessary information in the time available as small vergence eye movements are said to proceed very slowly (e.g. Carpenter, 1988, cited in Enright, 1991) whereas Enright forced *rapid* alternation of fixations. Enright also claimed that his apparatus prevented the possibility of stereopsis occurring in the midst of a saccade. From this, and the evidence that observers produce reasonable stereoacuity thresholds even when a dark interval occurs between stimuli, arose Enright's theory of stereopsis via iso-vergent saccades.

However, recent evidence (Frisby, Taroyan, Buckley and Porrill, unpublished) suggests that sequential stereopsis may not operate quite how Enright suggested. Enright's theory presupposes that, in order for stereoacuity to be high, targets to be adjusted to an *perpendicularly equidistant* position¹¹ must lie symmetrically with respect to the cyclopean (central) eye, on the same iso-vergence circle. Figure 3.2.2 illustrates an iso-vergence circle; showing how all different positions of possible fixation on the circle elicit the same vergence angle between the eyes. Figure 3.7.1 illustrates the situation in Enright (1996), Frisby et al (1997) and this experiment, with targets positioned symmetrically about the cyclopean eye. However, Frisby et al found that people are able to perform sequential stereopsis accurately even when the targets do not lay on an iso-vergence circle (e.g. see Figure 3.7.2).

Frisby et al positioned each observer's head at different gaze-angles relative to the targets, thereby ensuring that the targets no longer lay on an iso-vergence circle when equidistant. High stereoacuity was still found. However, a small but significant effect in the direction of the expected results from Enright's theory was found in both the unpractised, naive observers and the experienced observers in their first session only. This effect was not replicated in later sessions. The significance of this is therefore unclear. Frisby et al dismiss it as a small effect that was not replicated and not as large as Enright's theory would have predicted. Data

¹¹as opposed to cyclopean equidistance (equal distance from the cyclopean eye), a perpendicularly equidistant position is equidistant with respect to the body and the rest of the apparatus, so that the 2 targets would line up if viewed from above, regardless of head position

were equivocal on whether observers used cyclopean or perpendicular equidistance as the basis of their settings. Results are concluded to indicate that iso-vergent saccades are not necessary to be able to perform sequential stereopsis.

However, the concept of an iso-vergence circle could be considered to be somewhat flawed in this situation, since the targets are reasonably large areas rather than single points in space. It seems plausible that at some gaze angles at least, there may be an isovergence circle which can encompass at least one visible point of each target.

Figure 3.7.1. How isovergent saccades can be used to achieve equidistance of targets if they lie of an iso-vergence circle symmetrically with respect to the cyclopean eye.

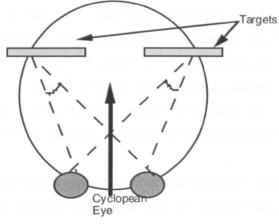
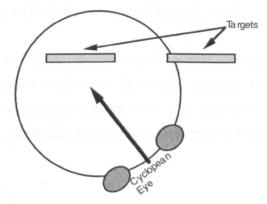


Figure 3.7.2. If gaze angle is altered, targets no longer lie on an iso-vergence circle when equidistant. Thus, iso-vergent saccades can not be used to achieve equidistance of targets.



Frisby et al describe Enright's suggestion that, "perhaps observers create geometrically non-isovergent positions even when the eye movement control system has issued commands for isovergence saccades and the visual system undertakes computations on disparity data as though true isovergence had been achieved". It is beyond the scope of this chapter to discuss possible mechanisms of sequential stereopsis further. However, it seems appropriate to conclude that despite such discussions and regardless of the exact mechanism, it is likely that fine oculomotor control is required.

3.8 Summary and Conclusions

No differences between a small sample of well compensated adults with dyslexia and control adults were found on this sequential stereopsis task. The exclusion of many participants on the basis that they could not do the task could indicate dyslexia subtypes, but the surprising result was that an equal number of control participants could not do the task either. Since optometric data were not available on the participants, it is unclear why this occurred, although the possibility of stereo capture was discussed. The sample size used in this experiment was certainly not large enough to infer or expect to find any subtypes.

The addition of a dual task appeared to have little effect on either group in terms of stereoacuity, but it is possible that the second task was not taxing enough. It is also possible that no differences between groups were found because participants were not sufficiently tired. If we had put stress on their visual system before the experiment, differences may have emerged as binocular anomalies often only become apparent under such conditions, although the group by time period interaction found on the simultaneous condition in Experiment 1 could be taken as evidence that the group with dyslexia were sufficiently tired.

It is also possible that in children and less well compensated adults with dyslexia, differences would be found on this task. However, as yet it is also not completely clear exactly which visual mechanisms this task involves. Nevertheless, by whichever mechanism sequential stereopsis is possible, it is likely to involve fine binocular control.

Chapter 4

Attention Shifting Deficits in Dyslexia?

An initial investigation of reported attentional deficits in dyslexia

Summary:

Chapter 3 concluded, with certain provisos, that visual differences in sequential stereopsis were not likely to be a causal factor in dyslexia, at least in those participants tested. The method used in Chapter 3 was novel, useful and easily applicable to reading, as well as producing an interesting result in terms of the fatigue effect. However, there was a sense that, despite the fact that the method did confer so many advantages over previous methods used to investigate the issue, it was not really reporting anything new (rather adding to a collection of other studies). Visual differences in dyslexia have been previously investigated in numerous different ways. In contrast to this, attentional difficulty has often been linked to dyslexia (see section 1.7), but relatively few studies have directly investigated the nature of the supposed attentional disorder. There is also considerable evidence that, in addition to phonological problems, children with dyslexia may have difficulties with rapid processing (see section 1.4). One intriguing possibility is that these rapid processing problems are associated with attentional difficulties and in particular impaired ability to switch attention rapidly. Nobody has investigated this possibility before. A rapid attention switching deficit would also offer an alternative explanation for dual task deficits so central to the DAD (see section 1.5); with dyslexic children having difficulty switching attention rapidly between the two tasks, rather than a difficulty automatising one of them. Furthermore, since Akshoomoff and Courchesne (1994) found a rapid attention switching deficit in cerebellar patients, such a deficit might be expected in dyslexic children from the CDH (see section 1.6). The hypothesis of a rapid attention shifting deficit in dyslexia was tested using the paradigm developed by Akshoomoff and Courchesne (1994). Fourteen normally achieving adolescents and adolescents with dyslexia participated. As predicted, 14

participants with dyslexia were significantly less accurate than the controls, specifically in a condition where they had to switch attention between two target types. However, contrary to prediction, a *rapid* attention shifting deficit was not found; the accuracy of participants with dyslexia was relatively good immediately after a target switch, but relatively poor several trials subsequently. Results are interpreted in terms of resource limitations together with difficulty using a verbal labelling strategy.

4.1 Attention

The concept of "attention" has often been considered more of a layman's term than a term of use in scientific research. William James (1890) stated that (from Eysenck and Keane, 1990, p97):

> "Everyone knows what attention is. It is the taking possession of the mind, in clear and vivid form, of one out of what seem several simultaneously possible objects or trains of thought. Focalisation, concentration, of consciousness are of its essence. It implies withdrawal from some things in order to deal effectively with others."

Attention has often been considered to be synonymous with concentration or mental set and it is thought to play a role in learning and memory (see section 1.7). Eysenck and Keane (1990) argue that, "there is an obvious danger that a concept that is used to explain everything will turn out to explain nothing", but that attention is most commonly used to refer to selectivity of processing, as described by James (1890). However, Posner and Boies (1971) suggested that the study of human attention could be divided into three components; alertness, selectivity and processing capacity. However, these components are not necessarily mutually exclusive.

Being alert can be considered the most basic form of attention; the ability to maintain attention over a period of time, and potentially during long and boring tasks. In a vigilance task requiring participants to watch a clock pointer moving in steps (in case it made a double step), Mackworth (1950) showed how performance rapidly declined over time. Using a similar task, however, Jerison (1957; 1959) showed how performance was less likely to decline if participants had to watch

three such clocks, although performance was likely to be lower throughout. Similarly, Deese (1955) showed how performance in a vigilance task is better if the rate at which signals occur is higher (all tasks cited in Broadbent, 1971). This apparent paradox, where a task apparently of greater effort is less likely to cause fatigue effects, generated a great deal of theoretical research. The main four approaches to explaining the effect were inhibition theory (related to extinction of a response following non-reward in classical conditioning), expectancy theory (where response was considered more likely where signals had a higher probability of occurrence), activation theory (a physiological approach which postulated that the body requires constant stimulation in order to continue to work efficiently) and filter theory (suggesting that information is filtered for attention on the basis of novelty, which obviously decreases as time goes on). Broadbent (1971) provides a thorough review of all the theories and the evidence supporting them (pre-1971).

Perhaps related to the ability to maintain attention are attentional lapses, absentmindedness or action slips. Reason (1979) categorised such action slips into: storage failures (repeating an action twice due to having no recollection of having already done it e.g. brushing teeth twice in a row); test failures (not monitoring a planned sequence sufficiently e.g. going to the post a letter but instead going to the shop); subroutine failures (inserting, omitting or re-ordering component stages of an action sequence e.g. sitting down to write and putting hand up to remove glasses from face when they are not there (Reason, 1979); discrimination failures (failures to discriminate between objects e.g. pouring orange juice instead of milk into coffee) and programme assembly failures (inappropriate combinations of actions e.g. throwing banana away and keeping peel). Reason (1979) suggested that such action slips tend to occur during highly practised tasks and may result from inappropriate use of (or switching between) automatic and controlled processing. Thus, automatised behaviour is easily forgotten and inflexible because little 'attention' is paid to it, although controlled processing can result in less fluid and less skilled movement precisely because it is not automatised.

Selectivity is Posner's second component of attention. It is possible to divide this into further components; the ability to focus, divide and/or shift attention i.e. attention allocation. Focusing attention involves concentration on only the relevant stream of information and screening out distracting information. It is possible to be alert, but unable to focus attention on just one thing, instead being distracted by other stimuli. How we focus our attention on particular stimuli, and in particular at

what point distracting/ irrelevant stimuli are rejected, has been a matter of some debate for many years (see e.g. Eysenck and Keane, 1990 for a brief introduction), particularly since information must have to be processed, at least in part, in order to be rejected. Shifting attention involves being able to stop oneself attending to one stream of information which is being focused on and re-orient attention to another. It is important to be able to do this voluntarily, rather than it being a result of distraction during a period of focused attention. Division of attention may be necessary if two tasks are to be performed simultaneously. The ability of people to do this is generally considered to be dependent on task similarity, task difficulty and automaticity; the more similar, more difficult or less automatic two tasks are, the more difficult it is to carry them out simultaneously. Evidence relating performing two tasks simultaneously has a bearing on arguments for central capacity vs. modularity of attention, i.e. whether each processing modality has its own attention module with limited capacity or whether attention has a limited capacity overall. However, it is not entirely clear whether simultaneous performance of tasks is what actually happens, or whether a rapid shifting of attention between tasks is what is really going on.

Processing capacity is Posner's third component of attention. We have limited attentional resources to divide and cannot attend to everything at once. How much we *can* attend to appears to be dependent on task similarity, difficulty and automaticity (as above). If a task is well practised and 'automatic', it will consume few (or no) attentional resources and so several automatic tasks can be performed simultaneously. Shiffrin and Schneider (1977) characterise automatic processing as not requiring attention, suffering no capacity limitations and difficulty to modify once learned. In contrast controlled processes are said to be of limited capacity, to require attention and to be flexible in changing circumstances. These concepts form the central tenet of the DAD.

Studies which have looked at the exact nature of the attentional disorder in children with dyslexia and ADHD have tended to focus on the distinction between selective and sustained attention and also on the re-orientation of attention.

4.2 Selective vs. Sustained Attention in ADHD and Dyslexia

Several experiments have investigated visual attention systems, selective attention, and sustained attention abilities in children with good and poor reading skills, children underachieving in either reading or arithmetic, and in children with ADD or ADHD. As already mentioned in section 3.1, in comparison with their controls,

teenagers with dyslexia make an increased number of express saccades to stimuli during trials where the fixation point disappears before the stimulus (gap trials), and a decreased number during overlap trials (Fischer and Weber, 1990). Fischer and Weber (1990) postulated that this reflects a deficit either in the visual attention system (or its control over the oculomotor system); possibly in engaging and disengaging attention. Various potential deficits in the attentional system are suggested, such as it being disengaged the majority of the time (so that saccades are not readily suppressed), difficulty in switching from the engaged to the disengaged state (and vice versa, so that a sequence of eye movements such as those required in reading would be difficult and erratic), or problems switching to the disengaged state after fixating a word (preventing the required saccade to move to the next word).

On a visual task hypothesised to elicit differences in selective and sustained attention, Dykman, Ackerman and Oglesby (1979) found many similarities between boys with dyslexia and boys with ADHD, in comparison with boys of normal achievement. Boys with ADHD and boys with dyslexia had more 'lapses' in their performance than their controls after they had already discovered the correct method for reward. However, Schacher, Logan, Wachsmuth and Chajczyk (1988) manipulated time on task in boys with ADHD to test the hypothesis of a sustained attention deficit. They found no differences between boys with ADHD and various control groups including normal achieving boys and boys with reading disability. Previous research (e.g. Cohen and Douglas, 1972; Sykes, Douglas and Morgenstern, 1973) has reported a sustained attention deficit in children with ADHD in comparison with normally achieving (and behaving) controls, although results have sometimes been contradictory (e.g. Michael, Klorman, Salzman, Borgstedt and Dainer, 1981 cited in Schacher et al 1988). Schachar et al (1988) suggest that inconsistencies in some instances may be due to the failure to control for other disorders (e.g. conduct or learning disorders) associated with hyperactivity.

Other research has suggested a *selective* attention deficit in children with dyslexia. Milberg, Whitman and Galpin (1981) investigated the hypothesis of atypical cortical lateralisation of language processes in children with dyslexia. They found that poor readers made a greater number of total errors and channel confusion errors in a dichotic listening task (where numbers from one ear only had to be reported). Similar results in terms of channel confusion errors were found in a listening task where pairs of numbers were separated spectrally (male or female voice). This evidence cast doubt on the atypical cortical lateralisation hypothesis, favouring a more general deficit in selective attention and/ or temporal ordering strategy. Milberg et al (1981) suggest that Broadbent's (1971) conceptualisations of 'filtering' and 'switching' can both offer explanations of the data in terms of a selective attention deficit; the poor reader either being unable to filter out irrelevant information, or randomly switching channels in an uncontrolled way.

Tarver, Hallahan, Kauffman and Ball (1976) concluded from their results that a developmental lag both in verbal rehearsal and selective attention existed for children with learning disabilities. They administered Hagen's Central-Incidental Task to two age groups of boys with learning disabilities (IQ>85 and >6 months below expected reading level) and their chronological age controls. In this task, children are presented a series of cards each with two items on: an animal and an object. They are asked to pay attention to only one set of pictures; in this case to remember the animals. Cards are placed in seven positions. Children are then given a probe card with a picture of an animal on, and asked to recall the positions of the animal on the seven overturned cards (central task). Following several probe trials in which the proportion of correct responses at each card position are noted (with the positions of the cards changing on each trial), participants are given a large card with pictures of all the animals on along with seven smaller object cards. This (incidental) task is to match the animals with the items that they appeared with in the central task. In the 8 year old children, results showed that the children with learning disability had a reduced primacy effect (i.e. did not recall more animals correctly in the first positions than the middle positions on the central task, suggesting poor verbal rehearsal strategy) and poorer performance overall. On the incidental task, however, there were no significant differences between groups (although the controls did perform slightly better). In addition, the central and incidental measures produced a negative (not significant) correlation in the controls, but a positive correlation (p<0.1) in the boys with learning disability. Together this evidence is taken as support for a selective attention deficit in younger children with learning disabilities. Since the central and incidental measures were negatively correlated in the control boys, Tarver et al posit that this indicates a trade-off between central and incidental recall in only that group (i.e. selective attention).

However, there are several problems with the experiment of Tarver et al (1976). First, regarding the correlational analyses, the negative correlation is not significant and the positive correlation in the learning disabled boys is only a trend. The positive correlation may only suggest that some boys in the learning disabled group were able to remember items better than others overall. A negative correlation of the size reported (r=-0.28, n.s.) seems to show really very little. Moreover, the higher mean recall of the control group in the incidental task does not lend support to the concept of a trade-off with the central task, although it is accepted that the control boys are likely to have superior memory skills overall. Stronger support for the concept of the boys with learning disability being unable to selectively attend would occur if they had performed better than their controls on the incidental task but worse on the central task.

A final, but possibly most important point regarding the Tarver et al study is that it is generally accepted that children with learning difficulties have difficulties with memory, and particularly sequencing, anyway. The evidence of a primacy effect in the controls, which is reduced in the learning disabled group, strengthens the argument that this task is tapping memory and rehearsal strategies and processes rather than selective attention alone. It should therefore be emphasised that the central task may have proved more difficult for the boys with learning disability for other reasons than those related to selective attention. The central task may well have been more difficult since it involved recall and order of position rather than just matching objects and animals together (recognition of a pair). There also is evidence to suggest that increasing the complexity of any task increases the likelihood of finding a deficit in children with dyslexia (e.g. Nicolson and Fawcett, 1994). These findings can therefore be equally well explained by a resources deficit in the learning disabled boys.

In spite of the main conclusions made by Tarver et al, they do comment that the two groups did not differ on a composite measure of proportion of correct central responses minus proportion of correct incidental responses which has been used as an indication of selective attention efficiency by researchers including themselves (e.g. Hallahan, Kauffman and Ball, 1974). A second experiment reported in Tarver et al (1976) suggested that verbal rehearsal aids selective attention analysed using this measure. The authors go on to suggest a developmental lag of selective attention in the boys with learning disabilities on the basis of additional analyses which include further (non-significant) correlations between the central and incidental tasks which go from positive to negative with age. In conclusion, although their reasoning for the deficit in verbal rehearsal playing a role in either selective attention, or at least the ability to recall selectively, may be correct, the evidence for a developmental lag of selective attention in boys learning disabilities

is fuzzy, not least because the evidence for a selective attention deficit at all does not appear completely sound.

Hallahan, Kauffman and Ball (1973) also put forward evidence for selective attention deficits and impulsivity in academic underachievers (90<IO<110) using Hagen's Central-Incidental task. Here, the data are more convincing; t tests conducted on mean performance in both central and incidental tasks elicited a group difference in the central task only, with underachievers showing poorer performance. Again, underachievers also show slightly poorer performance on the incidental task, but not significantly so. A significant negative correlation is found between central and incidental task performance for high achievers (r=-0.77, p<0.005 one tailed) and a positive correlation for low achievers (r=0.53, p<0.1 one tailed). This suggests a trade-off between central performance and incidental performance for high achievers, whereas low achievers score similarly on both. However, the lack of equivalence between the central and incidental tasks in this experiment, as in Tarver et al's, remains, as does the evidence for verbal rehearsal playing a large role. The central task involves *recall* of a position (undoubtedly requiring verbal rehearsal), the incidental task involves recognition of which two pictures make a pair. Hallahan et al also put forward some evidence for increased impulsivity in the low achieving group from results of Kagan's Matching Familiar Figures task¹, with a significantly increased number of errors in low achievers and a trend towards them having faster reaction times.

In contrast with the evidence of both Tarver et al (1976) and Hallahan et al (1973), Pelham (1979) reports *no* selective attention deficits in poor readers compared with good readers on *four* tests of selectivity (with poor readers IQ \geq 85). Pelham's tests included Hagan's Central-Incidental task (as discussed above). Moreover, low correlations between the four tasks suggested that they were not actually measuring the same construct. This certainly seems plausible: as discussed before, many of the tasks themselves involved far more than selective attention and may well have been tapping deficits in other areas. Pelham (1979) found no group effect on either the central or incidental visual task (contrary to results from Hallahan and colleagues). The equivalent auditory task used by Pelham suffered from similar methodological difficulties as the standard visual task. The auditory central task required the child to recall a list of relevant 'attended' words in the correct order. By contrast, the auditory incidental task required only free recall of the irrelevant

¹This task requires participants to match a picture with one of six other alternatives. The latency to the first response and the total number of errors are recorded.

words in any order. A group effect was found on the central task, with poor readers having poorer performance. Pelham's third task was one of speeded classification involving a choice reaction. Participants had to look for a particular dimension (e.g. a star) in a stimulus and press one of two buttons according to the value of that dimension (e.g. was it above or below the central dot). Stimuli could either contain distracting and irrelevant information or not. Poor readers were slower at this task, but there was no interaction with condition (distracting information or not); evidence consistent with that of Nicolson and Fawcett (1994) who found slower choice reaction times in children with dyslexia. The fourth task was a dichotic listening and shadowing task, again either with or without distracting information. Similar to the classification task, a group effect was found, but no group by condition interaction. The group effect can be easily accounted for by phonological theories and evidence for short term memory difficulties in children with dyslexia alone (see e.g. Snowling, 1987 or Jorm, 1983 for reviews of phonological theories and short term memory deficits respectively).

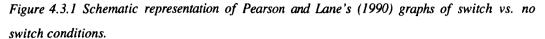
Thus, with respect to selective and sustained attention, findings have often been inconsistent, but hint towards some group differences. Some difficulty in interpretation of results arises when tasks designed to tap different aspects of attention are not equivalent, particularly when their small differences can be related to known areas of difficulty for dyslexic children.

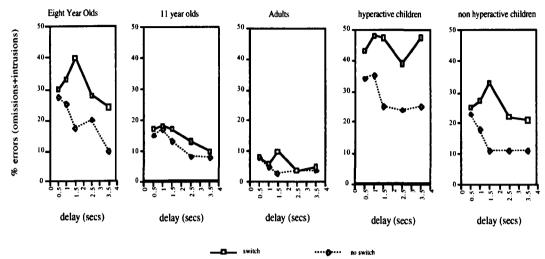
4.3 Reorientation and direction of attention

Reorientation of attention has also been a focus of research in the area of attention and literacy deficits, although not yet with children with dyslexia specifically. Brannan and Williams (1987), for example, utilised Posner et al's attention cueing paradigm (e.g. Posner, Nissen and Ogden, 1978; Posner and Snyder, 1974, Posner, Snyder and Davidson, 1980) to compare attention reorientation of participants with good and poor reading skills. The paradigm, in which valid or invalid cues are given to the position of the next target in the visual field (to which participants have to accurately direct their attention), showed that whereas those with good reading skills made use of the cues (when the likelihood was that they would be valid), those with poor reading skills did not. Moreover, analyses across both probability levels of the cue being valid (50% or 80% of the time) showed that whereas the poor readers demonstrated significantly lower accuracy rates at short stimulus onset asynchronies (50 milliseconds or less), at longer stimulus onset asynchronies this difference was not significant. However, Brannan and Williams' experiment was not conducted with people with dyslexia specifically (or rather psychometric details are not given to suggest this), but those with poor reading skills generally. Therefore, although it is likely that some participants in their poor reading group had dyslexia, others may have had more general learning difficulties. Results were interpreted in terms of either a transient system deficit or a rapid temporal ordering deficit.

Similarly, Pearson and Lane (1990) report a developmental lag in the reorientation of attention in hyperactive children. They used two tasks in order to investigate attention shifting in auditory and visual modalities. The auditory task compared the number of errors made in a target detection task. Lists were presented dichotically, but participants were instructed to attend to one ear only. A cue was given after around 10 seconds to either continue attending to the same ear (no switch condition) or to switch to the other ear (switch condition). The time before the onset of the target following this cue could vary from 0.5 to 3.5 seconds and it was during this time that participants had the opportunity to reorient attention. The results for the different groups which they tested are schematically represented in Figure 4.3.1.

Pearson and Lane report that an adult group and an 11 year old group 'recovered' performance (reached approximately the same level of performance as in the no switch condition) in the switch conditions after around 2.5 seconds delay, whereas an eight year old group did not manage to return to the same level of performance even after 3.5 seconds. A group of hyperactive children are reported to show similar performance to the 8 year old group in comparison to their controls. However, the graphs (see Figure 4.3.1: graphs 4 and 5) are not entirely convincing, failing somewhat to support this interpretation, since both hyperactive and non-hyperactive groups obviously had some difficulty reaching performance levels equal to the no switch condition. In fact, all five graphs shown could be said rather to reflect very well the differences between 'controlled' and 'automatic' processing reported by Weichselgartner and Sperling (1987) where 'first glimpse' attention is automatic and occurs rapidly following the cue with no effort required, but 'second glimpse' attention requires more controlled processing and effort and occurs later. It can be seen from Figure 4.3.1 below, that the maximum difference between switch and no switch conditions appears at 1.5 seconds rather than 0.5 or 1 second (which one would expect if time to 'recover' from the switch was necessary). In the comparison of hyperactive and non-hyperactive children, this also appears to be the case, although the hyperactive children show less evidence of a dissociation between 'automatic' and 'controlled' processing in addition to poorer performance overall.





In a more complex visual task (Pearson and Lane, 1990), a cue was given for the participants to direct their attention towards a target about to appear either to the left or the right of a computer screen. The target could appear at various inter-stimulus intervals following the cue. With a longer latency between cue and target, reaction times to the cue became faster, but only up to a certain point. When reaction times cease to become faster with increasing delay, the 'asymptotic stimulus onset asynchrony (asymptotic SOA)' is said to be reached; thought to be the time that it takes for attention to arrive at the target area. The time at which the asymptotic SOA occurred became earlier with age in the age groups studied: thus, visual reorientation became faster with age. In this task, the hyperactive group are reported to have higher error rates (sig.) and slower reaction times (n.s.), but are not different in their pattern of reorientation: asymptotic SOAs are reported to be similar even though mean RTs are different. There is therefore some evidence for differences between hyperactive and nonhyperactive children in the reorientation of attention, although the picture may be rather more complicated than first suspected. The deficit may involve speed, but is not necessarily entirely one of speed.

Speed deficits have been shown in many areas for children with dyslexia (see section 1.5). Naming speed has been found to be slower in both children and adults with dyslexia (e.g. Denckla and Rudel, 1976), whether due to reduced speed of information processing or linguistic deficits. Bowers and Wolf (1993) speculated that these naming difficulties may reflect disturbances of precise timing mechanisms necessary for the development of orthographic codes and their

integration with phonological codes. Nicolson and Fawcett (1994) found deficits in choice reaction time speed, but not simple reaction time in children with dyslexia. Lovegrove (e.g. 1994) posits a deficit in the transient system (the visual subsystem which responds to high temporal frequencies), since children with dyslexia are less able to detect flicker than their controls. Livingstone, Rosen, Drislane and Galaburda (1991) found abnormalities in the magnocellular system of brains of people believed to have had dyslexia; evidence consistent with, and providing an anatomical substrate for, the behavioural deficits found in rapid processing. Difficulty with speed of processing for children with language impairments has been investigated principally by Tallal and her colleagues (1973-1991). They found that children with language impairments and some children with dyslexia had difficulty in distinguishing the order of two rapidly sequentially presented sounds (Tallal, Miller and Fitch, 1993), although this view is controversial (Mody, Studdert-Kennedy and Brady, 1997).

4.4 The Experiment

Evidence of deficits in processing speed, timing, and speed of stimulus classification, coupled with evidence of an attentional deficit, suggest the hypothesis that at least some of these difficulties could be attributable to a deficit in rapid attention shifting. This chapter reports a study which investigated these abilities in children with dyslexia using a paradigm developed by Akshoomoff and Courchesne (1994). These authors identified a specific rapid attention shifting deficit in (four out of five) young patients with damage to the cerebellum. The present study used Akshoomoff and Courchesne's (1994) paradigm to compare children with dyslexia and their controls on one 'shift attention' and two 'focus attention' tasks. It therefore also has implications for both the cerebellar deficit hypothesis (because cerebellar patients have been found to have a rapid attention shifting deficit) and the DAD (because evidence of dual task deficits might actually represent difficulty rapidly shifting attention between two tasks rather than difficulty automatising skills). In the focus conditions, participants must respond to a pre-specified target shape in a constant stream of distractor shapes (which appear singly in the centre of a computer screen) whilst ignoring the others. In the shift condition, again the participant must respond only to the designated target shape, ignoring distractors, but in this case the target shape alternates following each correct response. On responding correctly to the first target shape, the target switches to the alternative shape, and the first target shape must be ignored, and so on. The paradigm is therefore of particular interest in that it provides a method of dissociating the three different components of attention; focus attention, shift attention and sustain attention. Furthermore, by analysing performance in terms of the interval between each stimulus and the time since the previous response was made, Akshoomoff and Courchesne established that their group of cerebellar patients had a deficit only for the shift attention condition, and then only for rapid attentional switches (i.e. when the interval time from the previous target was under 2.5 seconds).

Method

Participants

Participants with dyslexia had been diagnosed by a full psychometric assessment. They were of normal or above normal intelligence [operationalised as IQ of 90 or more on the full scale WISC-III (Wechsler, 1976)] and without known primary emotional, behavioural or socio-economic problems. Each participant's reading age or spelling age was at least 18 months behind his or her chronological age. Two age groups were used with mean ages 14.1 and 18.3 years (D14 and D18). Dyslexic participants had also all been previously tested on the Dyslexia Screening Test (DST)² (Fawcett and Nicolson, 1996) in order to establish their 'profile' of abilities on a range of skills.

Normally achieving control participants had also been given a short-form psychometric assessment and obtained normal or above normal IQ and reading and spelling ages in line with or above their chronological age. Two age groups were used, approximately matched for chronological age with the group with dyslexia (C14 and C18)³.

² The DST is a screening test for dyslexia for use by psychologists and non-psychologists. The DST comprises of 11 sub-tests in five areas (literacy skills, phonological awareness, verbal memory, motor skill and balance, and fluency). The sub-tests are as follows. The One Minute Reading and One Minute Writing tests reflect the number of words read or written correctly in one minute respectively. The Two Minute Spelling is the number of words spelled correctly in two minutes. Phonemic Segmentation tests the ability to break down a word into its constituent sounds, and to manipulate those sounds. Backwards Digit Span tests verbal memory for sequences of digits and the ability to repeat them in the opposite order. Rapid Naming involves the time taken to speak the names of pictures on a page full of common objects. Beads is the number of beads threaded in 30 seconds. Postural Stability reflects the degree of movement when pushed gently in the back. Verbal and Semantic Fluency tests are the numbers of words produced in one minute which either begin with a particular letter or are members of a particular category respectively.

³Two younger control participants were omitted from the experiment. One could not complete the experiment owing to apparent visual problems, another had unusual results which suggested that he tested the computer program to its limits by producing strange combinations of responses (e.g. what happens if the mouse is held down, if I click twice during the same trial and so on).

Participants had also all been assessed for clinical evidence of ADHD in accordance with the DSM IIIR (American Psychiatric Association, 1987). A score of at least 8 out of 14 markers of the disorder is required for clinical diagnosis. None of the participants showed evidence for ADHD (mean scores for each group are presented in Table 4.4.1) or showed signs of impulsivity/ hyperactivity. There were no significant differences between the score of the two groups although, as might be expected, there was a trend toward the dyslexic group having higher scores ($F_{1,24}=3.11$, p<0.1). All participants were given £3 for their co-operation. Psychometric data (means and ranges) for the four groups of participants are shown in Table 4.4.1. Most of the participants in this study had also taken part in the prism adaptation study (see Chapter 2) two years previously.

Mean IQ **Mean ADHD** Group Mean Age Mean RA n 7 108 (96-126) **D14** 14.1(13.2-15.0) 12.1 (8.3-15)* 1.43 (0-6) 6 **C14** 14.5 (13.8-15.2) 115 (101-124) 15.1(14.0-17+) 0.17 (0-1) 7 18.3 (16.5-20.1) 117 (101-133) **D18** 13.3 (14.0-17+) 0.29 (0-1)

Table 4.4.1. Mean psychometric data for the four groups (range in parentheses).

Design

C18

8

As in Akshoomoff and Courchesne's design, there were three conditions:

i) Focus condition 1 (350 trials):

TARGET: white ovals

18.2 (17.0-18.8)

ii) Shift condition (700 trials):

TARGET: white ovals and dark blue squares alternately

114 (96-122)

17+(17-17+)

iii) Focus condition 2 (350 trials):

TARGET: dark blue squares

Akshoomoff and Courchesne's repeated measures design was replicated, except that all participants experienced the conditions in the same order. This ensured that all participants had the same experience and that differences between groups could be more confidently ascertained. To allow investigation of the time required to either shift attention in the shift condition or re-engage attention after a response (focus conditions), four different interstimulus intervals were used, with trials

0

^{*} Two of the participants in this group had caught up with their reading since time of diagnosis. These were children of high IQ. Their spelling ages remained significantly lowered. It may also be worth noting that the older dyslexic group show a much greater average reading age deficit than the younger dyslexic group - see section 7.3 for further discussion of this point.

determined following a (pre-determined) pseudorandom order. Independent variables were therefore: group (dyslexic/ control), age group (14/18 years), condition (focus/ shift) and time since last target (continuous variable: wide range of different values).

The shapes used are shown in Appendix 4.4.1.

Procedure

The study was carried out on an Apple Macintosh computer with software designed to replicate the Akshoomoff and Courchesne procedure. Additional analysis software was used to categorise the results into various time periods since the last correct hit. Differences between this experiment and that of Akshoomoff and Courchesne (1994) are given in Appendix 4.4.2.

In brief, the computer presented a long sequence of shapes (white oval, white circle, dark blue square and light blue square) singly (at a mean rate of just over one per second) on the screen. Shapes occurred with different probabilities with potential target shapes (white oval and dark blue square) being less frequent (15% probability of occurrence each) than distractor shapes (35% probability of occurrence each). Four possible interstimulus intervals (ISIs) were presented randomly between stimuli (200, 400, 600 and 800 milliseconds). Stimuli and ISIs occurred in the same pseudorandom order for each participant; the random aspect of the data important only to preclude anticipatory responses to stimuli and to create a variety of time periods between targets.

Participants were instructed to ignore non-targets and respond to a specified target shape by pressing the mouse button. The target shape differed between conditions. In focus attention conditions, the target remained the same throughout the conditions (white oval in focus condition 1, dark blue square in focus condition 2), whereas in the shift attention condition, the target alternated between two possibilities (white oval and dark blue square). If targets were missed, then the target remained the same. Responses were recorded as hits if they came within 1000 ms of the target and were acknowledged as such by a short tone. In all conditions hits, misses, correct rejections and misses were recorded. While the procedure was closely modelled on that of Akshoomoff and Courchesne, a number of modifications were introduced following pilot testing (see Appendix 4.4.2 for details).

Participants were told that the computer would flash up various different shapes on the screen and that depending on the condition, they should press the mouse as fast as they could when they saw a particular shape. They were then given a 10 trial practice for the first condition which took them through the whole procedure and gave them direct feedback ("well done", "that was the target, you missed it" and so on). The main experiment followed the same procedure except that the stimulus durations and interstimulus intervals were shorter and that the only feedback was a short tone from the computer when a target was correctly detected. The first focus condition followed, in which participants were asked to respond to white ovals only. A 20 trial practice for the shift condition followed the first focus condition (longer in order to ensure that participants fully understood the procedure). It guided participants through the whole procedure with comments like "Well done. Now the target switches to the dark blue square". The shift condition then followed after a reminder of the instructions to respond to dark blue squares and white ovals alternately. Finally the second focus practice and condition were presented in which participants were asked to respond to dark blue squares only. The whole experiment lasted around thirty minutes.

Analyses

A critical component of the Akshoomoff and Courchesne design was the analysis in terms of the time since the last target was hit. For instance, in the shift attention condition, if a target appeared only 1 second after the previous target, then a rapid attention shift was required to detect both correctly. In the present experiment finer-grained temporal analyses were performed using 8 time bands (zero to 1, 1-2, 2-3....6-7 and >7 seconds).⁴

To perform this analysis, for each participant the time since the last correct hit occurred was calculated for every stimulus regardless of the response. Stimuli were then categorised according to the time since the last hit. The proportion of hits and false positives for each time category were then calculated for each person. The proportion of hits refers to the probability of a hit given a target (i.e. the number of hits divided by the number of possible hits); ideally 100%. The

⁴Due to the random aspect of the target order, the random interstimulus intervals, and the individual differences between targets hit, the time since the last correct hit for each trial is not a member of a discrete set of values. Akshoomoff and Courchesne chose to divide their scale into five time bands in order to examine accuracy and speed of attention shifting in their participants. In their analysis, rapid attention shifting was defined as 'from 0.4 to 2.5 seconds since the last target'. Other time bands used were; 2.5-4.5, 4.5-6.5, 6.5-10.5 and 10.5 to 30 seconds. We wanted a finer tuned analysis, with the ability to examine rapid attention shifting in a shorter time period than 2.5 seconds.

proportion of false positives refers to the number of false positives given a nontarget (i.e. the number of false positives divided by the number of possible false positives); ideally 0%. This information was used to obtain the d' measure of accuracy (from signal detection theory⁵) for each time category by finding the zvalue for each proportion (a negative value for proportions under 50% and a positive value for proportions over 50%). To calculate d', the z-value corresponding to the proportion of false positives was subtracted from that of the proportion of hits. Hit latencies were analysed in the same time categories.

Results

As noted earlier, each participant undertook three conditions, two 'focus attention' conditions and one 'shift attention' condition. Three-factor repeated measures ANOVAs were carried out on each condition separately to investigate the factors of group, age, and time since last hit. The dependent variables of accuracy $(d')^6$ and reaction time (s) were analysed separately.

Accuracy

For each condition, mean accuracy data and the mean percentage of hits and false positive responses are presented in Table 4.4.2. It may be seen that the overall performance accuracy was high, especially for the D18 and C18 groups.

Condition										
	Foc	Shift			Focus 2					
	d '	Hits	FAs	d '	Hits	FAs	d '	Hits	FAs	
D14	4.06 (2.73)	72	2.3	3.69 (1.86)	74	2.1	5.29 (1.81)	82	0.4	
C14	4.46 (1.95)	81	1.8	4.05 (1.42)	81	1.6	5.58 (1.54)	85	0.1	
D18	5.53 (2.48)	87	1.0	3.88 (1.85)	84	3.2	6.02 (1.66)	91	0.5	
C18	6.44 (1.75)	93	0.5	5.43 (1.83)	91	0.8	6.03 (1.56)	91	0.2	

Table 4.4.2. Mean accuracy data, percentage of hits and percentage of FAs in each group (standard deviation shown in parentheses)

⁵Using the standard formula (Swets, 1964): d' = Z(Yes/Target) - Z (Yes/ Non-Target).

⁶This d' measure is technically a measure of sensitivity, taking into account hits, misses and false positives. We use the term accuracy here in order to avoid any possible confusion.

i) Accuracy in focus attention conditions

Figure 4.4.1 shows the mean group accuracy on focus condition 1. Accuracy in focus condition 1 did not produce a significant difference between groups $(F_{1,24}=2.80)$ and there were no significant group-by-age interactions $(F_{1,24}=0.44)$.

Figure 4.4.2 shows the mean group accuracy on focus condition 2. Again, there was no group effect in this condition ($F_{1,24}=0.32$) or group-by-age interactions ($F_{1,24}=0.27$).

There were significant main effects of age in both focus conditions 1 and 2 with the younger groups being less accurate than the older groups ($F_{1,24}$ = 19.61, p<0.001 and $F_{1,24}$ = 4.93, p<0.05 respectively). A further set of three-factor ANOVAs (group, age, and shape as independent variables and the number of false positive responses made as the dependent variable) were carried out in each condition to investigate this issue further. In focus condition 1, a highly significant main effect of shape was found ($F_{2,48}$ =17.75, p<0.0001), suggesting that false positives were not randomly allocated amongst the shapes. This ANOVA also elicited a trend towards an effect of age ($F_{1,24}$ =4.06, p=0.055) and a significant age-by-shape interaction ($F_{2,48}$ =5.74, p<0.01). Such effects of age were not found in either of the other conditions. Analysis of the false positive responses for each shape suggested that the age effect was particularly apparent in the focus condition 1 because the younger participants were more likely than the older ones to hit white circles instead of white ovals. Figure 4.4.1. Mean accuracy for dyslexic and control groups across the different time periods in focus condition Standard error bars shown.

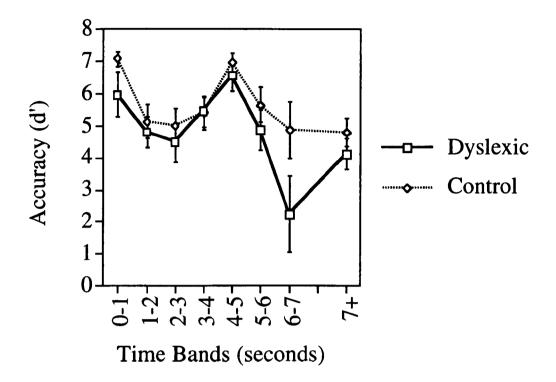
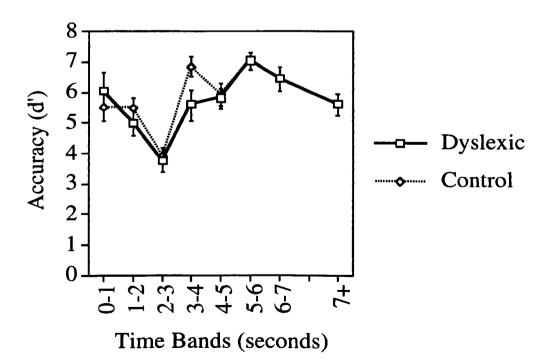


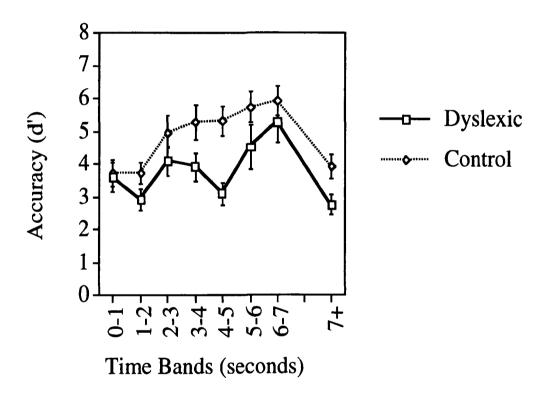
Figure 4.4.2. Mean accuracy for dyslexic and control groups across the different time periods in focus condition 2. Standard error bars shown.



ii) Accuracy in the shift attention condition

In contrast to the focus conditions, in the shift condition there was a significant main effect of group on accuracy ($F_{1,24}$ = 5.20, p<0.05). Although an age trend emerged ($F_{1,24}$ = 3.52, p<0.1), with younger participants being less accurate (see Table 4.4.2), there were no significant group-by-time or group-by-age interactions ($F_{7,168}$ =1.60, n.s., $F_{1,24}$ = 2.01, n.s.; see Figure 4.4.3). However, the data in Table 4.4.2 suggest that differences between dyslexic and control groups are greater amongst the older participants.

Figure 4.4.3. Mean accuracy for dyslexic and control groups across the different time periods in the shift condition. Standard error bars shown.



Hit latencies

Mean hit latencies are shown in Table 4.4.3. It may be seen that the groups with dyslexia did have slightly longer mean hit latencies than those of the normal achieving groups in all three conditions. However, as discussed below, these differences were significant only in the shift attention conditions. Separate three-factor repeated measures ANOVAs were again performed on each condition

separately to investigate the factors of group, age, and time since last hit on hit latency⁷.

i) Hit latency in focus attention conditions

Analyses revealed that groups were not significantly different in terms of hit latency in focus condition 1 ($F_{1,11}=2.71$, n.s.), and that there were no significant group-by-age interactions ($F_{1,11}=0.03$) or main effects of age ($F_{1,11}=1.72$, n.s.). Similarly, there were no main effects of group ($F_{1,23}=2.87$, n.s.) or group-by-age interactions ($F_{1,11}=0.00$) in focus condition 2. A main effect of age did emerge ($F_{1,23}=5.76$, p<0.05), with younger participants being slower than older ones.

ii) Hit latency in the shift attention condition

In the shift attention condition, there was a significant main effect of group on hit latency ($F_{1,22}$ = 6.67, p<0.05), with the normally achieving groups recording faster reaction times. There were no group-by-age or group-by-time interactions, or main effects of age ($F_{1,22}$ =0.00, $F_{7,154}$ =0.98 and $F_{1,22}$ = 2.64 respectively).

Table 4.4.3. Mean hit latencies for the four groups in each conditions (standard deviations shown in parentheses)

	Condition					
	Focus 1	Shift	Focus 2			
D14	0.65 (0.07)	0.64 (0.06)	0.64 (0.08)			
C14	0.60 (0.11)	0.58 (0.06)	0.60 (0.06)			
D18	0.61 (0.11)	0.60 (0.04)	0.60 (0.06)			
C18	0.54 (0.06)	0.55 (0.07)	0.56 (0.07)			

⁷Some participants' hit latency data could not be fully analysed, however, due to missing data points where they had not made any hits at all in a particular time category. This occurred particularly in focus condition 1, possibly owing to either fewer possibilities in this condition because of the random aspect of the time periods, or to practice effects (so that participants were correctly responding to more targets in the second focus condition). It is therefore possible that the participants with the lowest accuracy were omitted from the hit latency analysis. The fact that the F value changes very little between the two focus conditions, however, despite the large increase in included subjects (as can be seen by the degrees of freedom), suggests that this factor had little effect on the results.

Discussion

Comparison of the focus attention and shift attention conditions reveals a clear dissociation. For the focus attention task the performance of the normally achieving adolescents and those with dyslexia was similar in terms of both accuracy and speed. By contrast, for the shifting attention task, performance of the groups with dyslexia was significantly worse in terms of both accuracy and speed. This dissociation between shifting and focused attention tasks is an important finding, and to my knowledge this is the first study to have investigated this issue in dyslexia. It is particularly interesting as the participants did not show any clinical evidence of concomitant attention deficit disorder⁸.

However, whilst the findings do support an interpretation in terms of difficulties with more attentionally demanding tasks, the attention shifting deficit found was not specifically a rapid shifting deficit (as originally hypothesised). The intriguing result that emerged was that in the shift attention condition (see Figure 4.4.3), performance of the groups was near identical when very rapid attention shifting (under one second) was required and differed only as the time since shift increased. Whereas the control group appeared to benefit from a longer time to prepare for the next target stimulus (their performance improving with time since switch), the group with dyslexia did not appear to show this pattern. Overall, therefore, the dissociation between focus attention and shift attention performance suggests on the one hand that some kind of attentional deficit is present in children with dyslexia, but also that a rapid attention shifting deficit cannot explain the attentional difficulties reported. There may be some similarities here with Pearson and Lane's (1990) findings in hyperactive children (as discussed in section 4.3), where maximum deficits in comparison with controls occur after 1.5 seconds in the auditory shift condition, rather than immediately.

In order to eliminate potential artifacts, it was considered whether the lower performance of the groups with dyslexia could be due to boredom factors and increased difficulty in sustaining attention over the 15 minutes required by the shift attention condition, in contrast to the 7 or 8 minutes required by each focus condition. An additional analysis of the results from the first and the second half of the shift condition separately was performed to investigate this question. The

⁸It should be noted, however, that the boy in the young dyslexic group with the highest number of ADHD markers (six markers) did show the poorest performance on the shifting attention condition, with particularly poor performance on very rapid intervals and the longer intervals between targets. Watching this participant perform the task, it seemed that he missed several targets without realising that he had, and responded to neither of the potential targets for relatively long periods of time.

parallel lines in Figure 4.4.4 illustrate that the group with dyslexia showed no more difficulty sustaining their attention over the two halves of the experiment than the control group. The lower accuracy of the group with dyslexia in the main analysis was therefore not due to increased difficulty in sustaining attention.

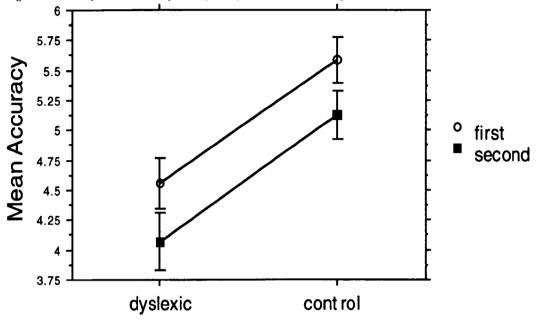
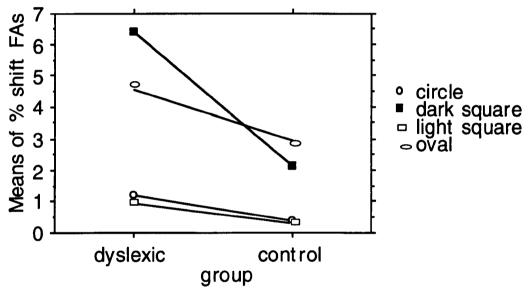


Figure 4.4.4. Shift Condition: split-half analysis interaction diagram (standard error bars shown)

It has also been suggested that groups with ADHD show greater impulsivity and inability to withhold responses (Barkley, 1994). It was considered whether this could also be the case with the participants with dyslexia, despite none of them showing significant evidence of ADHD. A post-hoc analysis of the number of false positives (only) made in the shift attention condition did reveal significant group differences ($F_{1,24}$ =8.09, p<0.01), with the groups with dyslexia producing more false positives than their controls, but the numbers involved were small (2.4% vs. 1.0% of the total number of trials in that condition; see also Table 4.4.2). Furthermore, mean false positive latencies indicated that in focus condition 1, the shift condition, and overall (though not in focus condition 2), the group with dyslexia had slightly slower false positive latencies than their controls (F_{1.20}=5.26, p<0.05 in the first focus condition, $F_{1,11}=0.008$, n.s. in the second focus condition, $F_{1,24} = 1.96$, n.s. in the shift condition). This suggests that false positives were not caused by impulsivity, and tallies with the impression that the participants with dyslexia were not particularly impulsive and they did not have difficulty withholding their responses; it was more that there was an air of confusion. Halperin, Wolf, Pascualvaca, Newcorn, Healey, O'Brien, Morganstein and Young (1988) suggest that a false positive response cannot be considered as a

measure of impulsivity without inspection of the latency of such a response. Further evidence arose from a plot of the number of FAs made to each shape in the shift condition (Figure 4.4.5). It can be seen that the pattern of errors is not random, with most errors being to the two potential targets (dark blue square and oval). Impulsivity generally might be expected to occur more randomly, although it is possible that impulsivity to the two potential targets has occurred. Overall, the evidence suggests that any false positives that the group with dyslexia did make over their counterparts were unlikely to have been due to any inability to withhold responses.

Figure 4.4.5. Plot of the number of false alarms made (as a percentage of the possible number) to each shape in the shift attention condition. it can be seen that the highest number of false alarms have occurred to the two potential targets (dark blue square and white oval).



Before considering possible interpretations of these results, it is important to consider their relevance to existing theories of dyslexia. Frith (1997) identifies three main causal theories of dyslexia; the phonological deficit hypothesis, the cerebellar deficit hypothesis and the rapid temporal processing deficit hypothesis.

The phonological deficit hypothesis is probably the most widely accepted and well established view of the aetiology of dyslexia (Snowling, 1987; Stanovich, 1988; Vellutino, 1979). As discussed in section 1.3, it posits that a deficit in the processing of phonological information is the basis of reading and spelling problems in dyslexia. The hypothesis is based on models of reading development which stress the importance of phonology in learning to read. Bradley and Bryant (1983) have shown that children with dyslexia suffer from poor rhyme detection

ability from as early as four years old and hence before learning to read, suggesting a causal relationship between phonology and literacy. In terms of the phonological deficit hypothesis, results of this experiment would initially appear to suggest only that children with dyslexia have additional deficits to those associated with a phonological impairment. However, further analyses in terms of verbal labelling (see below) do provide indirect support for the phonological deficit hypothesis.

As discussed in section 1.6, the cerebellar deficit hypothesis of dyslexia (Nicolson, Fawcett and Dean, 1995) was based on studies of children with dyslexia which demonstrated that they suffered a wide range of deficits in terms of phonology, motor skill, balance, automatisation and speeded performance (Nicolson and Fawcett, 1995). Further evidence derived from a study (Nicolson, Fawcett and Dean, 1995) establishing a dissociation between time estimation and loudness estimation previously claiming to be specific to patients with cerebellar damage (Ivry and Keele, 1989). A further study established that groups of children with dyslexia showed impairments (even in comparison with reading age controls) on a range of clinical cerebellar tasks (Fawcett, Nicolson and Dean, 1996). These authors suggest that children with dyslexia suffer a mild abnormality in the cerebellum from birth, and that this leads to incomplete automatisation of a range of skills, with articulation being one of the key early skills affected. Mild difficulties in articulation are likely to lead to phonological difficulties, and hence the symptoms highlighted by phonological deficit accounts.

While it is important to note the difference between a developmental disorder and an acquired disorder of the cerebellum, one might nonetheless expect the cerebellar deficit hypothesis to predict that children with dyslexia would suffer from many of the same deficits as patients with damage to the cerebellum. Akshoomoff and Courchesne (1994) found a rapid attention shifting deficit in patients with cerebellar damage and Courchesne et al (1994) found similar deficits in children with autism (in whom they report the cerebellum has been shown to have abnormalities). Unfortunately, there is considerable dispute as to whether rapid attention shifting is indeed associated with the cerebellum (e.g. Helmuth, Ivry and Shimizu, 1997; see also Yamaguchi, Tschuchiya and Kobayashi, 1998)⁹. However, Le, Pardo and Hu (1998) in an fMRI study of attention found that the cerebellum was indeed activated during shifting attention. Similarly, Allen, Buxton, Wong and Courchesne (1997) found cerebellar involvement in an attentional task, independent of motor involvement. Nevertheless, whether any cerebellar involvement in attentional tasks is related to the attention act or attentional switching itself, or whether, as Courchesne and Allen (1997) suggest, it is related to the cerebellar role in preparation of responses remains to be seen. These fundamental uncertainties limit the inferences that can be made from these null results.

Arguments for the rapid temporal processing hypothesis have also already been summarised in the introduction (see sections 4.3 and 1.4). The good performance in this study of adolescents with dyslexia under rapid conditions provides no support for such a deficit. It could be argued that the rapid temporal processing deficit theory predicts deficits in processing more rapid information than that used here (just under one second), although the original evidence for the theory only considered the processing of incoming information, allowing no time for any higher level control (such as a shift of attention). Thus, it seems that the pattern of results obtained here, where the performance of the participants with dyslexia was relatively improved on the rapid shifting conditions, would be unlikely if there was a pan-sensory rapid temporal processing deficit in dyslexia.

The results therefore are not directly accounted for by any of the causal theories of dyslexia discussed so far. One further plausible explanation is that the children with dyslexia have more limited attentional resources, as predicted by the Dyslexia Automatisation Deficit hypothesis (Nicolson and Fawcett, 1990: see section 1.5). Since a rapid attention shifting deficit was not found, the notion that dual task deficits in dyslexia are caused by a lack of automaticity on one of the tasks has not been questioned. If behaviour is not automatic in children with dyslexia, they need to input greater attentional resources to undertake any given task. While for easy tasks this greater attentional load can be carried without performance decrement, it

⁹In brief, the disputes centre around whether or not parietal abnormalities have been found in addition to cerebellar damage in participants used and whether the apparent decreased 'validity effect' found in autistic children (thought to have cerebellar abnormalities) using Posner's attention cueing task (see section 4.3) at short SOAs is that or more likely a decreased validity effect at longer SOAs for controls. In any case, Helmuth et al (1997) do not find a similar pattern in cerebellar patients. In addition, it is possible that part of the dispute may arise from differing definitions of shifting attention; namely whether or not a 'shift' of attention has to involve shifting from one thing to another or merely orienting towards something.

will lead to increasing difficulties as tasks demand more resources. Thus, adding to the complexity of any task, or to the resources required to perform it, may disproportionately disadvantage children with dyslexia. The shift attention condition was obviously more complex, with reduced accuracy in *both* groups. There is also some evidence for effortful processing deficits in children with ADHD (see e.g. Borcherding, Thompson, Kruesi, Bartko, Rapoport, Weingartner, 1988; also Ackerman and Dykman, 1982 and Ackerman, Anhalt, Dykman and Holcomb, 1986 discuss children with both attention and reading disorders).

In order to investigate the cause of the performance differences further, a correlational analysis was performed, limited to the participants with dyslexia. This used two dependent variables for the shift attention condition, namely (i) the accuracy (d') from the 0 to 1 seconds shift attention category and (ii) ability to shift attention generally (the mean of all the accuracy values in that condition), together with (iii) age, and (iv) a variety of measures from the Dyslexia Screening Test (Fawcett and Nicolson, 1996).

There were highly significant correlations between (amongst others) backwards digit span and ability to shift attention overall (r=0.73, p<0.01). This correlation, and that between rapid attention shifting and digit span backwards, became increasingly significant when effects of age were partialled out (r=0.77, p<0.01 and r=0.88, p<0.001 respectively). These correlations are shown in Table 4.4.4. The values are high and thus initially appear to support a relationship between the attention shifting condition in this experiment and working memory. However, it is interesting to note that score on the segmentation subtest, as well as correlating with backward digit span (even when effects of age were partialled out), correlates significantly (r=0.84, p<0.01) with ability to shift attention overall, but not with ability to shift attention rapidly (r=0.36, n.s.). This dissociation corresponds to that shown by the children with dyslexia, whereas backward digit span correlates significantly with both rapid shifting and mean shifting. Interesting, is that informal observations made of participants performing the task suggested that a verbal labelling strategy (rehearsing '[target is] dark blue square' and '[target is] white oval') while awaiting the target is both common and beneficial. These correlations suggest that the difficulty for the dyslexic group may lie in these verbal labelling and rehearsal strategies (Miles, 1983). This conclusion would be consistent with the phonological deficit hypothesis and is similar to Tarver et al's (1976) finding that verbal rehearsal aids selective attention (see section 4.2).

Tarver et al showed that encouraging verbal rehearsal significantly increased central task recall and decreased incidental task recall. (However, as discussed, this strategy effect is equally likely to be due to a change in memory rather than attention). Similarly, Torgesen and Goldman (1977) suggested that short-term memory deficits in dyslexic children may reflect lack of ability or inclination to use efficient task strategies on some memory tasks, finding that encouragement of rehearsal significantly improved recall scores in this group.

An explanation of the dissociation found in the present experiment in terms of different strategies the focus and shift conditions may elicit for the different groups was also considered. This was necessary because group differences in both reaction times *and* accuracy were found on the shift, but not the focus conditions. Ollman (1977) discussed how strategy shifts over conditions in an experiment can be problematic, particularly in situations of speed-accuracy trade-off. The group difference found in reaction time, for the shift condition only, may be considered to indicate a strategy shift for one of the groups (e.g. the dyslexic group performing the focus condition using a 'spatial template' of the target, but the shift condition by naming the current target). However, examination of the mean reaction times and standard deviations for each group (see Table 4.4.3) suggests that reaction times are actually remaining fairly constant and it is likely to be the smaller variance which has elicited the group effect for reaction times in the shift condition.

	Shift:	Shift:	Backspan	Segment	1 min	2 min	Nonsense	Naming	Stability	Beads
	mean	rapid			reading	spell	Read			
Shift: mean	1	0.68*	0.77**	0.84**	0.62*	0.50	0.55	-0.20	0.33	0.42
Shift: rapid	0.68*	1	0.88**	0.36	0.57*	0.02	0.45	-0.30	0.44	0.21
Backwards span	0.77**	0.88**	1	0.58*	0.60*	0.25	0.51	-0.40	0.24	0.29
Segmentation	0.84**	0.36	0.58*	1	0.46	0.75**	0.44	-0.28	0.29	0.23
1 min reading	0.62*	0.57*	0.60*	0.46	1	0.22	0.69**	-0.40	0.19	-0.16
2 min spelling	0.50	0.02	0.25	0.75**	0.22	1	0.35	-0.39	0.01	0.41
Nonsense Readin	g 0.55	0.45	0.51	0.44	0.69**	0.35	1	-0.41	0.53	0.08
Rapid Naming	-0.20	-0.30	-0.40	-0.28	-0.40	-0.39	-0.41	1	-0.00	0.18
Stability	0.33	0.44	0.24	0.29	0.19	0.01	0.53	-0.00	1	-0.15
Beads	0.42	0.21	0.29	0.23	-0.16	0.41	0.08	0.18	-0.15	1

Table 4.4.4. Correlation Matrix of DST, and shift attention d'variables with effects of age partialled out. Participants with dyslexia only (n=14). *=p<0.05, **=p<0.01

The explanation posited for the results, therefore, is that many of the participants with dyslexia had some difficulty keeping track of which stimulus was the current target under shift attention conditions; possibly attributable to failure to use a verbal labelling strategy in order to maintain target details over several distractor trials. This argument is consistent with the relatively good performance with shorter time intervals, together with the lack of improvement to the same extent as the control group with longer intervals. As well as offering support to the phonological deficit hypothesis and the dyslexia automatisation deficit hypothesis, the interpretation is also consistent with other evidence that adults with dyslexia have a deficit in dynamic working memory maintenance. Smith-Spark (1997) found no deficits in students with dyslexia on a static spatial memory task (recalling four positions highlighted in a grid) nor on a low memory load dynamic spatial memory task (recalling the last four positions highlighted on a grid with 4, 6 or 8 highlighted in total). However, on a high load dynamic spatial memory task (recalling the last four of 10 positions highlighted in a grid) students with dyslexia showed working memory deficits. As the task was purely spatial, results suggested dynamic working memory deficits under high dynamic load even on non-phonological tasks.

An alternative and novel explanation of the results is in terms of forgetting and/ or activation levels. This explanation assumes a model of memory where stimuli to be remembered become 'activated' in memory, with activation on a continuous scale (cf. Anderson, 1983). As time progresses, lack of rehearsal results in the gradual decay of the activation. If, in children with dyslexia, this decay occurs faster than usual, then the activation between two or more competing stimuli will soon become equal. In the case of the shifting attention paradigm reported here, two stimuli to be activated in working memory alternately would be likely to cause particular difficulty, since both would be activated more strongly than anything else, but the differential between them would not be large. The strongest differential between stimuli, regardless of the decay rate, would occur immediately following a target shift.

Explanation of the difficulty of children with dyslexia in this task using the idea of activation levels, has a possible analogy to common spelling errors reported in children and adults with dyslexia. Persistent confusion between homophones (e.g. stationery/ stationary or more commonly there/ they're/ their), for example, may result from knowledge of the different spellings, but neither being more strongly associated with the meaning of the word than the other. This could be since they are so often presented together during spelling correction. Furthermore, if the child with dyslexia does pick the correct version by chance, little or no feedback is given.

The explanations of the results found in this chapter are by no means the only possible, and are not mutually exclusive. In fact, it is highly plausible that any rapidly decaying memory trace in children with dyslexia may decay more rapidly precisely because resources are being allocated to other behaviours, leaving less for rehearsal or updating. A general resource deficit hypothesis in this case can be easily investigated by giving the same shifting attention task, but with fairly heavily degraded stimuli, so that it requires more effort to discern the targets (see Chapter 5). An investigation of the issue of memory decay might be to increase the number of potential targets; whilst keeping the demands on attentional resources constant, the balance of activation between the three (or more) targets would be less equal at any one time, since at least one target will have been left without any activation for longer. Analysis of false positives (examining whether they show a random pattern of errors, consistent with a working memory explanation, or a pattern suggesting that the target which would have the least activation at the time is less often mistakenly hit than others) may be able to give support to one or other

of the hypotheses. It is also plausible that the focus attention task performance reached ceiling level for control groups and that this is the reason no differences between groups were found on this condition. This possibility is actually similar to that of a general resources deficit, suggesting that as anything becomes more complex or difficult, children with dyslexia will show deficits. Thus, the concepts of both resource deficits in attention, and more rapid memory decay, offer interesting and potentially valuable avenues for further research in dyslexia and possibly also its remediation.

In summary, the present study has established two key findings. First, children with dyslexia showed a dissociation between two forms of attentional task; namely significantly impaired performance on the shift attention task despite normal performance on the focus attention task. Furthermore, the shift attention performance also showed a dissociation; namely impaired performance several seconds after a target switch, despite normal performance immediately after the target switch. These results are interpreted as reflecting a difficulty in maintaining a frequently changing target in mind while undertaking a resource-intensive task; a difficulty arising from limited attentional resources together with difficulties in using a verbal labelling strategy.

Chapter 5

Attention, Resources and Dyslexia: a further look

Investigation of general resources and automaticity explanations of the dissociation between focus and shift attention found in Chapter 4

Summary:

The experiment presented in this chapter attempted to further investigate the reported links between attention and dyslexia. Moreover, it presented an opportunity to distinguish between a more general resources deficit explanation and an automatisation deficit explanation as postulated (but left unspecified) for the results in Chapter 4. The focus and shift attention paradigms used in Chapter 4 were re-employed, but with some alterations. In this experiment there were four conditions: the standard focus and shift attention conditions (as before); together with two equivalent conditions using visually degraded stimuli. The reasoning behind the degraded stimuli was that using these a general resources deficit would disproportionately disadvantage children with dyslexia, because they would not have resources to cope with the extra processing required. In contrast, the Dyslexia Automaticity Deficit Hypothesis would predict that degraded conditions would disproportionately disadvantage the control children if there was a shape recognition automaticity deficit in children with dyslexia. This is because degraded conditions would prevent control children from performing the task automatically, whereas it would make little difference to the dyslexic children who were not processing the shapes automatically anyway. As predicted by both hypotheses, a group-by-visibility interaction was found. This showed that control performance was reduced more under degraded conditions. Results therefore support the Dyslexia Automatisation Deficit Hypothesis (Nicolson and Fawcett, 1990).

5.1 Introduction

In Chapter 4, Akshoomoff and Courchesne's rapid attention shifting paradigm was used to investigate a hypothesised rapid attention shifting deficit in children with dyslexia (Moores, Nicolson and Fawcett, submitted). This hypothesised difference was not found, but the group with dyslexia did show deficits in shifting attention overall, appearing not to show improvement with extra time to prepare for their responses. In fact, their performance declined over time relative to that of the controls. In contrast, there were no significant differences between groups in two 'focus attention' conditions. Two main possible explanations were investigated for these findings. One possibility, of a general resources deficit (causing increased difficulty under more complex and resource consuming conditions) together with difficulty using a verbal labelling strategy to remember the current target, is a focus of the present chapter. A further possibility, of more frequent 'lapses' of attention in children with dyslexia is considered in Chapter 6.

The general resources/ verbal labelling explanation of the dissociation found in Chapter 4 was derived jointly from the Dyslexia Automatisation Deficit Hypothesis (DAD: Nicolson and Fawcett, 1990) and the phonological deficit hypothesis (e.g. Snowling, 1987; Stanovich, 1988; Vellutino, 1979). The DAD postulates that children with dyslexia have difficulty making their performance automatic and that much of the time they have to put in greater effort than their controls in order to achieve the same level of performance (by conscious compensation): see section 1.5. This means that if a secondary task is added to the original task, or if the original task is made more difficult, their performance will suffer to a greater extent than that of their controls since they have fewer remaining resources in order to compensate further. However, if a task cannot be completed automatically anyway, no differences between dyslexic and control groups are predicted (at least in terms of accuracy, differences might occur in terms of speed). Similarly, group differences would not be expected if the task demanded relatively little attentional capacity even when performed in a controlled manner, since in this case resources would remain in order to allow 'conscious compensation' to be put into operation.

Part of the explanation suggested in Chapter 4 therefore hypothesised that the cause of the group difference was the extra complexity and the extra attention required in the shift attention condition in comparison with the focus conditions. However, the DAD would predict difficulties for dyslexic (in comparison with control) children on complex tasks *only* if at least part of the task would usually be performed automatically. It suggests that the failure of automatisation *leaves* fewer remaining resources in order to cope with any extra demands (within an otherwise normal capacity). However, an alternative, yet simpler, explanation is that children with dyslexia *have* fewer resources overall. This possibility has not yet been explored, but is important since it could potentially account for research findings which have formed the basis of the Dyslexia Automatisation Deficit Hypothesis (dual task balance for example: see section 1.5) as well as results found in Chapter 4. The experiment reported in this chapter therefore attempted to make a distinction between these two theoretically important possibilities and also to replicate the previously found dissociation between focus and shift attention conditions.

Seminal early work on the normal processes of automatisation was reported by Shiffrin and Schneider (1977, p155) who suggested a mechanism for an automatisation deficit in reading. In Shiffrin and Schneider's framework, a visually presented word could be processed at several different levels: contrast and colour, followed by features, then by letters, and so on until semantic meaning. They suggest that such a sequence should occur automatically in a skilled reader. It seems reasonable to suggest (as the DAD would) that the same might be true of shape recognition. There is some preliminary evidence that dyslexic children do not benefit from semantic priming to the same extent as controls (Hartley, Lindley and Nicolson, in preparation). There is also evidence for decreased phonological priming in dyslexic children with poor non-word decoding skills (McPherson, Ackerman, Holcomb and Dykman, 1998). This is therefore evidence that the normally automatic process of priming ('spreading of activation' in the brain through related concepts; see e.g. Meyer and Schaneveldt, 1971) may be reduced in children with dyslexia. Perhaps then, dyslexic children have to consciously think through a series of features before identifying shapes. In control children, one might expect that a shape with four sides of equal length and four 90 degree angles would automatically 'prime' the label square. However, if some kind of more resource-consuming search or process has to occur in order for dyslexic children to reach this point, then their poorer performance on the tasks presented in Chapter 4 can be elegantly accounted for by the DAD. Dyslexic children are of course slower at naming anyway, as already mentioned (see section 1.5).

Two potential explanations were investigated for the dissociation in Chapter 4:

i) a general resource deficit in dyslexia, i.e. that children with dyslexia have a decreased resource capacity.

ii) an automaticity deficit in children with dyslexia, i.e. fewer remaining resources because part of the task has not been automatised and is therefore using more resources than it might otherwise. These possibilities were investigated by using the same paradigm as in Chapter 4, but with visually degraded stimuli in addition to the normal stimuli. Degraded stimuli require more attentional capacity to process and therefore have the properties that (i) they use more resources and (ii) they prevent shape recognition occurring automatically. Thus, a general resources deficit would predict that the dyslexic children would be disproportionately affected by the degraded stimuli (since they would make the task that little bit more resource consuming: resources which the children with dyslexia may not have). In contrast, the DAD would predict that degradation of stimuli would make little difference to the children with dyslexia, since they perform the shape recognition component of the task with so much difficulty already. The control children should have relatively worse performance on the degraded conditions since the degradation of the stimuli would prevent them from performing the shape recognition component of the task in an automatic manner. The general resources deficit hypothesis would therefore predict a group-by-visibility interaction in one direction (children with dyslexia would perform worse in degraded conditions whereas control children can cope with the degradation) and the automatisation deficit hypothesis in the other direction (control children are affected more by the degradation since it precludes automatic performance of the shape recognition component of the task). Should neither of these possibilities hold true, a further explanation would be needed in order to account for the focus-shift dissociation observed in Chapter 4¹. Using degraded stimuli also has the potential advantage of reducing ceiling effects on focus conditions. Ceiling effects have previously been found to be problematic in these type of tests (e.g. Parasuraman, Mutter and Molloy, 1991 cited in Robertson et al, 1997).

5.2 The experiment Method

Participants

Psychometric data (means and ranges) for the four groups of participants are shown in Table 5.2.1. As in the Chapter 4 experiment, it can be seen that the older dyslexic group have, on average, more severe reading difficulties than the younger dyslexic group. See section 7.3 for further discussion of this point.

¹A similar design was used by Snowling, Goulandris, Bowlby and Howell (1986) in their examination of dyslexic children's difficulties with nonword repetition: auditory stimuli were heard in differing levels of background noise in order to investigate whether the dyslexic children were differentially affected by the noise. Because both groups were equally affected by the noise, a perceptual deficit was concluded less likely than one of speech segmentation.

Group	n	Mean Age	Mean IQ	Mean RA	Mean ADHD
D15	8	14.6(13.5-15.2)	113 (96-134)	13.4 (9.3-17)*	1.0 (0-6)
C15	9	15.0 (13.8-16.0)	116 (101-129)	16.2 (14.0-17+)	0.1 (0-1)
D19	6	19.0 (17.3-20.9)	115 (101-131)	12.8 (9.3-16.0)	0.3 (0-1)
C19	5	18.8 (17.8-19.5)	114 (96-130)	17+	0.0

Table 5.2.1. Psychometric data for the four groups of participants (range shown in parentheses).

Participants with dyslexia had been diagnosed by a full psychometric assessment. They were of normal or above normal IQ [operationalised as IQ of 90 or more on the full scale WISC-III (Wechsler, 1976)] and without known primary emotional, behavioural or socio-economic problems. Each participant's reading age or spelling age was at least 18 months behind his or her chronological age. Two age groups were used with mean ages 14.6 and 19.0 years (D15 and D19).

Normally achieving control participants had also been given a short-form psychometric assessment and obtained normal or above normal IQ and reading and spelling ages in line with or above their chronological age. Two age groups were used, approximately matched for chronological age with the group with dyslexia (C15 and C19).

Participants had also all been assessed for clinical evidence of ADHD in accordance with the Diagnostic and Statistical Manual of Mental Disorders: 3rd edition (DSM IIIR: American Psychiatric Association, 1987). A score of at least 8 out of 14 markers of the disorder is required for clinical diagnosis. None of the participants showed evidence for ADHD. There were no significant differences between the score of the two groups. All participants were given £5 for their co-operation in this study together with the one reported in Chapter 6. The majority of the participants (18 out of 28) had taken part in the experiment reported in Chapter 4 around 9 months earlier. All participants also took part in the experiment reported in Chapter 6, which in each case was performed five minutes before this one (with no break longer than around 2 minutes). These factors are not believed to have affected the main results in any way.

^{*} One boy's reading age from this group was lost at the time of the experiment. However, it has since been found as part of an experiment not reported in this thesis (6 months later) that his reading age remains lowered. In addition, three of the participants in this group had caught up with their reading since time of diagnosis. These were children of high IQ. Their spelling ages remained significantly lowered.

Design

There were four conditions:

- i) Focus condition normal (250 trials):
 - TARGET: triangles
- ii) Shift condition normal (500 trials):
 - TARGET: triangles and circles alternately
- iii) Focus condition degraded (250 trials):

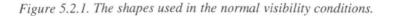
TARGET: squares

- iv) Shift condition degraded (500 trials):
 - TARGET: squares and diamonds alternately

A repeated measures design was used, counterbalanced so that half the participants in each group experienced the degraded conditions (iii and iv) first and the other half experienced the normal conditions (i and ii) first. Focus attention and shift attention conditions within each visibility condition were always performed in the same order (focus first). This design was used to ensure that any order effects of either practice or fatigue were accounted for across the factor of visibility.

As in the Chapter 4 experiment, a large variety of different time periods between targets was achieved by using different interstimulus intervals and a (predetermined) pseudo-random stimuli order. Independent variables were therefore: group (dyslexic/ control), age group (15/19 years), condition (focus/ shift) and visibility (normal/ degraded). Since the previous investigation suggested no real group differences, time since last response was not included as a factor.

Due to the possibility of confusion between the oval and the circle in the Chapter 4 experiment, and in order to make the degraded visibility conditions valid, 4 new shapes were chosen (all of the same colour so that in degraded conditions the shapes could not be recognised by colour alone). The shapes were all grey: triangle, square, circle and diamond. The degraded shapes had been altered within the Hypercard environment; firstly by a random pixel removing procedure so that the shapes were made up of dots, rather than solid, and secondly by further distortion of some of the edges (using the eraser). The shapes are shown in Figure 5.2.1 and 5.2.2.



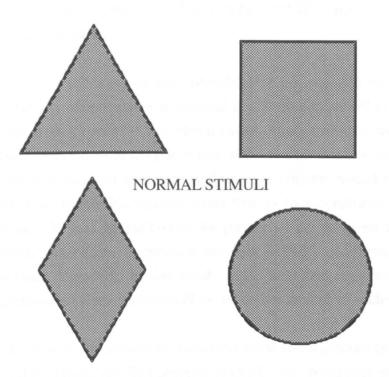
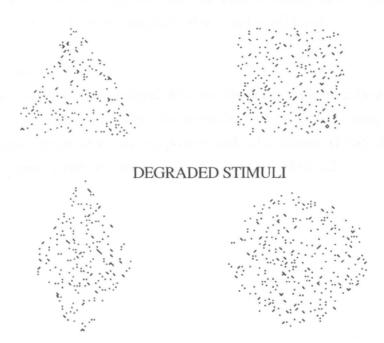


Figure 5.2.2. The shapes used in the degraded visibility conditions.



Procedure

As before, the study was carried out on an Apple Macintosh computer with software designed to partially replicate the Akshoomoff and Courchesne procedure (written by Prof. Rod Nicolson, Department of Psychology, University of Sheffield). Additional analysis software (also written within Hypercard) was used to determine the percentage of correct hits and false alarms (FAs) for each person in each condition.

Since a large variety of time periods were not required in this experiment, the probability of occurrence of potential targets was increased to 0.2. The number of trials in each condition was also reduced. These measures were possible because fewer trials were needed (since time since last hit was not being investigated) and were both taken in order to avoid an over lengthy procedure causing fatigue or boredom. Using this method meant that all four conditions still took around 30 minutes in total. In addition to the previous steps to avoid fatigue, a ten minute 'break' was given in between the two visibility conditions of the experiment (normal/ degraded). In this break, some short non-computer reading tests were administered along with a small interview not relating to studies reported here².

Participants' instructions remained the same and a practice prior to each condition was given as before. The analysis of results was performed in the same way as in Chapter 4 (examining measures of both accuracy and bias) except that in this experiment, the time since last hit was not considered.

Results

The converted (using signal detection theory: see Chapter 4) results consisted of (i) measures of accuracy, (ii) bias towards pressing/ not pressing the mouse, and (iii) reaction times of correct responses and false alarms (FAs). Mean results for the four groups can be seen in Table 5.2.2 and Table 5.2.3.

²This break was considered to be beneficial to both groups, rather than distracting, and was received well by all participants. Concentrating on a 'flashing' computer screen for any length of time is tiring for the eyes, at least, particularly for people unaccustomed to it.

		Condition										
	Normal Visibility					Degraded Stimuli						
	Focus Shift			Focus			Shift					
	d'	bias	RT	d'	bias	RT	d'	bias	RT	d'	bias	RT
d15	2.8	28	0.63	2.6	33	0.61	2.8	22	0.66	2.4	44	0.66
c15	3.2	36	0.60	3.3	34	0.57	3.1	27	0.61	2.8	44	0.60
d19	3.0	27	0.61	2.9	19	0.57	3.2	17	0.64	2.8	31	0.62
c19	4.6	.26	0.56	3.7	41	0.61	3.8	.05	0.60	3.2	34	0.61

Table 5.2.2. Mean results of accuracy, bias and reaction time (correct RT [secs] only: not including FAs) in each of the conditions for each of the four groups.

Table 5.2.3. Mean probability of hits and false alarms (FA) for the four groups in each of the conditions

	Condition							
	No	rmal	Visibil	ity	Degraded Stimuli			
	Fo	cus	Shift		Focus		Shift	
	Hit	FA	Hit	FA	Hit	FA	Hit	FA
d15	0.85	0.05	0.83	0.05	0.87	0.06	0.77	0.05
c15	0.89	0.03	0.89	0.03	0.89	0.04	0.82	0.04
d19	0.88	0.04	0.88	0.05	0.91	0.04	0.86	0.05
c19	0.98	0.02	0.91	0.02	0.94	0.04	0.90	0.03

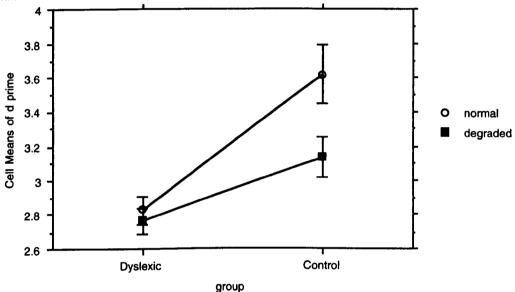
i) Accuracy

The d' measure of accuracy was used as the dependent variable in a four-factor mixed measures ANOVA investigating effects of group (dyslexic/ control), age (15/19 years), visibility (normal/ degraded) and condition (focus/ shift).

As expected, the effect of visibility was highly significant ($F_{1,23}=12.28$, p<0.005), with visually degraded stimuli leading to lower accuracy. The effect of condition was also highly significant ($F_{1,23}=20.38$, p<0.0005), with shift conditions proving to be most difficult. In addition, the main effects of group and age were significant ($F_{1,23}=21.23$, p<0.0001 and $F_{1,23}=13.48$, p<0.005), with dyslexic and younger groups being less accurate.

Consistent with both experimental hypotheses, a visibility-by-group interaction emerged ($F_{1,23}$ =8.10, p<0.01). This showed that whereas control groups became much worse in the degraded conditions, the dyslexic groups' performance was decreased to a lesser extent (see Figure 5.2.3). A condition-by-group-by-age interaction was also significant ($F_{1,23}$ =4.45, p<0.05), showing that the older controls had the largest difference in accuracy between focus and shift conditions, but the younger controls had the smallest.

Figure 5.2.3. Plot showing the significant visibility-by-group interaction (collapsed over age groups). It can be seen that the performance of the dyslexic groups is lower than that of the controls in both conditions, but that control performance is decreased more by the degraded stimuli. Standard error bars are shown.



Four three-factor ANOVAs were performed on the data, examining the effects of age and group on accuracy (i) across conditions in normal visibility, (ii) across conditions in degraded visibility, (iii) across visibility in focus conditions and (iv) across visibility in shift conditions. Results are presented in Appendix 5.1.1. Effects of group were significant in all conditions ($F_{1,23}$ = 20.92, p<0.0001; $F_{1,23}$ =10.86, p<0.005; $F_{1,23}$ =17.59, p<0.0005 and $F_{1,23}$ =15.36, p<0.001 respectively), with dyslexic groups having poorer performance. Other effects were also significant, as would be expected from the four-factor ANOVA and as are reflected by the two-factor ANOVAs (see below). The interesting effects specific to these particular analyses were those of visibility or condition. It was interesting to note that whereas the effect of visibility was significant under shift conditions ($F_{1,23}$ =13.69, p<0.005), it was not significant under focus conditions ($F_{1,23}$ =1.71). Similarly, the effect of condition (focus or shift) was significant

under degraded ($F_{1,23}=14.09$, p<0.001), but not under normal conditions ($F_{1,23}=3.25$, p=0.08). The extra difficulty presented by the shift in comparison to the focus condition therefore caused a significant effect only in degraded conditions, with performance being lower in the shift condition. Similarly, the extra difficulty presented by the degraded stimuli was significant only in the shift conditions.

Group-by-visibility/ condition interactions were non-significant in each case. However, under shift conditions, the group-by-visibility interactions showed a trend ($F_{1,23}=3.70$, p=0.07 compared to $F_{1,23}=2.85$, n.s.) toward the degraded stimuli impacting on the control groups more (a similar pattern to that shown in Figure 5.2.3). The equivalent analyses under normal and under degraded conditions for attentional conditions (focus/shift) showed no significant group-bycondition interactions ($F_{1,23}=0.61$ and $F_{1,23}=0.04$ respectively) suggesting that both groups were affected by the focus/ shift manipulation to the same extent.

In order to investigate potential group differences on each condition individually, and to examine possible dissociations, a further set of four independent two-factor ANOVAs (investigating effects of age and group) were performed on each condition.

a) Normal focus condition

Effects of group and age were significant ($F_{1,23}=14.39$, p<0.001 and $F_{1,23}=8.11$, p<0.01), with lower accuracy in dyslexic and younger groups. There was also a significant group-by-age interaction effect ($F_{1,23}=4.58$, p<0.05), which showed that the controls improved their accuracy with age, whereas the children with dyslexia did not.

b) Normal shift condition

The effect of group was significant ($F_{1,23}=11.47$, p<0.005), with lower accuracy in dyslexic groups. There was no significant age effect or group-by-age interaction ($F_{1,23}=2.45$ and $F_{1,23}=0.12$ respectively).

c) Degraded focus condition

Effects of group and age were significant ($F_{1,23}$ =4.30, p<0.05 and $F_{1,23}$ =6.45, p<0.05), with lower accuracy in dyslexic and younger groups. There was no significant group-by-age interaction effect ($F_{1,23}$ =0.72).

d) Degraded shift condition

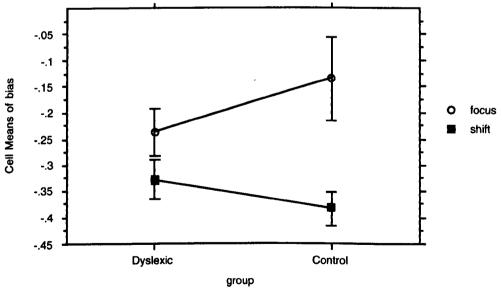
Effects of group and age were significant ($F_{1,23}=13.76$ p<0.005 and $F_{1,23}=16.25$, p<0.0005), with lower accuracy in dyslexic and younger groups. There was no significant group-by-age interaction effect ($F_{1,23}=0.06$).

ii) Bias

The bias measure (b) from signal detection theory³ was used as the dependent variable in a four-factor ANOVA investigating effects of group, age, visibility and condition.

Overall, the means indicated that participants were more likely to press the mouse to a non-target [FA] than not press the mouse to a target [miss] (indicated by negative values): i.e. they had a lower response criterion. Visibility had no effect on bias ($F_{1,23}=0.50$). Condition did have a significant effect, however ($F_{1,23}=34.45$, p<0.0001), with a lower response criterion in shift conditions. Younger participants were also more likely to respond than older participants ($F_{1,23}=5.22$, p<0.05), but there was no significant group effect ($F_{1,23}=0.41$).

Figure 5.2.4. Plot showing the interaction between condition and group on bias. It can be seen that the groups are about equally biased toward responding overall, but that the control groups have a lower response criterion (more bias towards responding) than the dyslexic groups in shift conditions and a higher criterion (less bias towards responding) in focus conditions. Standard error bars are shown.



³b=(Z(yes/signal)+Z(yes/non-signal))/2

A significant group-by-condition interaction also emerged ($F_{1,23}=11.14$, p<0.005), showing that control children were more likely to respond during shift conditions and less during focus conditions, whereas the difference for children with dyslexia was less (see Figure 5.2.4)⁴. Similarly, an age-by-condition interaction showed that the older participants had less bias overall (and a higher response criterion) and in particular a higher response criterion under focus attention conditions ($F_{1,23}=6.88$, p<0.05). The 3-way interaction between condition, group and age was also significant ($F_{1,23}=18.55$, p<0.0005), showing that the older dyslexic group the smallest.

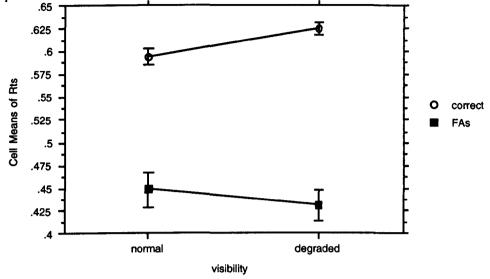
iii) Reaction times

A five-factor ANOVA was performed investigating the effect of group, age, response type (correct hit/ FA), condition and visibility on RT (see Appendix 5.2.3 for full results).

Mean RTs for control children were slightly faster than those for dyslexic children (see Table 5.2.2). However, main effects of group and age were not significant ($F_{1,23}=2.08$ and $F_{1,23}=0.45$). The effect of visibility was not significant ($F_{1,23}=0.15$). Effects of response type and condition were highly significant ($F_{1,23}=144.16$, p<0.0001 and $F_{1,23}=54.88$, p<0.0001 respectively), with longer latencies for correct hits than for FAs and also longer latencies for shift conditions than focus conditions.

⁴However, a breakdown of the means showed a more complex pattern. Dyslexic children had similar response criteria to controls on degraded focus and shift conditions, but lower criteria than controls on normal focus and higher criteria on normal shift conditions. An alternative (perhaps clearer) description of this pattern is that on degraded conditions, the group with dyslexia had a large differential between focus and shift conditions in one direction and on the normal visibility conditions they also had a large differential, but in the other direction. Although these effects are not significant, they appear to reflect the specific difficulty of the children with dyslexia in recognising particular shapes rather than real differences in bias. The increased bias on the normal focus condition was caused by frequently hitting diamonds instead of triangles. Similarly difficulty on the shift degraded condition can be accounted for by hitting triangles instead of diamonds: see section (iv) below.

Figure 5.2.5. Plot showing the interaction between visibility and response type on reaction times. It can be seen that in degraded conditions, correct response times tend to be longer and FA responses shorter. Standard error bars shown.



In addition to the significant main effects, there were two significant interactions. A response-by-visibility interaction ($F_{1,23}$ =8.20, p<0.01) showed that correct hits took longer in the degraded conditions and the FAs tended to be quicker, whereas in normal conditions the differences between FA and correct reactions times was less (see Figure 5.2.5). A response-by-condition interaction also emerged ($F_{1,23}$ =88.62, p<0.0001) showing that FA responses tended to be very fast overall, but particularly in focus conditions, whereas correct responses took approximately the same time regardless of condition.

Separate ANOVAs for correct responses and false alarms were also conducted. For the correct RTs, the effect of visibility was highly significant ($F_{1,23}$ =15.93, p<0.001). A condition-by-group-by age interaction ($F_{1,23}$ =6.12, p<0.05) showed generally slower responses in the focus condition, except for the older controls. No other effects were significant. For the false alarm RTs, only the main effect of condition was significant ($F_{1,23}$ =78.59, p<0.0001), with faster RTs in the focus conditions.

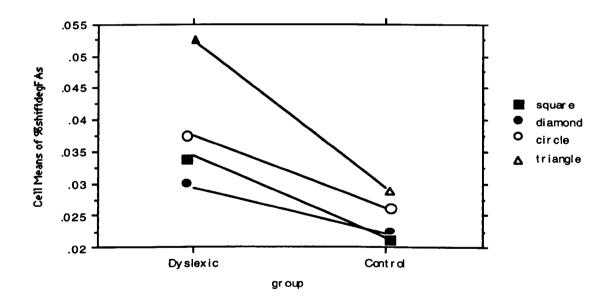
iv) Post-hoc analyses of errors made

If, in shift attention conditions, mistakes mostly occurred through difficulty remembering the current target, one would expect the FA rate to be highest for the two potential targets. Three factor ANOVAs were performed on each condition independently (focus and shift, normal and degraded) to examine the effects of

age, shape and group on the number of FAs (in terms of percentage of each shape occurring).

The effect of shape was significant in all conditions ($F_{2,46}=37.26$, p<0.0001 for normal focus; $F_{3,69}=5.01$, p<0.005 for normal shift; $F_{2,46}=30.00$, p<0.0001 for degraded focus; $F_{3,69}=4.94$, p<0.005 for degraded shift), showing that the pattern of mistakes was not random. Effects of group were also significant or showed trends towards the dyslexic groups making more FAs ($F_{1,23}=7.55$, p<0.05 for normal focus; $F_{1,23}=18.70$, p<0.0005 for normal shift; $F_{1,23}=5.03$, p<0.05 for degraded focus; $F_{1,23}=9.46$, p<0.01 for degraded shift). There were no significant age effects or interactions.

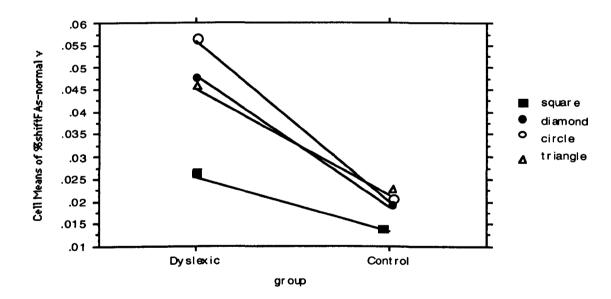
Figure 5.2.6. False alarm responses made in the degraded shift condition (where potential targets are square and diamond). It can be seen that FA responses in this case are not due to forgetting the current target, with most FAs being made to the triangle which is never a target. The second highest number of FAs were to circle, which somewhat resembled the diamond in its degraded state.



Plots of the FAs made in each condition and for each group were constructed (see Figures 5.2.6 and 5.2.7 for plots of the two shift conditions). These revealed that although the effect of shape was significant in all conditions, potential targets did not, in all cases, produce the highest number of FAs in shift conditions. For

example, Figure 5.2.6 plots the number of false alarms made by dyslexic and control groups in the degraded shift attention condition, where the potential targets were square and diamond. It can be seen that the highest number of FAs (in both groups) were made to triangle and circle. Figure 5.2.7 illustrates a similar pattern for the normal shift condition. Note that where potential targets are triangle and circle, the highest number of FAs were made to diamonds and circles. Data from the normal focus condition showed similar confusion between diamond and triangle in both groups. The degraded focus condition showed confusion between diamond and square, particularly for dyslexic groups. No group-by-shape interactions were significant, both groups tending to confuse the same shapes, although Figures 5.2.6 and 5.2.7 are typical of the pattern of all the plots in that differences between the shapes were far more pronounced in the dyslexic group.

Figure 5.2.7. False alarm responses made in the normal visibility shift condition (where potential targets are triangles and circles). Again, it can be seen that FA responses are not due entirely to forgetting the current target, with many FAs being made to the diamond.



Discussion

The main results were therefore consistent with the hypothesis of an automatisation deficit in the children with dyslexia. A group-by-visibility interaction was obtained in which it was the control children, rather than the children with dyslexia, who were disproportionately affected by the visual degradation of the stimuli. The main conclusions that can be made from the reaction time data are that correct responses generally take longer than FAs, a difference accentuated under degraded

conditions. There were no significant group effects, or interactions, for the reaction time data, suggesting that these patterns were the same for all groups. The dissociation between performance on focus and shift attention conditions was not replicated in this experiment (dyslexic performance this time being lower over all conditions) and the performance of both groups was lower in the present experiment than in the first. However, in terms of the concept of resource limitations being manipulated, it was reassuring to see that degrading the stimuli only had a significant effect in the (already more resource consuming) shift attention conditions, and that the effect of condition (focus or shift) only had a significant effect in the (already more resource consuming) degraded conditions.

Despite difficulty in interpreting some of the results, the result attesting to the original hypotheses is clear. The suggestion that increasing the attentional load necessary to perform a task will disproportionately affect children with dyslexia (a general resources deficit) can be rejected. The suggestion of a shape recognition automatisation deficit (from the DAD) in children with dyslexia has been supported. The degradation of stimuli made relatively little difference to the children with dyslexia, suggesting that they were performing the task with some difficulty already.

In addition to the convincing evidence from the group-by-visibility interaction found in this experiment, anecdotal evidence also arose to suggest that shape recognition may not be automatic in dyslexic children. On asking one of the participants after the experiment how he found it, he described how he "kept getting the four-sided one mixed up with the triangle". When asked "you mean the square?", he replied, "No, the four sided one on its side" (i.e. the diamond). This inability to name the diamond had persisted despite the name of the diamond being displayed several times throughout the experiment. This could be interpreted as either a reluctance and inability to label (consistent with phonological deficit theories) or, in terms of Shiffrin and Schneider's (1977) framework, that the patterns of features obviously being processed and considered were not being automatically converted into shape names, in a similar way to semantically related concepts (e.g. nurse and doctor) not priming each other (e.g. Hartley et al, in preparation). In fact, these two possibilities could be one and the same and could account for many of the difficulties of dyslexic children on this task⁵.

Thus, the dissociation between focus and shift conditions found in Chapter 4 can be explained by children with dyslexia having fewer *remaining* resources to cope in the shift attention condition, since the shape recognition component of both conditions is not automatic. The difference (n.s.) between the two focus conditions in Chapter 4 can be accounted for in the same way, since the second focus condition could be performed on the basis of colour alone. Shiffrin and Schneider (1977) suggest that recognition of degraded stimuli does require 'effortful', capacity loading, sequential processing of multiple individual target features. However, if a shape recognition automaticity deficit alone were the reason for reduced performance of dyslexic groups on normal visibility conditions, control group performance under degraded conditions might be expected to be further lowered to the level of the dyslexic groups (which it is not). This anomaly can be accounted for by considering other aspects of the tasks which are likely to need controlled processing in the children with dyslexia, such as decision making, attention shifting (in the shift condition) and responding. These are all factors which were not altered in the same way as the shape recognition component. although factors which could be manipulated in further research. A further minor anomaly is the equal performance decrement of both groups over the two halves of the shift attention condition in Chapter 4. The DAD might be expected to predict that a failure to automatise part of the task would lead to faster tiring and therefore a greater fatigue effect in children with dyslexia. Anecdotal evidence has suggested that dyslexic children do tend to become tired more easily in many tasks. However, faster tiring is not a central tenet of the DAD. Moreover, if the task was entirely automatic in control children, they would not be expected to produce a fatigue effect at all. It is possible, therefore, that any effects of the shape recognition component in the shift attention condition in Chapter 4 were subsumed by the more onerous task of shifting attention.

⁵An easy way of testing the idea of shape recognition difficulties would be to use novel and perhaps 'unnameable' shapes and squiggles. Assuming the above explanation of the results obtained here is valid, one might predict more equal performance of both groups on such a condition. However, this test would not discriminate between labelling/ phonological and recognition automaticity hypotheses since a squiggle is likely to be both unknown/ unlearned and difficult to label. Such an task would also be subject to strategic factors.

	<u>E</u>	xperiment	Experiment 2		
	focus 1	shift	focus 2	normal focus	normal shift
D14/15	4.1 (72%)	3.7 (74%)	5.3 (82%)	2.9 (85%)	2.6 (83%)
D18/19	4.5 (87%)	4.0 (84%)	5.6 (91%)	3.0 (88%)	2.9 (88%)
C14/15	5.5 (81%)	3.9 (81%)	6.0 (85%)	3.3 (89%)	3.3 (89%)
C18/19	6.4 (93%)	5.4 (91%)	6.0 (91%)	4.6 (98%)	3.7 (91%)

Table 5.2.3. A comparison of mean d prime values and percentage hits across the two experiments for the four groups

The DAD therefore accounts for both the group-by-condition interaction and the original focus-shift dissociation well. An inability to automatise the shape recognition component of the task is also sufficient on its own to explain the observed group difference in focus conditions in the present experiment. However, such a difference was not found in the first experiment and this inconsistency between studies also needs to be (and can be) explained. Table 5.2.3 shows each group's performance in normal visibility conditions in both experiments. It can be seen that performance generally was lower in the second experiment⁶.

In terms of necessary attentional resources, the performance decrease in the present experiment could be explained by the increase in target probability (0.2, rather than 0.15 as before: ISI's and actual speed of presentation remained the same). The probability increase was originally instigated in order to make conditions shorter (whilst still obtaining a reasonable amount of data), thereby avoiding fatigue and boredom effects. However, it is possible that the pace of this experiment appeared faster, thereby decreasing performance anyway. Posner (1978, p145) postulates that whereas a slow event rate may lead to poorer motor preparation and alertness, a fast event rate leads to declines in general (or state) arousal, whether participants are responding to most stimuli or not.

Van der Meere, Vreeling and Sergeant (1992) suggest that manipulation of event rate induces a type of strategy effect, in that response times are faster when the event rate is faster. They found that hyperactive children, "need a rapid presentation rate before they act like controls" (i.e. respond as fast as controls) and

⁶It should be noted that although some of the participants used in both experiments were the same, the results are not necessarily directly comparable since the experiment was performed nearly one year later and several new participants were used and old ones lost. The mean ages in the two experiments are also different.

that learning disabled children's performance was slower than that of the controls only in a slow event condition. In addition, Parasuraman (1979) found that vigilance decrements in sensitivity occur in successive-discrimination tasks (such as this one) when the event rate is high, whereas in low event tasks, sensitivity remains stable, but bias towards responding changes. Parasuraman (1985, p495) suggests that in high event tasks, "sensitivity may fall because the limited capacity attentional system...cannot devote resources consistently to target discrimination for a prolonged period". Similarly, Nuechterlein, Parasuraman and Jiang (1983) found rapid sensitivity decrements under conditions of high degradation of images. Such results suggested "a much closer link between selective attention and sustained attention...than previously supposed" (Parasuraman, 1985, p508). Hence, the probability of a target occurring may have increased the resource input necessary to perform the task (in a similar way to that hypothesised in the original shift attention task) even without image degradation. An increase in target probability can therefore account for the difference between groups in a way consistent with the hypothesis of an automatisation deficit in the shape recognition component of the task (leading to fewer remaining resources to cope with the apparently faster pace). In essence, it is possible that the attempt to avoid fatigue effects by making the conditions shorter, was negated by the apparent increase in pace instigated in order to enable this!

It seems therefore that the results obtained in this experiment, and that reported in Chapter 4, reflect an inability to automatise shape recognition or labelling in dyslexic children. Other possibilities can be entertained, but fail to account for the same wide range of findings as the DAD. There are five further possibilities which can each account for some, but not all, of the data: (1) a general shape recognition deficit, (2) a biased analysis method, (3) impulsivity in dyslexic children, (4) an attentional deficit and (5) that contrary to intentions data rather than resource limits were introduced by degrading the stimuli. The six possibilities (in total) are not mutually exclusive and could contribute to differing extents.

Shape recognition difficulties?

The first alternative explanation of the pattern of results found in this experiment is that they were essentially an artefact of the particular shapes used. In addition to the change in probability of a target across the two experiments, and the reminder of the target following a FA, the few colour cues present in the first experiment had been removed and the types of shapes changed. Results from the present experiment indicate that the difference in accuracy (n.s.) between the two focus conditions in the experiment reported in Chapter 4 may have been indicative of a general shape recognition deficit in the children with dyslexia, rather than a specific difficulty with circle and oval (see Figures 4.4.1 and 4.4.2). Furthermore, the decrease in performance over the two experiments may reflect general shape recognition deficits rather than any problems of automatising shape recognition. In the present experiment, discriminating between the triangle and the diamond for children with dyslexia appeared to be even more difficult than between the circle and oval in the first experiment (Figure 4.4.1), possibly contributing to a significant group difference. However, as discussed above, the increased target rate is also likely to have played a significant role. Moreover, all children clearly could recognise the shapes under conditions of less pressure, as illustrated in the slowed practices. Thus a difficulty under only speeded conditions may well amount to the same as a shape recognition automatisation deficit. However, in degraded conditions, it was noted by some participants that the square appeared similar to the circle. Although this was, in part, the point of the manipulation, the idea was that it would take more effort to correctly identify stimuli, rather than to make it almost impossible for some (see also data- vs. resource-limits argument below).

A plot of the FA responses made to the different shapes in the shift degraded condition provided evidence of shape recognition difficulties in both groups⁷. Figure 5.2.6 shows that although the two potential targets were square and diamond (so that forgetting the current target would elicit more FAs on one or other of these stimuli) the highest number of FAs in both groups (but in particular in the dyslexic groups) were made to triangles and circles. The equivalent plot in the first experiment showed very few false alarms to the distractor shapes (circles and light blue squares) and most to the potential target stimuli (oval and dark blue square) for both groups. In the first experiment, this, together with the correlatory evidence from the DST and the equal performance to the controls at short inter-target intervals, led to the conclusion that there was some confusion regarding the current target for the groups with dyslexia. In contrast, in the normal visibility shift condition in the present experiment, where potential targets are triangles and circles (see Figure 5.2.7).

In summary therefore, evidence points towards shape recognition difficulties for many of the children with dyslexia, but is unclear when considering this experiment alone whether or not this reflects a difficulty in automatisation. For the

⁷Unusual pattern of bias (see results section) also support this notion

present experiment, it is possible that shape recognition difficulties are more severe for some shapes than others and the specific shapes used as targets in each condition have contributed to the pattern of results obtained (as opposed to an automaticity deficit). For example, the smaller difference between groups on the degraded focus condition may result not (or not only) from decreased (possibly non-automatic) shape processing abilities of the controls, but also relatively (although not absolutely) increased abilities of the children with dyslexia. Dyslexic children may have less difficulty recognising a square than a triangle or a diamond. for example, thereby nullifying any effects of visual degradation. A pure difficulty with shape recognition is also able to explain the differences between the two focus conditions in Chapter 4 as well as the lower performance in all conditions of the present experiment (no colour cues for any shapes). It can account too for the lower performance of the children with dyslexia on both visibility conditions. However, importantly, this explanation fails to account for the original focus-shift dissociation found, since a pure shape recognition deficit would suggest that focus condition 1 should have been the most difficult for the dyslexic children, rather than the shift condition. It also seems less likely that certain shapes would present so much greater difficulty than others (and these shapes were unintentionally but coincidentally used to create the observed group-by-visibility interaction) compared to the possibility of a shape recognition automaticity deficit (as originally hypothesised). However, this existence of this debate does suggest that it would have been better to counterbalance the use of different shapes in the various conditions.

Analysis method?

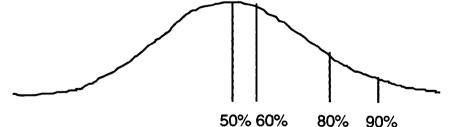
A further possibility for the pattern of results obtained is that the analysis method actually makes such a pattern likely. Since the d prime measure is based on a normal distribution, an already high level of performance (as in the control groups case) is easier to decrease than a lower level of performance, due to the bell-shaped curve (see Figure 5.2.8). This may also be reflected to some extent by the significant condition-by-group-by-age interaction found for accuracy. The older control group (highest accuracy) had the largest difference between focus and shift conditions.

It is interesting to note that if the probability of a correct hit is examined, rather than the d prime measure, then; the group difference is reduced to a trend (p<0.1), there is a significant age difference (p<0.05), the effect of visibility narrowly fails to reach significance and the only significant interaction is one of visibility-by-

condition. To some extent, this suggests that the degraded conditions only affect the probability of a hit (not considering FAs) in shift attention conditions. However, the equivalent analysis for FAs is rather more similar to the original d prime analysis, with large group differences, a strong effect of visibility and a significant group-by-visibility interaction (surprisingly, however, visibility had no effect on bias towards responding overall. All details are shown in Appendix 5.1.2).

A 'biased' analysis method is also unable to explain either the difference between the two focus conditions in Chapter 4, the focus-shift dissociation in Chapter 4, or the difference in level of performance between the two experiments. According to the reasoning, the dyslexic and control groups should be more likely to show a difference on the focus conditions (and in all conditions in the first compared to the second experiment) since performance is generally higher on them and therefore more easily reduced by seemingly small differences. Overall, therefore, the analysis method used is likely to be satisfactory and not a major contributory factor in the pattern of results obtained, since the pattern of FAs in the present experiment show the same interaction and since it is unable to account for results from Chapter 4.

Figure 5.2.8. A schematic diagram of a normal distribution if the mean proportion of hits is 50%. It can be seen that the distance between 50% and 60% of hits on the curve is smaller than the distance between 80% and 90%. In signal detection theory therefore, which is based on a normal distribution, a decrease in performance at a high level of accuracy has a more dramatic effect in terms of the lowering of the normalised Z score than the same decrease within already poor performance.



Bias and impulsivity?

Perhaps then it is plausible that it is the high number of FAs which cause group differences, and that FAs can account for the unexpected group-by-visibility interaction. Dyslexic groups did make an increased number of FAs in Chapter 4,

and the alternative analysis method (see above) suggested that FAs characterised the data with greater accuracy than did correct hits. Degradation of stimuli is not likely to increase (and may even decrease) impulsivity, even if it is likely to increase other types of FAs. It may therefore be possible that degradation decreases the impulsivity element of FAs in dyslexic children, but increases the difficulty of the shape recognition element for both groups, leading to the pattern of results obtained. However, the FA plots in Chapter 4 suggested that the increased number of FAs did not represent increased impulsivity, FAs being made principally to the two potential targets. Moreover, lack of an overall group difference in bias may be taken as evidence that dyslexic children do not suffer from excessive impulsivity. If children with dyslexia were particularly impulsive, they might be expected to show an increased bias towards responding overall. Lower response criterion (i.e. higher bias towards responding) has been found previously on the Continuous Performance Test for hyperactive children (e.g. Nuechterlein, 1982).

An impulsivity explanation is also unable to account for differences between the two focus conditions in Chapter 4 and the lower performance in Chapter 5. However, it can attempt to account for the focus-shift dissociation found in Chapter 4 by using the concept of activation levels already discussed in Chapter 4. Briefly, this idea suggested that stimuli to be remembered become activated in memory, with activation on a continuous scale (cf. Anderson, 1983). It was originally proposed as an explanation for the apparent confusion regarding the current target in the shift condition (activation of the current target decays quicker in dyslexic children so that the differential in activation between the two potential targets is small, yet absolute activation level is larger than that of the other stimuli). If this were the case, and if response criterion was responsive to these activation levels, then the greater activation levels of two targets, rather than one, might be expected to cause an increase in the number of FAs. This whole explanation, however, is highly speculative. The argument that explanation of greater impulsivity in children with dyslexia provides for the group-by-visibility interaction in the present experiment is only really slightly more convincing.

Not paying attention?

The most straight-forward and parsimonious explanation of the lower performance of the children with dyslexia on all conditions in this experiment and of the groupby-visibility interaction is that the dyslexic children are less attentive. It suggests that poorer performance than controls overall, coupled with less decline under degraded conditions, is far more easily interpreted as a result of dyslexic children not paying attention than it is as a 'shape recognition automaticity deficit' or similar. If dyslexic children were paying very little attention to any of the conditions, then their result might represent a type of floor effect. In short, degraded conditions would not be expected to reduce performance on a non-attended task!

Although the explanation of poor attention in children with dyslexia could account for a great deal in the context of this experiment alone, performance of all groups was actually very good, with a high percentage of hits and a low percentage of false alarms (see Table 5.2.3). Furthermore, examination of individual case studies and a consideration of both experiments together present strong evidence against this hypothesis, supporting instead the idea of shape recognition difficulties of some kind. Two of the more severely impaired dyslexic children in terms of their literacy skills (RH and JM), showed particularly revealing results. The results of RH and JM were typical of their group in many ways, although in perhaps a more exaggerated manner. The results of all 18 participants who took part in all the attentional experiments are shown in Appendix 7.1.

RH obtained a very high d prime score of 6.0 in the second focus condition in Experiment 1 compared to a respectable 3.5 in the first focus condition and 2.2 in the shift condition. It is therefore particularly striking that the low performance of RH in the shift attention condition was neither result of motivational differences, nor ability to selectively attend to a target stimulus. In focus condition two, where the target was the dark blue square (recognisable on the basis of colour alone), RH's accuracy was high. It is suggested that the increased difficulty of shape recognition decreased his accuracy in focus condition one, and having to shift his attention between two potential targets decreased his accuracy further. However, in the second experiment, similar to the majority of participants, his accuracy in the normal visibility conditions was lower (possibly due to a combination of different shapes used, an increase in apparent speed and lack of colour cues), and the difference between focus and shift conditions reduced (3.7 vs. 2.7). Evidence from RH therefore is supportive of a difficulty with shape recognition.

JM showed a similar pattern to RH, although within a profile of much lower performance overall. On the first experiment, JM obtained scores of 1.5 and 4.1 on focus conditions one and two respectively and 1.4 on the shift condition. Therefore JM could focus his attention reasonably well (as shown by his performance in focus condition two which can be performed on the basis of colour alone), although again difficulties with shape recognition decreased his performance. Surprising, and apparently unique to this group, was his improved performance in the second experiment for normal visibility conditions, obtaining accuracy of 2.8 and 2.5 on focus and shift conditions respectively. There are several potential explanations for this. Objectively, it could be an age related improvement as much as anything, or perhaps an artefact of the different analysis methods: In the first experiment, individual d prime values were calculated for the different time periods and these were averaged to obtained mean accuracy. In the second experiment, only an overall d prime value was calculated⁸. It may also be interesting to note that JM had the highest score for clinical diagnosis of ADHD (although did not display enough markers for diagnosis) and showed some of the most severe deficits on numerous measures from the DST, with particularly impaired backwards digit span, bead threading, segmentation, reading and spelling. JM's difficulties therefore appear to reflect shape recognition difficulties too (as well as a possible transient system deficit: see extended footnote 8). However, subjectively, it has

⁸This difference in methodology may have been expected to increase JM's performance in the second experiment to some extent, because of his unique pattern of behaviour. In the first experiment JM obtained a d prime value of zero in both focus conditions in the most rapid attention shifting category, initially suggesting some difficulty rapidly re-engaging his attention in the task following a successful response. However, in contrast, he had higher accuracy for the rapid shifting attention condition than for the mean shifting measurement (as used for correlations with DST subtests). This pattern is as predicted by the verbal labelling deficit explanation offered for the original group dissociation overall. JM showed a similar pattern of performance (although accuracy was higher) in the second experiment, with a dissociation between rapid focus and rapid shift conditions. The pattern of results may point towards evidence of a transient system deficit. It is possible that the difficulty with rapid reoccurrences of the same target in the focus conditions, was that he was unaware that two targets had been presented. This notion may be worth pursuing further (for JM). In a previous study (Moores, 1995), JM showed that he could discriminate between which of two shapes appeared on the screen first when presented side by side in rapid succession, although he had more difficulty with making a distinction between the order of two rapidly presented sounds. However, this study also found a significant correlation between auditory temporal processing accuracy and IQ (r = 0.65, p<0.01), and since JM has one of the lower IOs of his group, a lower auditory temporal processing score might be expected. This evidence, together with evidence from the attention experiment suggests that he can detect small differences in temporal presentation, although perhaps not if presentation is an identical stimulus in the same place. Such a deficit could severely impact on numerous aspects of reading and also other skills (see e.g. section 1.4 and Lovegrove, 1994). By contrast, RH showed good performance on the rapid condition of focus attention, but poor performance on the rapid shifting condition (although later mean performance did not improve substantially). Perhaps also of interest is JM's reading performance under coloured neon light, shown to help reading in poor readers in a small pilot study conducted by the author but not reported in this volume. Speed and accuracy of reading were compared under white 'placebo' light and under pink light, found (on average) to help poor readers (although results were variable with some children showing more dramatic results than others). This pink light, however, did not help JM, his performance actually deteriorating slightly. Although pink is the colour presumed by the manufacturers to help most readers, however, the transient system deficit theory would in fact predict blue to be of more benefit and red to be detrimental. A blue light was available for the smaller percentage of readers who were not helped by pink, but unfortunately only white and pink were used at the initial stage of the experiment in order to enable a well counterbalanced design. It is now planned to test JM on the blue light at some later date and compare this performance with that on the white. A transient system deficit theory might predict that JM (at least) would be helped by a blue light.

also been noted that JM typically takes a long time to 'settle in' to an experiment, often starting with poor performance and then improving. It is therefore possible that the since the second experiment was similar to the first, his typically poor performance whilst 'settling in' did not impact on these results.

Evidence from case studies therefore suggests a specific, rather than a general, problem. In addition, this explanation is unable to account for focus-shift dissociations, differences between different focus conditions or performance differences across experiments.

Resource- vs. data-limits?

A final possibility to account for the results is that the degradation of stimuli made the task 'data-limited' as opposed to 'resource-limited'. This distinction between a data vs. resource limit explanation and a controlled vs. automatic processing explanation is a fine one. A task is said to be resource-limited if a greater input of resources *could* lead to improved performance. A task is data-limited if only an increase in the quality of the data would increase performance (because all possible processing has been done). Data limits can apply to either the poor quality of the signal, or the poor quality of the representation of the signal in memory (Norman and Bobrow, 1975). Automatic processes could therefore be said to be data-limited (because all possible processing has been done) whereas controlled processes are resource-limited (because more processing could be done, so processing has not occurred automatically: an increase in resources would be helpful).

Navon (1984) offers a powerful critique of resource theory overall, and, in particular, circularity arising from measuring the resources required by a given task via the performance level achieved (often when performed with a second concurrent task). Navon also criticises (albeit rather lovingly!) the theory as unfalsifiable and lists the 'data-limits' explanation as just one of its 'built-in escapes'. To a considerable extent, the resource vs. data limits argument has been subsumed by the one of automatic vs. controlled processing (e.g. Shiffrin and Schneider, 1977). However, in terms of the present experiment and the DAD, there may be subtle but important differences which will be discussed following an overview of the work in this area and its implications for the present experiment.

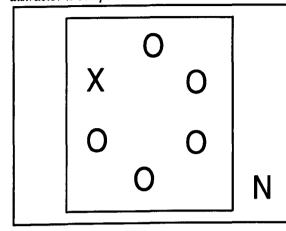
Lavie's (1996) work is particularly relevant in terms of the present experiment, since it suggests that degradation of stimuli does not increase attention load at all, but merely delays processing. Lavie's theory of the role of perceptual load in

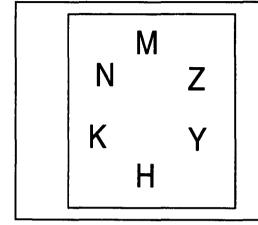
selective attention basically states that early selection of material for processing occurs only when attentional load is high, whereas if attentional load is low, distracting and irrelevant information will be processed (and therefore selection is late).

Lavie has used variations of the response competition paradigm (see e.g. Eriksen and Eriksen, 1974) in order to investigate her theory. In a visual search task, for example, participants had to respond according to which of two possible targets was present in a central display (see Figure 5.2.9). Lavie (1995 and personal communications 1997 & 1998) varied attentional load by increasing the similarity of distractors to targets (e.g. low load would be finding 'X' or 'N' amongst O's, high load would be finding 'X' or 'N' amongst 'K', 'M', 'Z', 'H' and so on⁹). An irrelevant distractor outside the main display could be either compatible or incompatible (e.g. X if target was X, or N if target was X). Lavie found that under conditions of high load, the irrelevant distractor had less effect on reaction time than it did under conditions of low load. Lavie suggested that under conditions of low load [automatic processing (or possibly 'pop-out'; Treisman 1991)] the distractor was automatically and involuntarily processed, whereas under conditions of high load [controlled (or serial) processing] there were no resources left in order to process the distractor. However, since reaction times were longer under conditions of high load, it was considered that this extra time may have removed the distractor's deleterious effects. Physical degradation of stimuli was therefore subsequently employed as a control for general task difficulty and applied to a low load condition (Lavie, 1996). Lavie considered this degradation to delay processing without increasing attention load. By finding large effects of the distractor under conditions of visual degradation, she illustrated that it is not the increased reaction time which removes distractor effects, since increased reaction time was necessary to process the degraded stimuli. Moreover, Lavie showed that (at least in terms of her theory), degradation of stimuli did not increase attentional load in that paradigm since irrelevant distractors were still 'automatically' processed: thus a degraded low load condition was still a data-limited process. whereas the high load condition was a resource-limited (and controlled) process (and hence the distractor was not processed).

⁹The latter being more difficult because it is essentially a combination search (see e.g. Treisman, 1991) of finding straight lines amongst straight lines.

Figure 5.2.9. An illustration of Lavie's response competition paradigm where the two potential targets are X and N. The left box illustrates an example of a low load condition where the target is X and easily found amongst the O's. The irrelevant distractor (N) is incompatible. The right hand box illustrates the equivalent high load condition, where the target is N and the irrelevant distractor is compatible.





Thus, one possible alternative way of viewing the present hypothesis in terms of a resource deficit is that task difficulty and necessary processing was increased by degradation, but attentional 'load', as such, was not. Although the decreased performance in control groups on the degraded condition suggests that the degraded condition was more difficult (at least for control groups), it is possible that the extra processing time needed for the degraded condition was not available due to the presentation speed. It may have been this lack of time, rather than any resources deficit, which reduced control group performance, thus a data-limited process. Meanwhile, the dyslexic group may well have continued to operate within a 'resource-limited' area, thereby maintaining their usual performance.

This explanation therefore uses one of resource theory's 'built-in escapes' (Navon, 1984); namely the data-limits argument. However, although this potential criticism makes the explanation less satisfactory, it is no less valid. More troublesome is that, as already mentioned, Shiffrin and Schneider (1977) suggest that recognition of degraded stimuli *does* require 'effortful', capacity loading sequential processing of multiple individual target features. Furthermore, if both data- and resource-limited processes can have longer latencies and if data-limited processes can also have higher error rates under visually degraded conditions, then the seemingly impossible question arises of where any 'resource-limit' ends and any 'data-limit'

starts¹⁰. Moreover, this explanation alone cannot account for the original focusshift dissociation, the lower performance in the present experiment in comparison with the first or differences between the two focus conditions in the first. Furthermore, Lavie used a different paradigm in her experiments which may not be generalisable to this one. In addition, the weight of the evidence seems to suggest that degradation of stimuli *does* prevent automatic processing, creating a resourcerather than a data-limited situation¹¹.

Summary

There are therefore several possible (non-exclusive) explanations of the data found in this experiment. However, consideration of the totality of the evidence points strongly towards a shape recognition automaticity deficit in dyslexia. Table 5.2.4 presents a summary of the main evidence.

¹⁰Norman and Bobrow (1975) make some attempt at resolving this issue by suggesting that in resource-limited processes, there is a speed-accuracy trade off, whereas in data-limited processes, there is usually an inverse relationship between speed and accuracy. It may therefore be of interest to note that correlational analyses of speed (correct RT only) and accuracy (mean d prime values) were performed (see Appendix 7.1) for both this experiment and the one in Chapter 4. In each case, a significant negative relationship between speed and accuracy (faster responses being more accurate) suggested data-limited processes. It may also be worth noting at this point that another difference between the present experiment and that in Chapter 4 is the administration of the SART (see Chapter 6) five minutes before this experiment. The SART is a difficult, demanding task which may have caused fatigue in some participants. Furthermore, since according to Norman and Bobrow's (1975) reasoning this was apparently a resource-limited task (with a significant speed-accuracy trade-off), and one on which the dyslexic groups made more mistakes, its administration may have conceivably had some effect on results presented here. However, I believe that this to be unlikely because of both the short length of this experiment and informal observation of participants perform all the tasks. ¹¹However, if Lavie's reasoning is correct, the results obtained in the present experiment are

¹¹However, if Lavie's reasoning is correct, the results obtained in the present experiment are interesting and offer further interesting opportunities to re-examine the ability of children with dyslexia to perform under conditions of high attentional load and perhaps also compare these abilities with performance under conditions of high task difficulty. A dissociation would be useful for dyslexia research generally in order to work towards a more fully specified theory.

Table 5.2.4. A summary table of the ability of the six discussed explanations to account for the data found in Chapters 4 and 5.

Sesa Sesa	Group-by- visibility interaction in Chapter 5	Focus-shift dissociation in Chapter 4	Lower performance in Chapter 5 (cf. Ch.4)	Smaller group difference in focus 2 than focus 1 (Ch.4)
Rennentrer	~	Less resources remaining	~	V
DAD Shape automaticity deficit	than dyslexic		Increased probability of potential targets makes all conditions more resource consuming. Leads to group difference in focus conditions	Focus condition 2 can be performed by colour recognition alone so is easier for dyslexic children
Resources			X Can't explain	
Data- limits	Control performance automatic in degraded conditions but reduced by poor quality of data	X Can't explain	data is poorer quality	possibly by suggesting that in the different conditions e recognition deficit
Analy- sis	Interaction artefact of analysis method	experiment should be les differences because perfor	X onditions and Chapter 5 as likely to produce group rmance is lower (nearer the the curve)	X Can't explain
Atten- tion	Interaction is a floor effect	X If dyslexic children not paying attention they should be lower on focus conditions too.	X	Dyslexic children should be significantly lower on all conditions. Expect no differences
Shape recog-	Interaction artefact of shapes used	X Shift condition should be easier than focus 1 (at least half the targets have	More difficult shapes in Chapter 5 experiment. No colour cues.	Can do focus 2 by using colour alone. Focus 1 requires shape recognition.
nition	u drahman mud	colour cue)	tion among appl	CONTROL OF
Impul- sivity	vish More impulsive if shapes clear. BUT: dyslexic children do not have a lower response criterion	Lish More impulsive when two potential targets already in mind?	X Can't explain	X Can't explain

Chapter 6

Performance of children with dyslexia on the Sustained Attention to Response Task

Another look at sustained attention in dyslexic children

Summary:

In Chapter 4, the link between dyslexia and attention was investigated and a dissociation between focus and shift attention conditions was found in children with dyslexia. This evidence, together with evidence from Chapter 5, suggested possible involvement of a shape recognition automaticity deficit. Analyses were also performed in order to investigate the (post-hoc) hypothesis of a sustained attention deficit for the children with dyslexia in the shift attention condition in Chapter 4, but no evidence was found. However, it is possible that dyslexia is more strongly associated with shorter periods of attentional lapse than a difficulty sustaining attention over a long period of time. The Sustained Attention to Response Task (SART: Robertson, Manly, Andrade, Baddeley and Yiend, 1997) was designed as a measure of sustained attention. It requires the withholding of responses to rare (one in nine) targets over a period of under five minutes and has been found to be sensitive to attentional lapse in traumatically brain injured patients. In order to further investigate the nature of the reported attentional disorder in dyslexia, this chapter reports an experiment which examined the performance of children with dyslexia and their controls on the SART. Consistent with the hypothesis of impaired ability to sustain attention. children with dyslexia made significantly more errors than controls. However, this group showed a qualitatively normal pattern of performance together with a marked speed-accuracy trade-off. Their responses were as fast as those of the controls. The differences in errors may reflect greater motivation combined with slower naming speed in the children with dyslexia, rather than a pure deficit of sustained attention.

6.1. Introduction

Three key aspects of attention include the abilities to focus, shift and sustain attention. Research presented in Chapter 4 suggested that these abilities may not be

equally impaired in children with dyslexia; they performed as well as controls on two focus attention conditions, but had significantly worse performance than controls on a shift attention condition. This dissociation was attributed to resource limitations of the children with dyslexia together with difficulty using a verbal labelling strategy (useful for rehearsing the current target in the shift condition)¹. A *rapid* attention shifting deficit, as hypothesised, was not found, and thus caution had to be exercised before making any firm conclusions regarding whether or not the dissociation reflected pure attentional difficulties in children with dyslexia. A crucial point is that none of the participants showed clinical evidence of ADHD.

Because of the possibility that the dissociation found between focus and shift attention conditions reflected only a difficulty for children with dyslexia sustaining their attention over the longer time period required by the shift condition, a 'splithalf' analysis of this condition was conducted. This showed that groups with dyslexia could sustain their attention over that condition (about 15 minutes) as well as control groups, in that performance in both groups suffered similarly in the second half. However, this evidence does not preclude the possibility of a sustained attention deficit in children with dyslexia. Recent neuroimaging evidence suggests that areas in the brain involved in sustained attention are active over periods as short as 40 seconds (Pardo, Fox and Raichle, 1991), rather than over tens of minutes as previously thought. It is therefore possible that local fluctuations and attentional lapses may characterise a deficit in sustained attention as validly as decreases in attention over long periods of time. An attentional lapse of this sort might therefore be able to explain the relatively good performance of children with dyslexia to controls at short intervals between targets, but poorer performance after some time, particularly if the task is reliant on continuing verbal rehearsal of the target. It could also account for reported difficulties of children with dyslexia 'keeping on track' (Augur, 1985).

The Sustained Attention to Response Task (SART) was originally designed to counteract the difficulties of finding a true measure of sustained attention. Traditionally, Continuous Performance Tests (e.g. Rosvold, Mirsky, Sarason, Bransome and Beck, 1956) are used for this purpose, where participants have to monitor long sequences of stimuli and respond to infrequent targets. However, these tasks fail to tap shorter 'lapses of attention' and also often have problems with ceiling effects, leading researchers either to perceptually degrade targets or to

¹The experiment reported in Chapter 5 aimed to further investigate the idea of a resource limitations deficit in children with dyslexia.

load working memory in order to reduce high levels of performance. Barkley (1996) reports an unpublished study by Conners, March, Fiore and Butcher (1994) which reversed the continuous performance test format (so that children respond to every trial but inhibit the response to the target) and a study by Schachar, Tannock and Logan (1993) using a stop-signal paradigm within a reaction time task to investigate impulsivity. Barkley concludes that, "early results suggest that such paradigms may be more sensitive to dose effects of stimulant medication on sustained attention and impulse control and to differences between children with ADHD and those without the disorder.". He also states that, "Continued exploration of creative variations on response formats with traditional tasks is to be encouraged...".

Fisk and Schneider (1981) make the distinction between automatic and controlled processing of stimuli (see also Schneider and Shiffrin, 1977). For tasks that have to be performed over relatively long periods, they show that performance decrements over time occur only when controlled processing is required and that "Maximum vigilance decrements occur when subjects must continually and redundantly allocate control-processing resources." (p737). Robertson et al (1997) argue that controlled processing would be taxed more heavily if the automatic response set could be transferred to the non-targets, so that controlled processing was necessary to cancel out the automatic response. Partly on the basis of this argument, the SART was developed, in which a response is required to every stimulus *except* for the targets, to which responses must be withheld. The task runs over a period of under five minutes. This also makes it a more clinically useful tool than continuous performance tests which usually last between 30 and 45 minutes. Forty five minutes is a long time to have to perform an extremely boring and apparently pointless task, which could potentially cause it to be stressful. Ballard (1996) investigates effects of stress and anxiety on performance on the continuous performance test (CPT), with fewer errors of omission by lowanxious participants. In addition, both post-test anxiety and anxiety change were both associated with the number of omission errors. She concludes that "performance on such tasks is clearly affected by a host of environmental and task parameters, as well as by a variety of individual subject differences, including anxiety." and that "Poor CPT scores by clinic patients suggest only a need for further evaluation of subject characteristics and environmental stressors that may be affecting performance." Although I would not wish to suggest that Ballard is suggesting that the length of such tasks is a major factor (or a factor at all), it is obviously something worthy of consideration for this reason and also for convenience of administration for both participant and experimenter.

Robertson et al (1997) found that the SART was more sensitive to everyday attentional failures and 'lapses' of attention (as measured by self report questionnaires) than were continuous performance tests in both control and traumatic brain injured (TBI) patients. Performance on the SART was not predicted by performance on tests presumed to be sensitive to response inhibition². In addition, Robertson et al found that TBI patients were less likely than controls to slow down after an error, and that all participants were more likely to have 'lapsed' into an automatic way of responding (shown by faster reaction times) before an error. Performance of either group did not deteriorate significantly with time on task, such that the authors conclude that "local fluctuations in attention or 'lapses' may provide a better account of poor performance on this task than a simple decrement over time." (p.755).

In an attempt to better characterise the attentional deficit associated with dyslexia, the present study assessed the performance of children with dyslexia and matched (for age and IQ) controls on the SART. Given the reports that children with dyslexia have difficulty 'keeping on track' (Augur, 1985), it was predicted that they would be more prone to attentional lapses than controls and therefore perform less well on the SART.

6.2 The SART experiment Method

Participants

The participants used in this experiment were the same as those used in Chapter 5 (and most had also taken part in experiments presented in Chapters 2 and 4).

Dyslexic participants had been diagnosed by a full psychometric assessment. They were of normal or above normal IQ [operationalised as IQ of 90 or more on the full scale WISC-III (Wechsler, 1976)] and without known primary emotional, behavioural or socio-economic problems. Each participant's reading age or spelling

²These included: the Stroop test (where colour words are printed in different coloured inks and the colour of the ink has to be read for each word as fast as possible, whilst ignoring the text of the word itself) and the Winconsin Card Sorting Test (in which cards have to be sorted according to particular criteria and when that criteria is arbitrarily changed, perseveration of responses is recorded). The validity of these as measures of response inhibition is debatable.

age was at least 18 months behind his or her chronological age. Two age groups were used with mean ages 14.6 and 19.0 years (D15 and D19).

Normally achieving control participants had also been given a short-form psychometric assessment and obtained normal or above normal IQ and reading and spelling ages in line with or above their chronological age. Two age groups were used, approximately matched for chronological age with the dyslexic groups (C15 and C19).

Participants had also all been assessed for clinical evidence of ADHD in accordance with the DSM IIIR (American Psychiatric Association, 1987). A score of at least 8 out of 14 markers of the disorder is required for clinical diagnosis. None of the participants showed evidence for ADHD. There were no significant differences between the score of the dyslexic and control groups. All participants were given £5 for their co-operation in this study and a study which followed this one (the experiment reported in Chapter 5). Psychometric data (means and ranges) for the four groups of participants are shown in Table 6.2.1.

Group	n	Mean Age	Mean IQ	Mean RA	Mean ADHD
D15	8	14.6(13.5-15.2)	113 (96-134)	13.4 (9.3-17) ³	1.0 (0-6)
C15	9	15.0 (13.8-16.0)	116 (101-129)	16.2 (14.0-17+)	0.1 (0-1)
D19	6	19.0 (17.3-20.9)	115 (101-131)	12.8 (9.3-16.0)	0.3 (0-1)
C19	5	18.8 (17.8-19.5)	114 (96-130)	17+	0.0

Table 6.2.1. Psychometric data for each group of participants

SART

The SART program was written by Robertson et al (1997). It presents a total of 225 digits (25 of each of nine digits) visually on the computer screen over a 4.3 minute period. Each digit is presented for 250 milliseconds followed by a 900 millisecond mask. Participants use their preferred hand to press the mouse button in response to every digit except the 'target' digit 3, for which they are instructed to

³As in Chapter 5, one participant's reading age from this group was lost (but was found in a subsequent experiment to have remained significantly lowered). Furthermore three participants had caught up with their reading since time of diagnosis, although their spelling ages remained significantly lowered.

withhold responses. The target digit is distributed throughout the trials in a prefixed pseudorandom order which is the same for all participants. To enhance the need for processing the numerical value of the number rather than looking for a set 'shape' the digits are presented in one of five randomly allocated font sizes (48, 72, 94, 100 and 120 point: symbol font): between 12 and 29mm. The mask is a circle with a cross in the middle (diameter 29mm). Digits and mask are presented centrally on the screen in white, against a black computer screen positioned in front of the participant. A practice is given before the main condition consisting of 18 presentations of digits, two of which are number 3.

Procedure

The method used was a replication of that used by Robertson et al (1997) using their SART program and run on an Apple Macintosh computer. Participants were instructed to press the computer's mouse button as quickly as possible when a number appeared on the screen, except when that number was a three. They were informed that in between each digit there would be a cross which they should ignore. They were also told that the digits would vary in size, but that they should ignore this. Participants were then reminded to press the mouse button for all numbers except three, to attempt to do the task quickly, but also to try not to make errors. A short 18 digit practice was given before the main condition which lasted under five minutes.

Results

The mean number of false alarms (FAs: non-withheld responses to 3) and mean reaction times (RTs) for the four groups are presented in Table 6.2.2.

	FAs (max. 25)	RTs to non-RTs to target		s Correct Hits	
		targets (ms)	(FAs)	(max 200)	
d15	5 14.8 (2.8)	326 (50)	278 (45)	189 (16)	
c15	10.3 (4.6)	373 (96)	282 (37)	196 (5)	
d19	13.7 (3.3)	334 (54)	295 (43)	197 (2)	
c19	11.0 (4.7)	297 (49)	253 (40)	198 (2)	

Table 6.2.2. Mean False Alarm (FA: non-withheld responses to 3's) and Reaction Time (RT: in milliseconds) data for the four groups (standard deviations shown in parentheses).

i) Errors made

Robertson et al suggested that FAs on the SART reflect attentional lapse. A twofactor ANOVA was used to investigate the effects of age and group on the number of FAs made. A main group effect was found ($F_{1,24}$ =5.48, p<0.05), with the children with dyslexia making more FAs (14.3 vs. 10.6). There were no age effects or group-by-age interactions ($F_{1,24}$ =0.02 and $F_{1,24}$ =0.33 respectively).

Misses to non-targets were also analysed in a two-factor ANOVA. No group or age effects were found ($F_{1,24}=0.86$ and $F_{1,24}=1.86$ respectively) and no groupby-age interactions ($F_{1,24}=0.69$). Performance on the non-targets was good (dyslexic groups: 3% misses, control groups: 2% misses). This is roughly equivalent to the percentages of false alarms made in conventional target detection tasks (e.g. see Tables 4.4.2 and 5.2.3). Informal observation suggested that misses occurred most frequently to the very first digit in the test and also as an 'aftermath' to false alarms (since participants were sometimes busy making various expressions of annoyance rather than immediately returning to the task). However, one participant (LF) in the D15 group missed a total of 47 (out of 200) non-targets. The reason for this is not clear, especially since he managed to 'hit' over half the targets (i.e. had a normal FA rate). A tally of how many digits of each type were missed did not help answer this question, with missed digits occurring for all the numbers. It was thought that there may have been some confusion between particular numbers, e.g. 3 and 8, but this was obviously not the case. Between group differences in number of FAs made remain significant with this participant excluded.

ii) Response latency and variability

Faster RTs may reflect a lesser degree of controlled attention and therefore more FAs. Two two-factor ANOVAs were used to investigate the effect of age and group on RTs of correct responses and FAs. Group and age had no effect on the correctly pressed RTs ($F_{1,24} = 0.01$ and $F_{1,24} = 2.46$ respectively), but there was a trend towards a group-by-age interaction ($F_{1,24} = 3.50$, p<0.1). Post-hoc comparisons (using Fisher's protected LSD test and considering all 4 groups individually) showed that the C19 group were significantly faster (p<0.05) than the C15 group. Other comparisons were not significant.

Similarly, group and age did not have a significant effect on RT of FAs ($F_{1,24} = 0.41$ and $F_{1,24} = 0.63$ respectively), although again there was a trend towards a group-by-age interaction ($F_{1,24} = 2.99$, p<0.1). The means showed that the D15 group were faster than the C15 group, but the D19 group were slower than the C19 group. No post-hoc comparisons were significant.

Robertson et al interpret a larger variability in RT (as assessed by the mean standard deviation for each group) in TBI patients as additional evidence that the SART reflects ability to maintain consistent performance. A two-factor ANOVA was performed in order to compare within-subject variability in RTs (to all stimuli) across factors of age and group. This used the standard deviation of the RTs for each individual as a dependent variable. The effect of group was not significant ($F_{1,24}=1.53$), but there was a main effect of age ($F_{1,24}=7.49$, p<0.05), with younger participants being more variable. There was no group-by-age interaction ($F_{1,24}=0.04$).

Table 6.2.3. Mean reaction times (in milliseconds) of the four responses either prior to or following either a false alarm to (FA) or a correctly withheld response (CW). Standard deviations are shown in parentheses.

	Reaction Times (msec)				
	pre- FA	post- FA	pre- CW	post- CW	
d15	303 (54)	322 (57)	340 (62)	332 (56)	
c15	336 (76)	344 (72)	397 (109)	383 (109)	
d19	318 (47)	351 (55)	342 (67)	322 (56)	
c19	259 (33)	267 (36)	290 (29)	277 (45)	

iii) Response style

Robertson et al found that TBI patients tended not to slow down following an FA, whereas controls did. The mean RTs for each of our groups, pre- and post-FA, are shown in Table 6.2.3.

The effect of a FA upon mean RT was examined in a three-factor ANOVA, investigating effects of age, group and time (with respect to a target digit: pre-/ post-FA) on the mean RT of the four responses either side of a FA⁴. There were no main group or age effects ($F_{1,24}=0.97$ and $F_{1,24}=1.48$ respectively), but a significant group-by-age interaction did emerge ($F_{1,24}=4.86$, p<0.05). The effect of time was highly significant ($F_{1,24}=14.40$, p<0.001), with post-FA responses being significantly slower than pre-FA responses. This main effect of time also interacted with group ($F_{1,24}=4.31$, p<0.05).

The interactions of time and age with group were further analysed using two-factor ANOVAs for the dyslexic and the control groups separately. These showed that time had a highly significant effect for the groups with dyslexia ($F_{1,12}=17.84$, p<0.005), who slowed down after a FA, but no effect in the control groups ($F_{1,12}=1.43$). Conversely, a main effect of age was found for the control groups ($F_{1,12}=4.81$, p<0.05), with the C19 group being faster overall, but not for the groups with dyslexia ($F_{1,12}=1.16$). There were no time-by-age interactions for either group ($F_{1,12}=0.00$ for the control groups, $F_{1,12}=0.62$ for the groups with dyslexia).

iv) Predicting errors

Robertson et al suggested that FAs could be predicted on the basis of pre-target digit RTs (the four responses before the target digit: 3). The mean RTs for each group [pre-FA and pre-CW (correctly withheld response)] are shown in Table 6.2.3.

A three-factor ANOVA investigated effects of age, group and response to the target digit (CW/ FA) on RTs before the target digit. A main effect of response was found ($F_{1,24}$ =16.90, p<0.0005) with pre-FA responses being faster than pre-CW responses, but there were no main group or age effects ($F_{1,24}$ =0.04 and $F_{1,24}$ =2.71 respectively). Again, a trend towards a group-by-age interaction was

⁴The mean RT was in the vast majority of cases the mean of four responses, although in certain cases either two targets were too close together or participants failed to make responses to non-target digits. In these cases the mean of fewer responses was used.

found ($F_{1,24}=3.96$, p<0.1), with the C19 group faster than the C15 group over both types of responses, but the D19 group slightly slower than the D15 group. Again, post-hoc comparisons showed that only the C19 and C15 groups were significantly different (p<0.05) on this measure.

The equivalent analysis for post-target digit RTs produced no main effects of group ($F_{1,24}=0.29$) or age ($F_{1,24}=2.548$), although there was a trend ($F_{1,24}=3.85$, p<0.1) towards the C19 group being faster than the C15 group over both types of response (CW/ FA), but the D19 group being slower than the D15 group. The effect of response (CW/ FA) was not significant on RTs following the responses ($F_{1,24}=0.87$). A response-by-group interaction narrowly failed to reach significance ($F_{1,24}=4.15$, p=0.05). This indicated that for the control groups, post-FA responses tended to be faster than post-CW response, whereas for the dyslexic groups, RTs after either were similar. A response-by-group trend; the older groups being slower after a FA. Post-hoc comparisons showed that for the C15 and the D19 groups, but not the D15 group. For post-CW RTs only the C19 and C15 groups differed significantly (p<0.05).

v) Speed-accuracy trade-off?

Robertson et al discuss, but tentatively dismiss (on the basis of regression analyses with other measures) the notion that the slowing of RT following FAs may arise because participants adopt a more conservative response criterion. To investigate the possibility of a speed-accuracy trade-off in the present participants, a correlation between mean RT and the number of FAs made was performed (see Figure 6.2.1). There was a strong negative relationship overall between mean RT and the number of errors made (r=-0.61, p<0.001); i.e. a speed-accuracy trade-off. Analyses containing each group separately showed that this correlation was slightly stronger for the dyslexic than the control groups (r=-0.72, p<0.005 vs. r=-0.63, p<0.05), although the correlation coefficients do not differ significantly from each other (using Fisher's r' statistic, z=0.41). A significant correlation can also be seen between the number of FAs and the mean RT of responses before a *correctly withheld response* (r=-0.53, p<0.005)⁵. This indicates that the main correlation is

⁵In this case, whereas the same correlation for the group with dyslexia on their own is significant (r=-0.68, p<0.01), the correlation for the control group is only a trend (r=-0.48, p=0.08). These two coefficients do not differ significantly (z=0.72).

not merely due to the speeding of responses during attentional lapses. An increased number of attentional lapses may be expected to increase the mean RT.

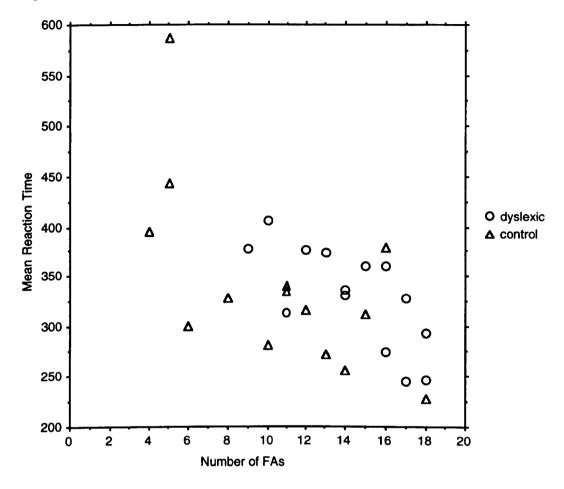


Figure 6.2.1. Plot of mean RT against number of FAs for individual participants

vi) Further analyses

Since RT was related to the number of FAs made, it was considered that this may have had some bearing on the increased number of FAs made by the children with dyslexia (although there were no significant differences between groups on this measure alone). Mean RT (for correct responses) was therefore added into the original two-factor ANOVA (investigating the effects of age and group on FAs) as a covariate. As expected, and reflecting the strong relationship between RT and FAs made, the effect of the covariate (RT) was highly significant ($F_{1,23}=21.68$, p<0.0001). However, rather than being able to explain the increased number of FAs made in the dyslexic groups in terms of RT, the group effect was actually increased ($F_{1,23}=10.85$, p<0.005). There were no main age effects or group-byage interactions ($F_{1,23}=2.55$ and $F_{1,23}=0.85$ respectively).

Discussion

The main result of this experiment was that the children with dyslexia made more errors on the SART than their matched controls, even (or especially) when RT was also taken into consideration. This finding on its own would suggest that the children with dyslexia have difficulty sustaining attention and are more prone to attentional lapses even during a task that lasts under five minutes. However, the *qualitatively* normal pattern of performance in the groups with dyslexia prevents a straightforward conclusion and suggests that, in the present experiment, performance on the SART may reflect more than the ability to sustain attention.

In Robertson et al's study, TBI patients more often failed to withhold responses to targets than the controls (mean number of FAs 7.6 vs. 4.0). Their reaction times were also more variable than those of the controls and they showed a different response style in that they failed to slow down after an error. Except for their high number of failures to withhold responses, the children with dyslexia in the present experiment behaved like Robertson et al's controls; slowing down after making errors and showing only moderate variation in reaction times. Compared with their matched controls in this experiment, they responded equally quickly and with similar variation in reaction times. Both groups showed significant evidence of a speed-accuracy trade-off during SART performance. However, covarying for the effects of RT on the number of FAs enhanced rather than reduced group differences, probably reflecting either a slowed choice RT (Nicolson and Fawcett, 1994) or greater difficulty inhibiting a response within a given time period. In either case, it seems likely that the children with dyslexia were under greater time pressure than the controls, despite their similar reaction times.

There are several reasons why children with dyslexia might be expected to be slower than controls (to achieve the same accuracy) in this task. Nicolson and Fawcett (1994) found that they were slower than controls at choice RT (including omission choice RT) tasks, though not simple RT tasks. If the SART is then considered essentially an omission choice RT task, children with dyslexia would appear to be responding at a speed beyond their true ability. In other words, their error rate is what might plausibly be expected from control children if they were encouraged to respond more quickly at the expense of accuracy. It is interesting to note the trends toward a group-by-age interaction for RT: there was a tendency for RTs to be fastest in the younger group with dyslexia (compared to their older counterparts), whereas the older controls were faster than the younger controls. It is possible that the children with dyslexia slow down as they get older, with increasing awareness of their own strengths and weaknesses, whereas in control children, the number recognition component of the task becomes increasingly automatic and can be performed effortlessly⁶. The Dyslexia Automatisation Deficit Hypothesis (Nicolson and Fawcett, 1990) posits that children with dyslexia have difficulty automatising performance. However, it is obvious from the high error rates in the control groups that they too had difficulty performing this task accurately, although their subskills are likely to be more automatic.

It is a moot point whether the SART is better characterised as a choice RT task than a simple RT task with response inhibition necessary for target digits. Robertson et al (1997) argue that it is not a response inhibition task, but failed to use a pure measure of response inhibition in reaching their conclusions. Instead they relied on the Stroop test and the WCST, which they admit are of questionable validity. However, even if the SART is considered to be essentially a simple RT task, one might still expect the children with dyslexia to exhibit poorer performance, even without showing attentional difficulties. This argument assumes that presented digits in this task do need to be named and cannot merely be recognised visually (which Robertson et al suggest is the case, since randomly allocated font sizes were used in order to enhance the demands for processing the numerical value). If the children with dyslexia took longer to convert the Arabic numerals e.g. '3' into the phonological ones, 'three', yet responded as quickly as the controls, they must have devoted less time to deciding whether to press the mouse button or whether to inhibit their response⁷. Ellis (1981) found that dyslexic and control children were equally fast at responding to whether pairs of letters in the same case (e.g. 'AA' and 'aa') were the same or different, but dyslexic children were slower at making the same judgement for different case letter pairs (e.g. 'Aa'), where the comparison required name coding. Naming speed deficits generally are also well established in both children and adults with dyslexia (e.g. Denckla and Rudel, 1976).

Logan (1981), in a chapter entitled "Attention, automaticity and the ability to stop a speeded choice response" investigated the point at which responses become 'ballistic' as opposed to controllable. Parameters affecting the probability of inhibition, included the ability to predict the occurrence of the stop signal (in this

⁶It is also possible that the response-by-group trend found for post-target digit RTs, where older groups tend to slow down to a greater extent following a FA, may be indicative of better strategy use.

⁷Note that informal observations leave no doubt that all children were well aware of a high percentage of their mistakes (giving audible signs of annoyance). Therefore the differences in the number of FAs made do not reflect difficulty in number recognition within the time alone.

case a tone), the delay between the onset of the choice stimulus and the onset of the stop signal, and the time required to instigate the response (its complexity). It was postulated that the probability of inhibition reflects the proportion of the reaction-time distribution that is slower than the response to the stop signal. It is suggested that in the present experiment, the 'stop signal' may effectively have arrived later for the children with dyslexia since their recognition of the target was delayed⁸.

It could be argued that the task only becomes one of simple RT during periods of attentional lapse (i.e. when the digits are not being labelled before responding) and should, in the normal course of events, be characterised as one of omission choice RT. However, whether best characterised as either a simple or a choice (or omission choice) RT task, it would be expected that dyslexic children would be slower at achieving equal accuracy to controls. Thus the similar reaction times obtained for both groups lead to an expectation of lower accuracy, either with or without any attentional deficit. However, it is also plausible that an additional difficulty with response inhibition generally may contribute to the high error rates in the group with dyslexia⁹. This is possible given the links between dyslexia and ADHD combined with the suggestion that groups with ADHD show greater impulsivity and inability to withhold responses (e.g. Barkley, 1994). Robertson et al found no evidence that the SART reflected ability to inhibit responses in their patients, but since they admit that they were unable to demonstrate that it sits better

⁸ There is also some evidence that adults with dyslexia display increased anxiety even in simple RT tasks (responding to any tone) (e.g. Cox, 1995), possibly suggesting a greater amount of attentional resources needed to achieve a certain level of performance even in a task not usually associated with group differences. If anxiety does also affect performance on this task therefore (as Ballard, 1996, might predict), effects of anxiety in each group might be interesting to investigate in future research, perhaps also in comparison with error rates.

⁹However, it should be noted that Manly (personal communication) suggests that response inhibition may be broadly *the same* as sustained attention to action in this particular context. He is suggesting that lack of response inhibition is caused by lack of sustained attention to action. Furthermore, he appears to dislike the term 'trade-off', since it "suggests that there is some strategic, attentionally demanding titration going on", rather than the speeding resulting from a lapse in attention (see Appendix 6.2.2).

with tests of sustained attention than with a 'pure' measure of response inhibition, this suggests a question worthy of future research¹⁰.

None of the children used in this experiment showed clinical evidence of ADHD and there were no significant differences between the groups in the number of clinical signs of ADHD reported. Furthermore, no differences between groups in terms of bias towards responding were found in the experiment reported in Chapter 5, casting doubt on the issue of impulsivity if not response inhibition. In addition, Sergeant and van der Meere (1988), cited in van der Meere et al (1992), found that hyperactive children failed to adjust their response speed following a mistake: in agreement with their lack of clinical hyperactivity/ ADHD signs, this pattern was not evident in the dyslexic children in this experiment. Nonetheless, the links between dyslexia and ADHD still exists, which suggests that the possibility of some similar symptoms, even if ADHD is not exhibited in full, should not be dismissed out of hand.

The question is then raised of why the children with dyslexia were responding so quickly, which, it has been suggested, is the root cause of their errors. Rabbitt (1979, p305) states that, "It is possible that people control their performance by responding faster and faster until an error occurs, by detecting this error, and by then responding more slowly... in order to maintain a "safe" rate of responding at which both speed and accuracy can be optimized.". This statement both explains the predictability of errors on the SART from preceding RTs and characterises the response style of the control participants in Robertson et al and of our participants

¹⁰A more recent paper [Manly, Robertson, Galloway and Hawkins (1999)] does in fact suggest further evidence that poor performance on the SART arises because of inefficient maintenance of attention rather than an inability to respond per se. They varied both the probability of targets and the task duration and found that the inter-target interval determined task performance far more than the absolute time on task. Results are discussed in terms of the need for endogenous, 'selfsustained' attention (when target probability is low) verses exogenous activation via environmental input from the alternative response (when target probability is high). However, it could be that by varying the probability of targets, they may have actually changed the nature of the task from one of simple RT, or indeed omission choice RT, to one of normal choice RT. The slower RTs shown in conditions with higher probability of targets would support this view. Also of possible interest is that recent work (following on from this thesis) has found that using 'squiggles'/ unnameable objects in place of digits on the SART task removes differences between dyslexic and control groups. If the SART were measuring only sustained attention in dyslexic and control children, the stimuli used in the task would be relatively unimportant and would be expected to impact on reaction times (because recognition can no longer be 'automatic'), but not errors. However, whereas control children make significantly more errors in the squiggles condition than the standard condition, it makes less difference (no significant difference) for the dyslexic children. This might suggest that the reduced possibilities for labelling the stimuli made the recognition time needed for the stimuli more equal for both groups. However, Manly et al might argue that the 'more interesting' squiggles decreased the need for self-sustained attention. Obviously, there are also other alternative explanations for the results.

with dyslexia. Rabbitt also outlines a few of the parameters of control necessary to achieve an efficient speed-accuracy trade-off: detection of errors, monitoring of RT and control of RT. The finding that the children with dyslexia slowed down after making an error suggests that they were able to detect their errors and monitor and control their RT. However, it is possible that children with dyslexia cannot control their response speed as accurately as the control children, and that perhaps their system is not as finely tuned. Previous evidence has found time estimation deficits in dyslexia (Nicolson, Fawcett and Dean, 1995), possibly suggesting a deficient internal timing mechanism. However, any additional noise in the responses of the children with dyslexia might be expected to cause greater variability in their reaction times, which showed only moderate variation.

An anomalous finding in this experiment was that the control children did not (at least in a statistically significant sense) slow down after making an error. This contrasts with the pattern of performance shown by the children with dyslexia and by the controls in Robertson et al's study. One possible explanation of this is that it has been informally noted that in the panel of participants used, many of the children with dyslexia consistently put in more visible effort than the control children and tend to consider many of the tasks a challenge. The control children also try hard but perhaps attach less importance to their performance, although it is probably not often that they are presented with laboratory tasks which are as challenging for them as this one. These results therefore illustrate a general methodological problem in research, namely that of controlling for baseline response rates in tasks that require speed-accuracy trade-offs. Equal response rates may in some instances reflect equal ability, but in others result from motivational differences between groups. A further surprising finding in the present experiment was the extremely high error rate even in comparison to Robertson et al's patients in both dyslexic and control groups. Given the speed-accuracy trade-off found in the present experiment, however, together with the faster mean reaction times (e.g. in Robertson et al's controls before correctly withheld responses mean RT=397ms, compared to the equivalent for our older controls of mean RT=290ms), it is perhaps not entirely surprising. It is also plausible that age differences play a role in the ability to perform the SART, since Robertson et al's participants were much older than those used here.

To summarise, compared with matched controls, children with dyslexia more often failed to withhold responses to target digits on the SART. However, both groups made a high proportion of errors (over 40%). Furthermore, the dyslexic group showed an otherwise normal pattern of responding (were faster on trials immediately preceding errors than on those after errors), and their reaction times to non-targets were (unexpectedly) similar to those of the control children. One explanation, that children with dyslexia have difficulty inhibiting responses, is not supported by Robertson et al's (1997) data but remains an intriguing question for dyslexia research. Meanwhile, from the present results it can be concluded that the performance of the children with dyslexia on the SART is as likely to a general difficulty performing under time pressure as it is to reflect a pure deficit in sustained attention. Further research might address these issues by selectively manipulating different elements of the SART, the nameability and frequency of the target and non-target stimuli, for example. The SART paradigm therefore offers a valuable method for examining the cognitive processes underlying reported attentional deficits in dyslexia.

Chapter 7

General Discussion and Conclusions

Summary:

Evidence collated in this thesis is summarised and related to major theories of dyslexia. Difficulties of research within particularly the area of attention and dyslexia are then discussed together with limitations of the research, especially with respect to the limited opportunity of identifying potential subtypes. Particular reference is made to the large range of deficits which children with dyslexia have already been shown to have and the method by which these can impact on future investigations. Ideas for future research are suggested.

7.1 Issues addressed

The aim of this thesis was to explore theories of dyslexia in terms of their ability to satisfactorily explain the cause of dyslexia. As outlined in the introduction, although phonological deficits in dyslexia are well-established, their cause is less so. Indeed, it is arguable whether they arise from a neurological abnormality specifically related to a 'phonology' area of the brain, whether there is a more general cerebellar deficit or whether a magnocellular deficit causing difficulties with rapid temporal processing is the real root of the problem. A less well considered alternative is that attention deficits are the source of many difficulties with learning and performance. Furthermore, although a possible visual deficit has now been ruled out as the *only* possible difficulty in dyslexia, there remains dispute over whether visual deficits do exist or moreover whether they contribute to any difficulties experienced by children with dyslexia. It is now generally accepted that phonological deficits are not the only deficits which need to be encompassed in a causal theory of dyslexia. Other difficulties, including memory, motor skills and speed deficits all need consideration.

The thesis approached the difficulty of comparing theories of dyslexia from several different angles. Studies 1a and 1b sought to further test the Cerebellar Deficit Hypothesis using a prism adaptation paradigm, but with little success. Studies 2a and 2b more successfully investigated the visual deficit hypothesis using an indirect measure of vergence control across saccades. Study 3 served as an investigation of three hypotheses; cerebellar deficit, rapid temporal processing

deficit and attentional deficit, using Akshoomoff and Courchesne's attention shifting paradigm to examine focus and shift attention abilities as well as time required to re-orient attention. Study 4 focused explicitly on teasing out hypotheses of an automatisation deficit in dyslexia verses a general resources deficit, again using a manipulation of Akshoomoff and Courchesne's paradigm. Study 5 further investigated the attentional deficit theory by comparing group performance on a short test thought to be sensitive to attentional lapse. The studies conducted all presented 'food for thought' in terms of at least one of the theories. In addition, the research had implications for research programmes within the realm of the theories themselves or for the further characterisation of the dyslexic syndrome.

7.2 Summary of results

i) Chapter 2: Prism Adaptation

The prism adaptation study in Chapter 2 found no differences between children with dyslexia and their controls, but results were noisy and difficult to interpret, despite mean results seeming equivalent to those in similar studies reported in the literature. Moreover, the controls failed to show a cerebellar adaptation effect making interpretation of dyslexic behaviour difficult to interpret. Implications for the Cerebellar Deficit Hypothesis of Dyslexia were therefore difficult to infer. This was especially true because of the subsequent PET study (Clower et al, 1997) which, contrary to much of the previous evidence, suggested no direct involvement of the cerebellum in adaptation. The result from the Straight Ahead Shift experiment reported in the same chapter, which suggested no effect of the prisms in an illuminated room, also cast some doubt on results from previous adaptation experiments performed in illuminated conditions. However, it successfully showed that dyslexic and control children did not differ on this (non-cerebellar related) component of adaptation.

As already discussed in Chapter 2, methodological differences between the study presented here and Weiner et al's study on cerebellar patients (particularly with respect to 'automatisable' components in the design) may help to resolve differences in results. However, the process of prism adaptation has so many difficult-to-separate components, some of which may have already been implicated in dyslexia (such as motor control and visual accuracy), that firm conclusions were difficult to make. Furthermore, it has been found that in monkeys (Baizer and Glickstein, 1973), only damage to specific parts of the cerebellum abolishes adaptation to prisms. The cerebellum is the largest structure in the brain in terms of

the number of neurons it contains (Williams and Herrup, 1988) and has many different functions. Since the cerebellar deficit hypothesis of dyslexia is a relatively recent hypothesis, it has not yet specified a particular area of the cerebellum in its predictions. Further work on prism adaptation in dyslexic children might therefore be better left for several years and until such predictions have been specified. There are perhaps too many 'unknown quantities' in this type of experiment. Rather more focused studies, including differences relating to the timing functions of the cerebellum and more complex motor skills, *have* thus far been successful in finding differences between dyslexic and control children.

ii) Chapter 3: Visual deficits?

Results from Chapter 3, which investigated binocular coordination abilities in adults with dyslexia, were easier to interpret than those in Chapter 2. No differences between groups were found on the sequential stereopsis task employed under either single or dual task conditions. In contrast to many previous studies in this area, the sequential stereopsis task was one which did not involve reading, but which did have the advantage that the type of eye movements required were likely to be similar. The technique allowed the assessment of both eyes working together and in a dynamic fashion. The finding of no differences between groups might therefore be considered as strong evidence against there being any visual factor involving either eye movements or binocular stability. However, the sample consisted of well-compensated adults and so is not necessarily representative of the dyslexic population as a whole. It would therefore be interesting to investigate the performance of a sample of mixed ability children with dyslexia on this task.

In terms of other theories of dyslexia, good sequential stereopsis performance of dyslexic students under dual task conditions, might cast some doubt on the DAD hypothesis. However, the DAD predicts that deficits under dual task conditions will be more marked in dyslexic than control students only if either or both tasks are automatic in controls and *only* when all resources have been used. It seems plausible that resources may not have all been used, i.e. the dual task was not sufficiently demanding. However, it is interesting to note the fatigue effect shown in the dyslexic students in the simultaneous stereopsis condition in the first experiment reported in Chapter 3, suggesting that some effort does need to be made in order to perform this type of task, at least in dyslexic students¹. This

¹The number of people who appeared not to be able to perform this sequential stereopsis task, both dyslexic and control (excluded from some analyses in Chapter 3) may also have implications for the method with which people make accurate judgements of distance. However, this issue is beyond the scope of this thesis.

fatigue effect *is* consistent with the dyslexia automatisation deficit hypothesis, which suggests that because dyslexic children have more difficulty making skills automatic, they are more likely to use controlled processing and thus tire more easily. Moreover, it has important implications for future investigations of visual function in dyslexia, suggesting that differences may only be exhibited after considerable visual stress. Thus, although the visual deficit hypothesis of dyslexia would predict a difference in dyslexic and control ability to perform sequential stereopsis, which was not found, the possibility of any type of visual deficit cannot be completely ruled out.

iii) Chapter 4: Attention Shifting

Chapter 4 presented the first investigation of attention and dyslexia. Using a rapid attention shifting paradigm, this experiment looked at the ability to focus and the ability to shift attention in children with dyslexia. The paradigm was originally developed by Akshoomoff and Courchesne and used with cerebellar patients. Four out of five patients with cerebellar damage were shown to have difficulty rapidly shifting their attention. The experiment therefore had the advantage of investigating and characterising any attentional or rapid processing difficulties in dyslexia, as well as having possible implications for the cerebellar deficit hypothesis. The paradigm also had potential implications for the dyslexia automatisation deficit hypothesis since dual task performance deficits could reflect a difficulty in rapid attention switching between two tasks. However, this argument has already been dismissed by Fawcett (1990), because of data which suggested that two tasks did have to exceed some crucial resource threshold before difficulties arose; the difficulty could therefore not reflect an attention switching deficit alone. Nevertheless, a difficulty in attention allocation could also potentially underlie the automatisation deficit, due to poor concentration in the initial skill acquisition stages.

Results from Chapter 4 indicated that rather than a rapid attention shifting deficit, or a focus attention deficit, children with dyslexia were found to have difficulty shifting attention overall. Further examination of mean performance of dyslexic and control groups suggested that children with dyslexia could initially shift their attention as well as controls, but that, unlike controls, they did not improve with further time to prepare their responses. Two non-exclusive explanations were considered. Firstly (from the DAD) that the shift attention condition was more complex than the focus condition, required more resources and was therefore more difficult for dyslexic children because components of the task which had been automatised in control children required controlled processing in dyslexic children. The second possibility (from the phonological deficit hypothesis) was that the children with dyslexia had difficulty remembering the current target because of a reluctance or a difficulty to label and rehearse the shapes. This difficulty in labelling could, of course, be why the shift condition was so much more difficult for the dyslexic children, so that other resources were not available. The phonological deficit theory and the DAD therefore make similar predictions in such situations, with the DAD postulating that dyslexic children are less automatic at phonological tasks. It should be noted that a split-half analysis of data from the shift attention condition suggested that dyslexic children could sustain their attention over the 15 minute period of the shift attention condition equally as well as controls, suggesting that a sustained attention deficit was not a cause of lower performance.

Thus, this attention shifting paradigm too failed to provide support for the Cerebellar Deficit Hypothesis, but this may not be surprising in that more recent attempts to replicate the findings in cerebellar patients have been unsuccessful. Furthermore, the paradigm cast some doubt on the rapid temporal processing deficit hypothesis. Indeed, rather than a rapid shifting deficit, the difficulties appeared to arise later on, so that the problem appeared to be one of keeping the target in mind at the same time as doing everything else. Although the rapid temporal processing deficit hypothesis would normally predict difficulties with much faster acts of processing than required in this experiment, the specific pattern of results obtained were still considered to be unlikely if such a deficit were present. Instead, results offered potential support to both the dyslexia automatisation deficit hypothesis and the phonological deficit hypothesis. The DAD could account for the pattern of results shown since the shift condition required more resources, in addition the dual task performance deficits found by Fawcett (1990) were unlikely to be accounted for by difficulties in rapidly switching attention between two tasks. Performance on the shift attention condition correlated very strongly with performance on a phonological task suggesting that ability to label the target might be important subsequent to the initial switch, thus providing evidence that the phonological deficit theory may be able to account for differential performance of children with dyslexia and their controls on some attentional tasks. Further tests of the automatisation deficit argument were presented in Chapter 5.

iv) Chapter 5: Investigation of resources

Chapter 5 asked whether the original focus-shift dissociation could be explained by the DAD (as speculated in Chapter 4). An important theoretical question associated with this is whether the difficulties of dyslexic children on dual tasks and under more resource-consuming conditions is an automatisation deficit, or whether the difficulties are better characterised by a simple resources deficit. In addition, the apparent difficulty in making skills automatic is able to successfully explain many findings so one might therefore ask whether it is, in fact, too powerful as an explanation. There is evidence that as soon as a task is made more complex or more difficult in some way, the performance of children with dyslexia becomes disproportionately worse (e.g. Nicolson and Fawcett, 1994). This is perhaps not entirely surprising, because as a group, dyslexic children are impaired in such a wide range of skills. Adding one task involving one area of difficulty to another in another area of difficulty, might be expected to produce far more difficulty than a simple additive model might suggest (even without consideration of resource levels). It was reasoned that by visually degrading the shapes in Akshoomoff and Courchesne's paradigm it would be possible to dissociate the two possibilities of automatistion and resource deficit in a way where making the task more difficult would not necessarily disproportionately disadvantage the dyslexic children if they had an automatisation deficit. Visually degrading the shapes had the properties of (i) increasing the resources necessary to perform the task and (ii) preventing automatic recognition of the shapes. Thus, if dyslexic children were more strongly affected by the degradation then it is likely that the extra resources required to recognise the shapes pushed them to their resource ceiling before the controls and therefore a lower resource capacity is the best explanation. On the other hand, if the dyslexic children were not affected to the same extent as the controls by the degradation, it seems plausible that this is because they are performing the task with such great difficulty (relatively) already whereas the control children can do the shape recognition part of the task (at least) automatically. The latter interpretation, namely a deficit in automatic shape recognition in children with dyslexia was supported. The evidence was particularly strong because in this situation (in contrast to the more usual situation of dyslexic children's performance decreasing most under more complex conditions) the control children suffered more under a more difficult condition. In Chapter 4, it was difficult to separate hypotheses generated by phonological and automatisation theories, since the DAD postulated automaticity deficits in phonology. However, in Chapter 5 the evidence does seem to suggest that the deficit is more likely related to shape recognition since the dyslexic groups' performance barely decreases at all under degraded

conditions. If the difficulty were principally one of phonology, or automaticity of phonology, the degradation would surely still be expected to reduce performance in both groups on top of any difficulty with phonology. This argument assumes that automaticity of labelling is not the *only* difficulty presented by degradation of stimuli, but that extra visual or cognitive resources in forming an image of the shape itself is also required. A shape recognition automaticity deficit does therefore seem the most likely explanation of the finding.

v) Chapter 6: Attentional lapses

Chapter 6 further investigated difficulties of dyslexic children in 'keeping on track' (Augur, 1985), together with other reported attentional difficulties. It asked whether these observations could be characterised better by shorter 'lapses' in attention; a deficit in sustained attention over a period of seconds rather than minutes. Significant differences between groups on the Sustained Attention to Response Task (Robertson et al, 1997) suggested that this may be the case, although alternative explanations were possible. An important finding on this task was a speed-accuracy trade-off. Robertson et al (1997) had already suggested that a quickening of response times occurred prior to errors. However, results from Chapter 6 suggested a trade-off even when only responses before a correctly withheld response were considered, suggesting that some people had a tendency to respond faster (leading to decreased accuracy) anyway. It is therefore possible that although the SART may well reflect sustained attention in the controls and patients used by Robertson et al, in people with dyslexia it may involve different cognitive components. These components could involve difficulty in recognising the numbers quickly, so that the response has been initiated before the signal to withhold responding has been processed; this would be consistent with automaticity explanations. Alternatively, dyslexic children, similar to children with ADHD, may have difficulty inhibiting responses. These are questions which are being addressed in ongoing research.

vi) Overall Synopsis

Figure 7.2.1 provides a summary of the bearing of each chapter on each theory. It can be seen that despite the fact that at no point was the phonological deficit theory explicitly investigated, it could have potentially explained results from Chapters 4 and 6. Such findings highlight the difficulty in investigating (particularly) attention in dyslexic children without encroaching on something with which they already have known difficulties. This issue is discussed further in section 7.3. The phonological deficit is probably already the most well investigated of all the

theories, with consistent supportive results (though most investigations have been limited to phonological tasks). However, as an exclusive or stand-alone theory, it is unable to explain many of the findings either in this thesis or of other researchers in the field (as already discussed in section 1.3), including the visual fatigue effect found. Because the phonological deficit theory and the dyslexia automatisation deficit hypothesis make similar predictions in terms of the automaticity of phonological tasks, in many situations they can be difficult to separate. However, the weight of the evidence in Chapter 5 supports an automatisation deficit, rather than a phonological deficit alone.

For the DAD, however, the picture is more positive. This hypothesis has been able to account successfully (and in many cases elegantly) for a wide range of findings throughout this thesis. These findings have ranged from the fatigue effects found in Chapter 3, through to the focus-shift dissociation found in Chapter 4. The group-by-visibility interaction in Chapter 5 provided particularly strong evidence for an automatisation deficit. It may also be able to explain the lack of a strong adaptation effect in Chapter 2 and (through a slow non-automatic labelling process) the difficulties of dyslexic children in Chapter 6. The hypothesis has therefore not been presented with anything which it cannot account for within the scope of this thesis. However, as outlined in the introduction, the DAD provides no causal mechanism for the automatisation deficit; it is only a cognitive level explanation. Instead, the Cerebellar Deficit Hypothesis provides the biological mechanism for automatisation deficits and other deficits. However, no evidence for cerebellar involvement specifically, over and above automatisation deficits, was found in this thesis. However, the prism adaptation paradigm was not successful and some doubt has now been cast on the original attention shifting experiments in cerebellar patients.

Similarly, the visual deficit hypothesis has received little support. No differences between groups were found on the sequential stereopsis task used. However, the groups were all well-compensated adults. In addition, a fatigue effect was found in the dyslexic group on one condition suggesting that visual differences might arise in the dyslexic group given sufficient visual stress beforehand. Nevertheless, visual deficits cannot explain the focus-shift dissociation found in Chapter 4 or the group-by-visibility interaction found in Chapter 5.

		Theoretical Approaches					
Cha	apters	Phonology	DAD	CDH	Vision	RTPD	Attention
2	Prism Adaptation	nothing to explain!	- nothing to explain	(X) predicts difference but paradigm unsuccessful	- nothing to explain	– nothing to explain	nothing to explain
3	Sequential Stereopsis	X Can't explain fatigue effect	Can explain fatigue effect	explains nothing above the DAD	? predicts difference - although fatigue effect found	X Can't explain fatigue effect	? fatigue effect related to sustained attention deficit?
4	Attention Shifting	? could explain - labelling required to track current target	? could explain - shift condition requires more resources does not falsify dual task deficits	(X) no rapid switch deficit but some doubt on original experiments	X can't explain focus shift dissociation	X no rapid switch deficit can't explain focus- shift dissociation	X no focus attention deficit can't explain dissociation
5	Resources	Controls suffer most in resource consuming conditions (labelling effort constant)	shape recognition automaticity deficit means degradation has little effect	explains nothing above the DAD	X might expect more difficulty in degraded conditions for dyslexic group	X can't explain interaction with visibility	X can't explain interaction with visibility - NOT a floor effect for dyslexic group
6	Sustained Attention	? could explain - later stop signal if consider task one of response inhibition or omission choice RT	? could explain - dyslexic children going too fast Automatic Shape Recognition Deficit	- explains nothing above the DAD	can't explain	? could explain in terms of slow recognition of target, but dyslexics do realise they have made mistakes	? could explain - group difference as predicted, but why the speed-accuracy trade-off and otherwise normal performance?

The rapid temporal processing deficit hypothesis was given opportunity for support in Chapter 4, where a rapid attention shifting deficit was hypothesised. As already discussed, such a deficit was not found. In fact, dyslexic and control groups' performance was most equal when rapid shifting was required. However, this did not result in a group-by-time period interaction, making results a little difficult to interpret (see section 7.3 below). Although the rapid temporal processing deficit hypothesis actually relates to processing much quicker than the 'less than one second' category used in this experiment (in a pure auditory task. for example, differences between groups are found within a time frame of tens of milliseconds: Tallal, Miller and Fitch, 1993), this does not include time for higher level processes such as a shift of attention. One might therefore still expect that if a rapid temporal processing deficit were present, performance of dyslexic children would be worse under more rapid conditions; this was not the case. A previous study which explicitly looked at the possibility of rapid temporal processing deficits in dyslexia using many of the same participants (Moores, 1995; unpublished dissertation) also failed to find differences between groups. It is quite plausible, however, that a subgroup of dyslexic children do show rapid temporal processing deficits. JM's performance in Chapter 4 (and in Moores, 1995) is suggestive of such a deficit. In Chapter 4, JM showed that he had difficulty on the focus conditions under conditions where two targets appeared one after another. but not on the equivalent shift condition under the same time constraints. Furthermore, Moores (1995) showed that JM could distinguish between which of two shapes appeared first when presented on the screen side-by-side in rapid succession, but had more difficulty deciding which of two sounds was played first when the time period between them was small. A plausible interpretation of this pattern of deficits is that JM has difficulty in processing two successive stimuli when they are apparently superimposed onto one another: a rapid temporal processing deficit, perhaps reflected by visible persistence in the visual system. However, a rapid temporal processing deficit cannot account for the visual fatigue effect in found in Chapter 3, the focus-shift dissociation found in Chapter 4. or the group by visibility interaction found in Chapter 5. Results in Chapter 6 could potentially be explained by a rapid processing deficit (the digits were flashing up quickly), although the fact that the dyslexic children obviously recognised when they had made mistakes casts some doubt on this interpretation.

Little evidence was found which supported the idea that an attentional deficit played a major role in dyslexia. The main evidence regarding attentional function came from Chapter 4, where no focus or sustained attention deficits were found and the initial act of shifting attention was concluded to be sound. Furthermore, the greater number of errors made on the SART task in Chapter 6, were concluded to be as likely to be related to automaticity deficits or response inhibition deficits as difficulties in sustaining attention, with dyslexic children showing otherwise normal performance on the task. Moreover, an attentional deficit is unable to explain the group-by-visibility interaction found in Chapter 5. Had this effect been a floor effect then an attentional explanation would have been satisfactory, but it was not. In the same way, the apparent fatigue effect could potentially be explained by a sustained attention deficit, i.e. boredom with the task. However, the equal decrement in performance over time shown in both groups in the shift attention condition of Chapter 4 suggested that this was unlikely to be the case

7.3 Limitations of research and future directions

Determining the specific deficit in dyslexia

In many of the experiments investigating aspects of attention, it has once again been highlighted how difficult it is to look at attention alone. This methodological difficulty (which is by no means new!) becomes particularly problematic when comparing groups on tasks with which children with dyslexia have known difficulties (which are now thought to be far reaching: see e.g. Nicolson and Fawcett, 1995). Confounding factors in experiments may include memory loads, response time, automaticity of tasks, the effect of a dual task, temporal processing, timing ability, or phonological ability.

If investigating memory abilities or response time, it is obviously necessary to consider attention; if someone is not paying attention they will not be able to remember or respond correctly. Equally important, is that when investigating attention, it may be necessary to consider memory abilities and response time. It is certainly difficult to investigate attention without using another task. Some kind of response is usually made by the participant in order to indicate that he or she has been 'paying attention', either by responding to a stimulus (involving recognition of that stimulus and a motor response) or by repeating what had been heard or seen (involving memory abilities as well as vision or audition). Because the range of deficits shown in children with dyslexia is large, careful manipulations become increasingly important to control for such factors. Halperin (1996) offers a reminder that psychological tests only measure the final product of a number of cognitive processes and states that, "to infer more confidently that the process being assessed is attention, one must directly manipulate the attentional component

of the task while maintaining equivalence across all other parameters". This is particularly true in dyslexia research.

Halperin (1996) suggests that when comparing groups, a group-by-task interaction is necessary in order to demonstrate a specific deficit, rather than a main group effect. A rapid attention shifting deficit, for example (as originally hypothesised in Chapter 4), would be illustrated by a group-by-time period interaction, with equal performance on all the time periods except the most rapid. A main group effect on a shift attention condition (as found in Chapter 4) would not be able to pin-point the exact difficulty to a problem shifting attention. However, in Chapter 4, two equivalent focus conditions found no difference between groups. Furthermore, the split-half analysis of the shift attention condition showed that dyslexic groups showed no greater sustained attention deficit than their controls. Thus, the possible interpretations of the deficit in the shift attention condition are more limited than they may otherwise have been. However, whether the problem is in shifting attention per se, or due only to extra resources required (or another factor) in the shifting attention condition, is not known. Nonetheless, the fact that both groups showed equal performance on the rapid attention shifting condition does suggest that the dyslexic group were as able as the control group at the specific shifting component of the task. Results from Chapter 4 alone therefore suggested either a resources deficit of some kind, a problem keeping track of the current target, or a combination of the two.

Chapter 5 was rather more successful than Chapter 4 at pinpointing a deficit in dyslexia. In conditions where two focus and two shift conditions differed only by their visibility, a strong group-by-visibility interaction was found. Because the only difference between the conditions was the visibility of the stimuli, it was possible to conclude that it was this component specifically which was affecting the results.

The experiment presented in Chapter 6, however, was less successful in pinpointing the exact deficit in dyslexia. It revealed similar potential complications to those reported for Chapter 4. The task used had been designed to measure sustained attention and had already been postulated to reflect attentional lapses in TBI patients. Nevertheless, the possibility of the task reflecting response inhibition had also been discussed by Robertson et al. Thus, the significant relationship found in Chapter 6 between speed of responding and accuracy gave some cause for concern. Firstly, because one reason for investigating attention in dyslexia was the connections between ADHD and dyslexia and because children with ADHD have been found to be impulsive in their judgements (Barkely, 1994). It therefore seems plausible that children with dyslexia might also be impulsive, which would be likely to decrease their performance on the SART for reasons other than those of sustained attention; namely that they were trying to perform the task too quickly. A second reason is because even though groups were shown to have very similar reaction times, children with dyslexia have been shown previously to have slower reaction times to stimuli under most conditions. The similar reaction times could therefore be taken to suggest that children with dyslexia were responding at a speed beyond their true ability, even if not 'impulsively'². Subsequent work using the SART paradigm has used modifications of the paradigm together with the original test in an attempt to tease out different possibilities of the differences found via discovery of dissociations between different conditions. This approach has been successful.

Significant amounts of care therefore need to be exercised both in designing and interpreting data from experiments using children with dyslexia. It is possible that a relatively small number of basic deficits, such as that of response time, are manifesting themselves in other ways. This may be creating the illusion that children with dyslexia are seemingly poor at a larger variety of things than they actually are. The DAD suggests that children with dyslexia have poorer performance than their counterparts on a large variety of tasks. It would be interesting to break down the components of some of these tasks in order to investigate whether several difficulties can be explained by a fewer number of underlying factors. In terms of attentional deficits, it may also be interesting to note at this point that *apparent* sustained attention deficits may actually be expected in children with dyslexia on tasks which have not been automatised. In terms of the DAD, however, this would not necessarily indicate a sustained attention deficit, but rather an automatisation deficit with faster tiring for children with dyslexia, due

²It may be interesting to note at this point that an exploratory analysis of the performance of participants who had taken part in all the various attention tests was performed (see Appendix 7.1). Small sample sizes limit the conclusions which can be drawn. However, in correlational analysis of dyslexic and control participants separately (separated because otherwise the groups might have a tendency to form two clusters rather than a linear correlation), it can be seen that despite the small sample size, many of the measures of attention used correlate highly (in the expected directions) in the control participants. This is perhaps suggestive of the presence of an underlying measure of 'attention' (generally) throughout the tests. Correlations are lower in dyslexic group sample size was even smaller than that of the controls. However, it is possible that the different varieties of attention and the different methods used to assess them (whether or not labelling or shape recognition was necessary, for example, or speed of presentation), may have had substantial effects on the dyslexic group.

to the extra effort required on their part to reach a certain level of performance. Different interpretations of many findings are therefore plausible. Future research should therefore concentrate on looking for interactions within or dissociations between tasks.

Pin-pointing cerebellar function

As already mentioned in section 1.6, the cerebellum has only relatively recently been considered as anything but a motor area. The exact functions of the cerebellum in areas such as attention are therefore rather controversial, with neuropsychological studies of cerebellar patients often eliciting different results either with different methodologies or with different (usually small) samples. Involvement of the cerebellum in even the prism adaptation paradigm has been questioned, despite adaptation having long been associated with the cerebellum. This paradigm was found to be additionally difficult to use because of the difficulties in keeping the cognitive and sensory components of adaptation separate and controlled. It is possible that although the cerebellum does play an important role in adaptation, it is not a sensory one as previously thought. Such difficulties have somewhat limited investigation of the cerebellum in the hypothesis of dyslexia, particularly since a specific area of the cerebellum in the hypothesis has not been specified; thus if it is clear which small area of the cerebellum is involved in a particular function it may not necessarily be an area involved in dyslexia.

Many of the controversies over cerebellar function have arisen due to functional imaging technologies, allowing us to see directly which parts of the brain are activated during various tasks. However, the interpretation of such results still requires handling with some care. Subtraction methods are generally used in functional imaging. Like many behavioural methods, these require that two or more tasks differ in only a single cognitive component of interest e.g. focusing attention vs. shifting attention. However, like behavioural methods, the component of interest often involves much more than just the specific component e.g. shifting attention requires more resources generally or a greater amount of memory. Thus, in the fMRI study examining focusing and shifting of attention (Le et al, 1998), the cerebellar activation for the shifting attention condition could be due to a number of functions inherent in shifting attention, possibly increased need for prediction and preparation (Allen et al, 1997), general resources or memory (see Chapter 4 discussion). Moreover, since Akshoomoff and Courchesne (1994) found a deficit in rapidly shifting attention, rather than a shifting attention deficit overall, the finding of cerebellar activation in a shifting attention paradigm is perhaps not quite

as strong corroboration as it might first appear. Nevertheless, the idea that the cerebellum plays a role in prediction and preparation, or pattern extraction (Courchesne and Allen, 1997) opens up further possibilities for research into dyslexia and the CDH. For example, a preparation deficit could potentially explain the finding of slower choice reaction times in dyslexic children (e.g. Nicolson and Fawcett, 1994). If dyslexic children fail to 'prepare' a template for which stimulus they are looking for, perhaps instead waiting for the stimulus to appear before deciding on their criteria, then reaction time might be expected to be slower. It may be interesting to note that van der Meere, Vreeling and Sergeant (1992) argue for a motor preparation deficit in hyperactive, though *not* learning disabled (academic underachievers with average IQ), children. They cite evidence suggesting that hyperactive children only act like controls under conditions of rapid presentation rates (contrary to the usual opinion of impulsivity in hyperactive children) they show slower reaction times.

It has therefore been seen above that pin-pointing cerebellar functions on which to compare samples can be difficult. However, with respect to evaluating the different theories of dyslexia in this thesis, it is important to separate explanations at the cognitive level from those at the brain level (cf. Frith, 1997). Although at the brain level there is no direct support for the CDH in this thesis, support does exist at the cognitive level of automatisation deficits (cf. DAD). It should also be re-iterated that the attentional test was not a direct test of either the CDH or the RTPDH and that the prism adaptation paradigm was less than successful and difficult to interpret anyway. In contrast, automatisation is an established role of the cerebellum. Furthermore, studies in this thesis show little support at either level of explanation for theories other than the DAD and the CDH. Future research at the cognitive level of explanation might address issues of prediction and preparation as discussed above, or make further attempts to falsify the hypothesis of an automatisation deficit. Results from the initial attention switching experiment suggest that rapid attention switching difficulties cannot account for dual task deficits. Results from the degradation of stimuli suggests automatic shape recognition difficulties rather than a general resources deficit. However, an interaction in the opposite direction could have also been plausibly interpreted as an automatisation deficit, though only in a skill other than shape recognition, such as the motor skill required in making the response. It might therefore be interesting to attempt to make the response component non-automatisable in some way. It might also be valuable to further separate phonology and automaticity explanations by

presenting symbols and signs which would be expected to be recognised automatically and quickly, but which are less likely to ever be named. A study following the SART study in Chapter 6 used squiggles in place of digits, because they are less likely to be labelled, but they are also less likely to be recognised automatically. It also found evidence of a response inhibition component in the SART for control, but not dyslexic, children. Road signs, for example, might be expected to be recognised automatically, but the length of their names might tend to prohibit labelling: the roadworks sign for example might (intuitively) be expected to processed quickly and automatically in terms of its semantic meaning, without the need for labels³. However, whether either in the shifting attention paradigm or in the SART participants do actually label the stimuli at all, be they shapes or digits, is a matter for debate.

Related to the ability to process symbols and shapes automatically, further research might also examine dyslexic performance on visual search paradigms where either serial or parallel search (controlled vs. automatic) is required: pop-out (Treisman, 1991). Treisman (1991) argues that if one stimulus differs from the others on the basis of one distinctive feature, such as colour or shape, then 'pop-out' will occur, whereas if a combination of features is necessary to distinguish the stimulus from the others, a serial search will be necessary. It might be interesting to use this technique to investigate at what level of processing and under which conditions difficulties arise for dyslexic children. Preliminary evidence (Procter, 1994) suggests no differences between dyslexic and control children on a simple colour (black/white) pop out task. However, it would be interesting to look at shape recognition, possibly using a basic level first (e.g. vertical/ horizontal lines) and then working up to whether, for example, a triangle would pop out amongst circles. It might be informative to finally investigate whether or not digits would pop out amongst letters.

Further research on the differences on a primed lexical decision task discussed in section 5.1 (Hartley, Lindley and Nicolson, in preparation), might use negative, rather than positive priming, in order to offset reaction time differences often displayed by children with dyslexia anyway. The original study found that dyslexic children's reaction times to a lexical decision task did not benefit as much as those of the controls from a semantically related (vs. an unrelated) prime.

³ Brachacki, Nicolson and Fawcett (1995) found impaired ability in dyslexic adults to distinguish between real and false traffic signs, and no correlation in the dyslexic group between this ability and driving experience.

However, it is likely that the aspect of needing to read the prime is likely to affect the results. Reaction time differences may also be difficult to interpret when reaction time differences exist between dyslexic and control children anyway. It might be that slower reaction times are more variable and not as easily affected by priming because it is the motor response, rather than the decision aspect which makes it slow (or vice versa because a fast reaction time is more difficult to improve). This may be a question for future research into the area of priming. However, a negative priming paradigm might be able to avoid at least some of these issues, particularly if compared with a positive priming paradigm at the same time. Negative priming assumes that presentation of an identical stimulus to the target will actually slow down the response to the target. Thus, if a priming difference between dyslexic and control children exists, there would also be expected to be a difference on a negative priming paradigm. The advantage of using negative priming is because a 'priming effect' for negative priming would be demonstrated by a slower response; controls being slower than dyslexics is more surprising, given the evidence, than the other way around. However, it could still be the case, that the negative priming had not worked because the dyslexic children had not read the prime! There is no reason for the prime not to be displayed for some considerable time though and if results could be dissociated from a positive priming paradigm, then at least other explanations of the original findings could be considered.

Subtypes and sample size

The possibility of subtypes in dyslexia has already been mentioned in Chapter 3 and in section 7.2 above. There is some controversy over whether or not distinctive subtypes exist in dyslexia. Ellis, McDougall and Monk (1996b) argue that the variability exhibited amongst dyslexic children is no greater than that exhibited by non-dyslexic children. However, it is not unreasonable to suggest that subtypes of dyslexia do exist. There may be several underlying causes of dyslexia which manifest themselves in subtly different ways. In terms of methodology, the argument for dyslexia subtypes is important with regard to numbers of participants needed to find differences between groups and whether those participants should be pre-selected for particular difficulties or not. The non-consensus on the types of subtypes, however, further complicates the issue. In any case, none of the experiments reported in this thesis used sufficient participants in order to determine particular subtypes. Furthermore, it is unlikely that differences between dyslexic and control groups would have been found where differences may have existed only for particular subtypes. However, my personal opinion is that this is unlikely to have had any major adverse effects on any main conclusions, although this point is certainly arguable.

Specificity to dyslexia

One important issue which has not been tackled at all throughout the course of this thesis is that of slow learners. Although this thesis is first and foremost about dyslexia, it is important with regard to theories of dyslexia to consider differences between children with dyslexia and slow learners. The phonological theory of dyslexia, in particular, has received some criticism because slow learners appear to have phonological problems too; the deficit is therefore not specific to dyslexia. Thus, although it is accepted that phonology plays a large part in learning to read. the difficulty with phonology may be no worse in children with dyslexia than it is in slow learners (who experience the same difficulties in learning to read). None of the experiments within this thesis have had such controls and therefore it is not possible to be certain whether the deficits which have been shown are specific to dyslexia. Particularly in terms of an attentional deficit, it would be very interesting to have at least one other extra control group, perhaps of children with ADHD. Many previous studies have compared children with dyslexia, children with ADHD and children with both ADHD and dyslexia on various tasks (see sections 4.1, 4.2 and 4.3 for a selection). This is an obvious avenue for further research.

Reading age matches and other methodological limitations

The lack of control reading age matches for the dyslexic participants in this thesis also somewhat limits the conclusions which can be drawn from the experiments presented, particularly in terms of looking for *causal* explanations of dyslexia. The reading-level match design, as advocated by Bryant and Goswami (1986), is now commonly used. The advantage of using such a design is to determine whether differences between dyslexic and normal readers are basically quantitative or gualitative. A quantitative difference would manifest itself in the way that dyslexic children simply have not reached the same stage in reading as normal readers, but they are following the same pattern and may or may not get to the same level eventually. If chronological age matches alone are used, it is not possible to determine whether any deficit found is associated only with a low reading age, or is indeed an important symptom or predictor of dyslexia. A qualitative difference would occur if dyslexic children differed from chronological and reading age matches; the dyslexic children would obviously be learning to read via different routes or stages, or using different mechanisms. A difference between groups if matched on reading age would be less likely than one between groups matched just on chronological age. Reading level matches could either be younger children without dyslexia but with the same reading age, or slow readers of the same age who have not been diagnosed as dyslexic, despite having the same reading age (due to a low IQ - "garden-variety" poor readers). Future research programmes, as they become closer to finding any true differences between dyslexic and normal children, may consider it useful to use both groups in order to either verify or nullify the current definition and criteria for diagnosis of dyslexia as discussed in the introduction.

In addition, the use of longitudinal designs for some types of research might be preferable. Shaywitz and Shaywitz (1994) discuss the benefits and limitations of analysing performance over time. In particular, they discuss how individual growth curve models allow a dynamic view of learning (at least for quantitative changes), can be used to differentiate between delays and deficits in developmental research and can be used to study the effects of intervention. Yap and van der Leij (1994b), for example, examined the performance level, rate of progress and sensitivity to intervention of dyslexic children on speeded and unspeeded reading tasks. By using a combination of longitudinal, cross-sectional and intervention studies, they were able to show that dyslexic children specifically (as opposed to either reading age or chronological age matches) had a specific impairment in reading pseudowords under speeded conditions. In a second study they showed that, compared to reading age matches, dyslexic children progressed at a similar rate on an unspeeded but not a speeded reading task. An intervention study showed that although dyslexic children could improve their speeded reading performance on real word reading tasks, they were more limited in their ability to improve on pseudoword reading tasks. Results were interpreted in terms of both phonological and automatisation *deficit* theories. Furthermore, as discussed in section 1.1, a causal theory of dyslexia should be able to provide evidence that the deficit is *a precursor* to the behavioural signs; a longitudinal design is obviously essential in doing this. Ellis (1994) offers a useful evaluation of models of spelling and reading development in terms of longitudinal studies performed by a number of researchers over several years.

Another important limitation of the research presented is that, thus far, the principal findings have not been replicated either within samples or with further samples. Many of the participants in each experiment were the same and therefore the results might not necessarily generalise to other groups. Furthermore, it can be seen from the psychometric details of participants, particularly in later experiments, that the

older dyslexic group had particularly severe reading impairments to the extent that by Chapter 5 their mean reading age was actually lower than that of the younger dyslexic group. Thus, if the strength of any results found in this thesis is related to the severity of the reading deficit in any group, future replications may show inconsistencies. Thus, as with all such research replication will be necessary in order to ensure reliably generalisable results.

Fatigue effects

The visual fatigue effect found in the dyslexic group in Chapter 3 can be categorised as both a limitation of the present research and an important finding in itself. Although it somewhat limits conclusions which can be made from the present experiment, it has important implications for future research and is in itself interesting. Future research may include visual investigations following conditions of visual stress in order to investigate whether visual deficits can be (or can be more consistently) found. Another possibility would be to investigate whether the coloured filters proposed by Wilkins (1995) have an increased effect following such visual stress in order to work towards an understanding of the mechanisms on which they operate.

7.4 Summary and Conclusions

This thesis has investigated a wide variety of abilities in dyslexic children and has also had implications for existing theories of dyslexia. The pattern of results shown throughout all the experiments has suggested a greater range of deficits than the most established theory of dyslexia, the phonological deficit theory, might suggest.

The DAD, albeit a very general hypothesis, was able to account well for the totality of the findings throughout the thesis. Further convincing evidence for the DAD not only dissociated the possibilities of a general resource deficit and an automaticity deficit for the first time, but moreover illustrated a situation where making a task more difficult impacted on dyslexic performance less. However, no evidence was found for a specific cerebellar deficit in this thesis, despite the fact that the CDH grew, at least in part, from research into the DAD. Although automatisation is thought to be a cerebellar function, no evidence of impairment in other functions which are generally associated with the cerebellum was found. Thus, not withstanding recent evidence casting doubt on the cerebellar role in adaptation and attention shifting anyway, it is possible either that any cerebellar deficit in dyslexia might be restricted to timing functions, or that the site of the deficit is in fact elsewhere. Timing is crucial for motor control and indeed Nicolson, Fawcett and Dean (1995) did find explicit evidence of time estimation difficulties in children with dyslexia. It might therefore be interesting to further examine the process by which a task *becomes* automatic; it is quite plausible that timing does play an important role in motor tasks at least. Nicolson et al (1999) did find explicit evidence of differences in cerebellar brain activation during a simple task, but this too was a motor task for which timing was likely to play a role. If timing difficulties *are* the main difficulty in dyslexia then differences in adaptation to prisms would not be expected to be found unless perhaps (as discussed in section 2.5) an assimilative corrective response is possible. Similarly, timing is not involved in rapid attention shifting other than to do it quickly!

Attributing dyslexic difficulties to a timing deficit, possibly sited in the cerebellum, may ultimately be able to account for difficulties in rapid temporal processing. phonology and motor skills. As discussed in section 1.4, there is some confusion over the exact nature of any auditory rapid processing deficit. It is an interesting hypothesis that some of the difficulties in consonant discrimination may be attributable to deficits in timing, rather than rapid processing. One possibility, for example, is that voice onset time, known to be particularly important in distinguishing between some consonant sounds (e.g. d and t) is mis-estimated in dyslexic children. This could be easily investigated by taking advantage of phenomenon such as the Ganong effect, dissociating frequency and timing information. There is therefore little evidence so far that other functions of the cerebellum necessarily need to be implicated in dyslexia at this stage. However, a timing deficit cannot account for the results found in Chapter 5, attributed to a shape recognition automaticity deficit. Further work is therefore necessary to examine the nature of any automaticity deficit in dyslexia, investigating functions both with and without the involvement of a timing mechanism.

To my knowledge, the investigation of attentional deficits in dyslexia has been one of the few systematic investigations of its type to date. However, some of the results which suggested attentional deficits in dyslexia are probably attributable, at least in part, to either labelling or automatic recognition difficulties. The investigation of attentional function in dyslexia is therefore likely to prove difficult and to separate from other functions with which children with dyslexia already have known difficulties. Pilot work has suggested, however, that performance on the SART *does* correlate with an attentional lapse questionnaire in dyslexic adults, although replacing the digits with squiggles has also been shown to eliminate group differences in dyslexic children. Future work is planned in order to examine the performance of children with ADHD on the SART.

The apparent fatigue effect on the simultaneous stereopsis task, together with good performance on the sequential task, may offer further leads to those investigating visual factors in dyslexia. In terms of implications for methodology, such findings may be particularly relevant to Arnold Wilkins' work on visual stress and the use of coloured overlays (see e.g. Wilkins, 1995). It may also be able to explain the apparent unreliability of any reported visual differences in dyslexia.

The major limitation of work presented is that the deficits found are not necessarily specific to people with dyslexia. Further work involving children with ADHD and children with other learning difficulties would be beneficial to understanding, as well as providing answers to important theoretical questions in terms of the causal factor in dyslexia.

- Ackerman, P.T., Anhalt, J.M., Dykman, R.A. and Holcomb, P.J. (1986). Effortful processing deficits in children with reading and/ or attention disorders. *Brain and Cognition*, 5, 22-40.
- Ackerman, P.T., Dykman, R.A. and Peters, J.E. (1976). Hierarchical factor patterns on the WISC as related to areas of learning deficit, *Perceptual and Motor Skills*, 42, 583-615.
- Ackerman, P.T. and Dykman, R.A. (1982). Automatic and effortful informationprocessing deficits in children with learning and attention disorders. *Topics in Learning and Learning Disabilities*, **2**, 12-22.
- Ackerman, P.T., Dykman, R.A. and Ogelsby, D.M.(1994). Visual event-related potnetial of dyslexic children to rhyming and nonrhyming stimuli. *Journal of Clinical and Experimental Neuropsychology*, **16**, 138-154.
- Adams, M.J. (1990). *Beginning to read: thinking and learning about print.* Cambridge, MA : MIT Press.
- Akshoomoff, N.A. and Courchesne, E. (1994). ERP evidence for a shifting attention deficit in patients with damage to the cerebellum. *Journal of Cognitive Neuroscience*, **6**, 388-399.
- Allen, G., Buxton, R.B., Wong, E.C. and Courchesne, E. (1997). Attentional activation of the cerebellum independent of motor involvement. *Science*, 275, 1940-43.
- American Psychiatric Association (1987). *Diagnostic and Statistical Manual of Mental Disorders, 3rd edition, Revised.* Washington DC: American Psychiatric Association.
- Anderson, J.R. (1983). *The architecture of cognition*. Harvard: Harvard University Press.
- Anderson, J.R. (1982). Acquisition of cognitive skill. *Psychological Review*, **89**, 369-406.
- Applebee, A.N. (1971). Research in reading retardation: two critical problems. Journal of child psychology and psychiatry, **12**, 91-113.
- Arnett, J.L. and DiLollo, V. (1979). Visual information processing in realtion to age and reading ability. *Journal of Experimental Child Psychology*, 27, 143-152.
- Atzmon, D. (1985). Positive effect of improving relative fusional vergence on reading and learning disabilities. *Binocular Vision*, 1, 39-43.
- Augur, J. (1985). Guidelines for teachers, parents and learners. In M.J. Snowling (Ed.), Children's written language difficulties: Assessment and management. London: Routledge. p147-169.

- Badal (1888). Archives d'Ophthalmologie, March-April, 1888. Cited in Hinshelwood (1895). Lancet, Dec.21st 1895.
- Baddeley, A.D. (1966). Short-term memory for word sequences as a function of acoustic semantic and formal similarity. *Quartely Journal of Experimental Psychology*, 18, 262-265.
- Baddeley, A.D., Thompson, N. and Buchanan, M. (1975). Word length and the structure of short term memory. *Journal of Verbal Learning and Verbal Behaviour*, **19**, 450-66.
- Badian, N.A. (1994). Preschool prediction: Orthographic and phonological skills and reading. *Annals of Dyslexia*, 44, 3-25.
- Baizer, J. and Glickstein, M. (1973). Role of cerebellum in prism adaptation. Journal of Physiology, 236, 34-35P
- Baizer, J. and Glickstein, M. (1994). Cerebellar lesion effects on prism adaptation in a Macaque monkey. *Journal of Physiology*, **476p**: 27p.
- Ballard, J.C. (1996). Computerized assessment of sustained attention: interactive effects of task demand, noise and anxiety. *Journal of Clinical and Experimental Neuropsychology*, 18, 864-882.
- Barkley, R.A. (1994). Delayed responding and attention deficit hyperactivity disorder: toward a unified theory. In D.K. Routh (Ed.), Disruptive behavior disorders in children: Essays in honor of Herbert Quay (p11-57). New York: Plenum. Cited in Chapter 4 in Attention Memory and Executive Function, G.R.Lyon and N.A.Krasnegor (eds.), Paul Brookes Publishing Co., Maple Press Company, York: Pennsylvania.
- Barkley, R.A. (1996). Critical Issues in Research on Attention. In G.R.Lyon and N.A.Krasnegor (eds) Attention, Memory and Executive Function. Paul Brookes Publishing Co. Maple Press Company, York, Pennsylvania.
- Bauer, R.H. and Emhert, J. (1984). Information processing in reading disabled and non-disabled readers. *Journal of Experimental Child Psychology*, 37, 271-281.
- Bedwell, C.H., Grant, R., and McKeown, J.R. (1980). Visual and ocular control anomalies in relation to reading difficulty, *British Journal of Educational Psychology*, **50**, 61-70.
- Bigelow, E.R. and McKenzie, B.E. (1985). Unstable ocular dominance and reading ability. *Perception*, 14, 329-335.
- Birnbaum, P. and Birnbaum, M.H. (1968). Binocular coordination as a factor in reading achievement. Journal of the American Optometric Association, 39, 48-57.

- Biscaldi, M., Fischer, B. and Aiple, F. (1994). Saccadic eye movements of dyslexic and normal reading children, *Perception*, 23, 45-64.
- Bishop, D.V.M. (1989a). Unstable vergence control and dyslexia a critique. British Journal of Ophthalmology, 73, 223-245.
- Bishop, D.V.M. (1989b). Unfixed reference, monocular occlusion and developmental dyslexia - a critique, *British Journal of Ophthalmology*, 73, 209-215.
- Bishop, D.V.M., Jancey, C. and Steel, A.M. (1979). Orthoptic status and reading disability. *Cortex*, 15, 659-66.
- Black, J.L., Collins, D.W.K., DeRoach, J.N., and Zubrick, S.R. (1984a).
 Dyslexia: Saccadic eye movements, *Perceptual and Motor Skills*, 58, 903-910.
- Black, J.L., Collins, D.W.K., DeRoach, J.N., and Zubrick, S.R. (1984b). A detailed study of sequential saccadic eye movements for normal and poor-reading children, *Perceptual and Motor Skills*, **59**, 423-434.
- Boder, E.M. (1973). Developmental dyslexia: a diagnostic approach based on three atypical reading-spelling patterns, *Developmental Medicine and Child Neurology*, 15, 663-687.
- Borcherding, B., Thompson, K., Kruesi, M., Bartko, J., Rapoport, J.L., Weingartner, H. (1988). Journal of Abnormal Child Psychology, 16, 333-45.
- Borsting, E., Ridder, W.H., Dudeck, K., Kelley, C., Matsui, L., and Motoyama, J. (1996). The presence of a magnocellular defect depends on the type of dyslexia, Vision Research, 36, 1047-1053.
- Bowers, P.G. and Wolf, M. (1993). Theoretical links among naming speed, precise timing mechanisms and orthographic skill in dyslexia. *Reading and Writing: An Interdisciplinary Journal*, **5**, 69-85.
- Brachacki, G.W.Z., Nicolson, R.I. and Fawcett, A.J. (1995). Impaired recognition of traffic signs in adults with dyslexia. *Journal of Learning Disabilities*, **28**, p297
- Bradley, L. and Bryant, P. (1978).Difficulties in auditory organisation as a possible cause of reading backwardness. *Nature*, **271**, 746-7.
- Bradley, L. and Bryant, P. (1983). Categorising sounds and learning to read: A causal connection. *Nature*, **301**, 419-421.
- Bradley, L. and Bryant, P. (1985). *Rhyme and Reason in Reading and Spelling*. Michigan.
- Brannan, J.R. and Williams, M.C. (1987). Allocation of visual attention in good and poor readers. *Perception and Psychophysics*, **41**, 23-28.

Breitmeyer, B.G. and Ganz, L. (1976). Implications of sustained and transient channels for theories of visual pattern masking, saccadic suppression, and information processing. *Psychological Review*, **83**, 1-36.

Broadbent, D.E. (1971). Decision and Stress. Academic Press, NewYork.

- Brown, B., Haegerstrom-Portnoy, G., Adams, A.J., Yingling, C.D., Galin, D., Herron, J. and Marcus, M. (1983). Predictive eye movements do not discriminate between dyslexic and control children. *Neuropsychologia*, 21, 121-8.
- Bryant, P. and Goswami, U. (1986). Strengths and weaknesses of the reading level design: a comment on Backman, Mamen, and Ferguson. *Psychological Bulletin*, 100, 101-103.
- Buzzelli, A.R. (1991). Stereopsis, Accommodative and Vergence Facility: Do they relate to Dyslexia?, *Optometry and Vision Science*, **68**, 842-846.
- Canon, L.K. (1971). Directed attention and maladaptive adaptation to displacement of the visual field. *Journal of Experimental Psychology*, **88**, 403-408.
- Cantwell, D.P. and Satterfield, J.H. (1978). Prevalence of academic achievement in hyperactive children. Journal of Pediatric Psychology, 3, 168-171. cited in Light, J.G., Pennington, B.F., Gilger, J.W., and DeFries, J.C. (1995).
 Reading disability and hyperactivity disorder: evidence for a common genetic etiology. *Developmental Neuropsychology*, 11, 323-335.
- Carroll, J.B. (1977). Developmental Parameters in Reading Comprehension. In J.T.Gutherie (ed). Cognition, Curriculum and Comprehension 1-15. Newark, DE: IRA
- Carpenter, R. (1988). Movements of the eyes (2nd ed.), London: Pion.cited in Enright (1991).
- Charcot (1892). Clinical lectures on diseases of the nervous system, vol iii. New Sydenham Society's Series. Cited in Hinshelwood (1895). Lancet, Dec.21st 1895.
- Clower, D.M., Hoffman, J.M., Votaw, J.R., Faber, T.L., Woods, R.P. and Alexander, G.E. (1996). Role of posterior parietal cortex in the recalibration of visually guided reaching. *Nature*, **383**, 618-621.
- Cohen, N.J. and Douglas, V.I. (1972). Characteristics of the orienting response in hyperactive and normal children. *Psychophysiology*, **9**, 238-245.
- Conners, C.K., March, J.S., Fiore, C., and Butcher, T. (1994). Information processing deficits in ADHD: Effect of stimulus rate and methylphenidate. Unpublished paper, Duke University, Durham, NC cited in Barkley 1996.

- Cornelissen, P, Bradley, L., Fowler, S. and Stein, J. (1991). What children see affects how they read. *Developmental Medicine and Child Neurology*, 33, 755-762.
- Cornelissen, P, Bradley, L., Fowler, S. and Stein, J. (1992). Covering one eye affects how some children read. *Developmental Medicine and Child Neurology*, 34, 296-304.
- Cornelissen, P., Evangelinou, E., Hansen, P.C. and Stein, J.F. (1997).
 Sensitivity to coherent motion predicts the pattern of children's reading errors. Paper presented at BDA 25th Anniversary Conference. Dyslexia: Biological Bases, Identification and Intervention. York, 1-4 April.
- Courchesne, E. and Allen, G. (1997). Prediction and preparation: Fundamaental functions of the cerebellum. *Learning and Memory*, **4**, 1-35.
- Cox, E. (1995). *Dyslexia and Anxiety*. Unpublished dissertation at Dept. of psychology, University of Sheffield.
- Deese, J. (1955). Some problems in the theory of vigilance. Psychological Review, 62, 359-68. Cited in Broadbent (1971). Decision and Stress.
 London: Academic Press.
- DeFries, J.C., Alarcón, M., and Olson, R.K. (1997). Genetic aetiologies of reading and spelling deficits: developmental differences. In C. Hulme and M. Snowling, (eds.) Dyslexia: Biology, Cognition and Intervention. London: England. Whurr.
- Denckla, M.B. and Rudel, R.G. (1976). Rapid 'Automatized' Naming (R.A.N.):
 Dyslexia differentiated from other learning disabilities. *Neuropsychologia*, 14, 471-479.
- DiLollo, V., Clark, C. and Hogben, J.H. (1988). Separating visible persistence from retinal after-images. *Perception and Psychophysics*, **44**, 363-368.
- DiLollo, V., Hansen, D. and McIntyre, J.S. (1983). Initial stages of visual information processing in dyslexia. *Journal of Experimental Psychology: Human Perception and Performance*, 9, 923-35.
- Dodgen, C.E. and Pavlidis, G.Th. (1989). Sequential, timing, rhythmic and eye movement problems in dyslexics. In Pavlidis (ed) Perspectives on Dyslexia
 Vol. 1: Neurology, Neuropsychology and Genetics. John Wiley and Sons: Chichester.
- Done, D.J. and Miles, T.R. (1978). Learning, Memory and Dyslexia. In M.M.Gruneberg, P.E. Morris and R.N.Sykes Practical Aspects of Memory. London: Academic Press.
- Dow, R.S. and Moruzzi, G. (1958). The physiology and pathology of the cerebellum. Minneapolis, University of Minnesota Press.

- Dunlop, P. (1972). Dyslexia: The orthoptic approach, Australian Journal of Orthoptics, 12, 16-20.
- Dunlop, P. (1976). The changing role of orthoptics in dyslexia, British Orthoptic Journal, 33, 22-28.
- Dunlop, Dunlop and Fenelon, B. (1973). Vision laterality analysis in children with reading disability: the results of new techniques of examination, *Cortex*, 9, 227-36
- Dykman, R.A., Ackerman, P.T. and Oglesby, D.M. (1979). Selective and Sustained Attention in hyperactive, learning-disabled and normal boys. Journal of Nervous and Mental Disease, 167, 288-297.
- Eden, G.F., VanMeter, J.W., Rumsey, J.M., Maisog, J.M., Woods, R.P. and Zeffiro, T.A. (1996). Abnormal processing of visual motion in dyslexia revealed by functional brain imaging. *Nature*, **382**, 66-9.
- Ehrlich, D.L. (1987). Near vision stress: vergence adaptation and accommodative fatigue, *Ophthalmic and Physiological Optics*, **7**, 353-357.
- Ellis, A.W., McDougall, S.J.P. and Monk, A.F. (1996a). Are dyslexics different?I. A comparison between dyslexics, reading age controls, poor readers and precocious readers. *Dyslexia*, 2, 31-58.
- Ellis, A.W., McDougall, S.J.P. and Monk, A.F. (1996b). Are dyslexics different?
 II. Individual differences among dyslexics, reading age controls, poor readers and precocious readers. *Dyslexia*, 2, 59-68.
- Ellis, N. (1981). Visual and name encoding in dyslexic children. *Psychological Research*, **43**, 201-18.
- Ellis, N. (1984). Longitudinal studies of spelling development. In G.D.A.Brown and N.C.Ellis, Handbook of Spelling: Theory, Process and Intervention. John Wiley and Sons: England.
- Ellis, N.C. and Miles, T.R. (1978). Visual Information Processing in Dyslexic Children. In M.M.Gruneberg, P.E. Morris and R.N.Sykes *Practical Aspects* of Memory. London: Academic Press
- Elterman, R.D., Abel, L.A., Daroff, R.B., Dell'Osso, L.F., and Bornstein, J.L. (1980). Eye movement patterns in dyslexic children. *Journal of Learning Disabilities*, **13**, 16-21.
- Enright, J.T. (1984). Changes in vergence mediated by saccades. Journal of *Physiology*, **350**, 9-31.
- Enright, J.T. (1991). Exploring the third dimension with eye movements: better than stereopsis, Vision Research, 31, 1549-62.
- Enright, J.T.(1996). Sequential Stereopsis: a simple demonstration, Vision Research, 36, 307-312.

- Eriksen, B.A. and Eriksen, C.W. (1974). Effects of noise letters upon the identification of a target letter in a non search task. *Perception and Psychophysics*, 16, 143-149.
- Evans, B.J.W. (1998). The underachieving child. Ophthalmic and Physiological Optics, 18, 153-159.
- Evans, B.J.W. and Drasdo, N. (1990). Review of ophthalmic factors in dyslexia, *Ophthalmic and Physiological Optics*, **10**, 123-132.
- Evans B.J.W., Drasdo, N. and Richards, I.L. (1994). Investigation of accommodative and binocular function in dyslexia, *Ophthalmic and Physiological Optics*, 14, 5-19.
- Evans B.J.W., Drasdo, N. and Richards, I.L. (1996). Dyslexia: the link with visual deficits, *Ophthalmic and Physiological Optics*, 16, 3-10.
- Eysenck, M.W. and Keane, M.T. (1990). Cognitive Psychology: A student's handbook, LEA, Hove: UK.
- Fawcett, A.J. (1990). *Dyslexia: A cognitive archictecture*. Unpublished Doctoral Thesis. University of Sheffield.
- Fawcett, A.J. and Nicolson, R.I. (1992). Automatisation deficits in balance for dyslexic children, *Perceptual and Motor Skills*, **75**, 507-529.
- Fawcett, A.J. and Nicolson, R.I. (1994). Dyslexia in Children: Multidisciplinary Perspectives, Harvester Wheatsheaf.
- Fawcett, A.J. and Nicolson, R.I. (1995a). Perisistance of phonological deficits, Reading and Writing, 7, 361-376.
- Fawcett, A.J. and Nicolson, R.I. (1995b). Persistent Deficits in motor skill of children with dyslexia. *Journal of Motor Behaviour*, **27**, 235-240.
- Fawcett, A.J. and Nicolson, R.I. (1996). *The Dyslexia Screening Test.* London: The Psychological Corporation (Europe).
- Fawcett, A.J., Nicolson, R.I. and Dean, P. (1996). Impaired performance of children with dyslexia on a range of cerebellar tasks. *Annals of Dyslexia*, 46, 259-282.
- Fawcett, A.J. and Nicolson, R.I. (1998a). Screening and Diagnosis of Dyslexia in Adults in the UK.
- Fawcett, A.J. and Nicolson, R.I. (1998b). The Dyslexia Adult Screening Test. Psychological Corporation.
- Felmingham, K.L. and Jakobson, L.S. (1995). Visual and visuomotor performance in dyslexic children. *Experimental Brain Research*, 106, 467-474.

- Finucci, J.M., Guthrie, J.T., Childs, A.L., Abbey, H. and Childs, B. (1976). The genetics of specific reading disability. Annals of Human Genetics, 50, 1-23.
- Fiez, J.A., Raichle, M.E., Miezin, F.M. and Petersen, S.E. (1995). PET studies of auditory and phonological Processing: Effects of stimulus characteristics and task demands, *Journal of Cognitive Neuroscience*, 7, 357-75.
- Fischer, B., Biscaldi, M., and Otto, P. (1993). Saccadic eye movements of dyslexic adult subjects, *Neuropsychologia*, **31**, 9, 887-906.
- Fischer, B. and Weber, H. (1990). Saccadic reaction times of dyslexic and agematched normal subjects. *Perception*, **19**, 805-818.
- Fisk, A.D. and Schneider, W. (1981). Controlled and automatic processing during tasks requiring sustained attention: A new approach to vigilance. *Human Factors*, **23**, 737-750.
- Fowler, M.S. and Stein, J.F. (1980). New evidence for "visual ambilaterality" in some dyslexics. *British Orthoptic Journal*, **37**, 11.
- Frank, J. and Levinson, H.N. (1973). Dysmetric dyslexia and dyspraxia: hypothesis and study. *Journal of American Academy of Child Psychiatry*, 12, 690-701.
- Frisby, J.P., Catherall, C., Porrill, J., and Buckley, D. (1997). Sequential stereopsis using high pass spatial frequency filtered images, *Vision Research*, 37, 3109-3116.
- Frisby, J.P., Taroyan, N., Buckley, D. and Porrill, J. (unpublished). Evidence against the isovergent saccades theory.
- Frith, U. (1985). Beneath the surface of developmental dyslexia. In K.E.Patterson, J.C.Marshall and M.Coltheart (Eds). Surface Dyslexia. Routledge and Kegan Paul.
- Frith, U. (1997). Brain, mind and behaviour in dyslexia. In C. Hulme and M.Snowling, (eds.) Dyslexia: Biology, Cognition and Intervention. London: England. Whurr.
- Galaburda, A.M. (1989). Ordinary and extraordinary brain development: anatomical variation in developmental dyslexia. *Annals of Dyslexia*, **39**, 67-80.
- Galaburda, A.M., Sherman, G.F., Rosen, G., Aboitiz, F. and Geschwind, N.(1985). Developmental dyslexia: four consecutive patients with cortical anomalies. Annals of Neurology, 18, 222-233.
- Goulandris, N., McIntytre, A., Snowling, M., Bethel, J.M. and Lee, J.P. (1998). A comparison of dyslexic and normal readers using orthoptic assessment procedures, *Dyslexia*, 4, 30-48.

- Grasby, P.M., Frith, C.D., Friston, K.J., Bench, C., Fracowiak, R.S.J., and Dolan, R.J. (1993). Functional mapping of brain areas implicated in auditory-verbal memory function. *Brain*, **116**, 1-20.
- Griffin, D.C., Walton, H.N. and Ives, V. (1974). Saccades as related to reading disorders. Journal of Learning Disabilities, 7, 52-58.
- Griffin, J.R. (1982). Binocular Anomalies: Procedures for Vision Therapy (2nd ed.). New York: Fairchild Publications, p345-62. cited in Kulp and Schmidt (1996a). Effect of oculomotor and other visual skills on reading performance: a literature review, Optometry and Vision Science, 73, 283-292.
- Gross-Glenn, K., Duara, R., Barker, W.W., Loewenstein, D., Chang, J.-Y.,
 Yoshii, F., Apicella, A.M., Pascal, S., Boothe, T., Sevush, S., Jallad,
 B.J., Novoa, L. and Lubs, H.A. (1991). Positron Emission Tomographic studies during serial word-reading by normal and dyslexic adults. *Journal of Clinical and Experimental Neuropsychology*, 15, 531-544.
- Gross-Glenn, K., Skottun, B.C., Glenn, W., Kushch, A., Lingua, R., Dunbar, M., Jallad, B., Lubs, H.A., Levin, B., Rabin, M., Parke, L.A. and Duara, R. (1995). Contrast sensitivity in dyslexia. Visual Neuroscience, 12, 153-63.
- Hagman, J.O., Wood, M.S., Buchsbaum, F., Tallal, P., Flowers, L. and Katz, W. (1992). Cerebral brain metabolism in adult dyslexic subjects assessed with positron emission tomography during performance of an auditory task. *Archives of Neurology*, 49, 734-9.
- Hallahan, D.P., Kauffman, J.M. and Ball, D.W. (1973). Selective attention and cognitive tempo of low achieving and high achieving sixth grade males. *Perceptual and Motor Skills*, 36, 579-583.
- Halperin, J.M. (1996). Conceptualising, describing and measuring comonents of attention: a summary. In G.R.Lyon and N.A.Krasnegor (eds) Attention, Memory and Executive Function. Paul Brookes Publishing Co. Maple Press Company, York, Pennsylvania.
- Halperin, J.M., Wolf, L.E., Pascualvaca, D.M., Newcorn, J.H., Healey, J.M., O'Brien, J.D., Morganstein, A. and Young, G. (1988). Differential assessment of attention and impulsivity in children. *Journal of the American Academy of Child and Adolescent Psychiatry*, 27, 326-329.
- Hamilton, C.R., and Bossom, J. (1964). Decay of prism aftereffects. Journal of Experimental Psychology, 67, 148-150
- Harris, C.S. (1963). Adaptation to Displaced Vision: Visual, Motor, or Proprioceptive Change? Science, 140, 812-3.

- Harris, C.S. (1974). Beware of the straight-ahead shift a nonperceptual change in experiments on adaptation to displaced vision, *Perception*, **3**, 461-476.
- Hartley, S., Lindley, C. and Nicolson, R.I. (in preparation). Semantic memory processing in children with developmental dyslexia: insights from semantic priming and lexical decision.
- Hayduk, S., Bruck, M. and Cavanagh, P. (1996). Low-level visual processing skills of adults and chidlren with dyslexia, *Cognitive Neuropsychology*, 13, 975-1015.
- Helmholtz (1866). Translated 1962. *Treatise on physiological optics*, translated and edited by Southall, 1962, vol 3 (Dover, New York).
- Helmuth, L.L., Ivry, R.B. and Shimizu, N. (1997). Preserved performance by cerebellar patients on tests of word generation, discrimination learning and attention. *Learning and Memory*, **3**, 456-474.
- Helverston, E.M. (1990). Scotopic Sensitivity Syndrome. Archives of Ophthalmology, 108, 1232-3.
- Hendriks, A.W. (1996). Vergence eye movements during fixations in reading. Acta Psychologica, 92, 131-151.
- Hess (1956) cited in Gross, R. (1990). Psychology: The science of mind and behaviour. Hodder and Stoughton.
- Hinshelwood (1895). Word Blindness and Visual Memory. *The Lancet*, p1564-70.
- Hogben, J.H., Rodino, Clark, C.D. and Pratt, C. (1995). A comparison of temporal integration in children with a specific reading disability and normal readers. Vision Research, 35, 2067-74.
- Howard, I.P., Anstis, T. and Lucia, H.C. (1974). The relative lability of mobile and stationary components in a visual-motor adaptation task. *Quartely Journal of Experimental Psychology*, **26**, 293-300.
- Howell, E.R., Smith, G.A. and Stanley, G. (1981). Reading disability and visual spatial frequency specific effects. *Australian Journal of Psychology*, **33**, 97-102.
- Hulme, C.(1997). Verbal working memory, phonological representations and learning to read. Paper presented at BDA 25th Anniversary Conference.
 Dyslexia: Biological Bases, Identification and Interventin. York, 1-4 April.
- Humphries, P., Kaufman, W.E., Galaburda, A.M. (1990). Developmental dyslexia in women: neuropathological findings in three patients. Annals of Neurology, 28, 727-38.
- Hung, G.K. (1989). Reduced vergence response velocities in dyslexics: a preliminary report, *Ophthalmic and Physiological Optics*, 9, 420-423.

- Hynd, G.W. and Hiemenz, J.R. (1997). Dyslexia and gyral morphology variation. In C. Hulme and M. Snowling, (eds.) *Dyslexia: Biology, Cognition and Intervention.* London: England. Whurr.
- Hynd, G.W., Semrud-Clikeman, M., Lorys, A.R. Novey, E.S. and Eliopulos, D. (1990). Brain Morphology in Developmental Dyslexia and Attention Deficit Disorder/ Hyperactivity, Arch Neurol., 47, 919-926
- Ivry, R.B. and Keele, S.W. (1989). Timing functions of the cerebellum. *Journal* of Cognitive Neuroscience, 1, 136-152.
- James, W. (1890). Principles of Psychology. New York: Holt.

Jenkins, I.H., Brooks, D.J., Nixon, P.D., Frackowiak, R.S.J. and Passingham, R.E. (1994). Motor sequence learning: a study with positron emission tomography. *Journal of Neuroscience*, 14, 3775-90.

Jerison, H.J. (1957). Performance on a simple vigilance task in noise and quiet. Journal of Acoust. Soc. Am., 29, 1163-1165. Cited in Broadbent (1971). Decision and Stress. London: Academic Press.

Jerison, H.J. (1959). Effects of noise on human performance. Journal of Applied Psychology, 43, 96-101. Cited in Broadbent (1971). Decision and Stress. London: Academic Press.

Johannes, S., Kussmaul, C.L., Münte, T.F. and Mangun, G.R. (1996). Developmental dyslexia: passive visual stimuluation provides no evidence for a magnocellular processing defect. *Neuropsychologia*, **34**, 1123-27.

Jorm, A.F. (1983).Specific reading retardation and working memory: A review. British Journal of Psychology, 74, 311-342.

Jorm, A.F., Share, D.L., Maclean, R. and Matthews, D. (1984). Phonological confusability in short-term memory for sentences as a predictor of reading ability. *British Journal of Psychology*, **75**, 393-400.

- Jorm, A.F., Share, D.L., Maclean, R. and Matthews, D. (1986). Cognitive factors at school entry predictive of specific reading retardation and general reading backwardness: A research note. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, **27**, 45-54.
- Jueptner, M., Rijntjes, M., Weiller, C., Faiss, J.H., Timmann, D., Mueller, S.P., and Diener, H.C. (1995). Localization of a cerebellar timing function using PET. Neurology, 45, 1540-1545.
- Kahane, J. and Auerbach, C. (1973). Effect of prior body experience on adaptation to visual displacement. *Perception and Psychophysics*, 13, 461-466.
- Kahneman, D. (1973). Attention and Effort. Englewood Cliffs, N.J.: Prentice-Hall.

- Kennedy, A. and Murray, W.S. (1991). The effects of flicker on eye movement control, *The Quartely Journal of Experimental Psychology*, **43A**, 79-99.
- Kitazawa, S., Kohno, T. and Uka, T. (1995). Effects of Delayed Visual Information on the Rate and Amount of Prism Adaptation in the Human. *The Journal of Neuroscience*, 15, 7644-7652.
- Korkman, M. and Pesonen, A. (1994). A comparison of neuropsychological test profiles of children with attention deficit hyperactivity disorder and/or learning disorder. *Journal of Learning Disabilities*, **27**, 383-392.
- Krupa, D.J., Thompson, J.K. and Thompson, R.F. (1993). Localization of a memory trace in the mammalian brain. *Science*, **260**, 989-91.
- Kulp, M.T., and Schmidt, P.P. (1996a). Effect of oculomotor and other visual skills on reading performance: a literature review, Optometry and Vision Science, 73, 283-292.
- Kulp, M.T., and Schmidt, P.P. (1996b). Visual predictors of reading performance in kindergarten and first grade children. *Optometry and Vision Science*, **73**, 255-262.
- Kussmaul (1877). Disturbances of Speech, Ziemessen's Cyclopaedia, 1877, vol xiv.
- LaBerge, D. and Samuels, S.J. (1974). Toward a theory of automatic information processing in reading. *Cognitive Psychology*, **6**, 293-323.
- Larsen, J.P., Höien, T., Lundberg, I. and Ödegaard, H.(1990). MRI evaluation of the size and symmetry of the planum temporale in adolescents with developmental dyslexia. *Brain and Language*, **39**, 289-301.
- Lashley, K.S. (1929). Brain mechanisms and intelligence. Chicago University Press, Chicago.
- Lavie, N. (1995). Perceptual load as a necessary condition for selective attention.
 Journal of Experimental Psychology: Human Perception and Performance,
 21, 451-468.
- Lavie, N. (1996). The roles of data versus resource limits in selective visual attention. *Presented to XXVI International Congress of Psychology* (August 1996), Montreal, Canada.
- Lazareff, J.A. and Castro-Sierra, E. (1996). Preoperative and postoperative analysis of visual and auditory memory in children with cerebellar tumours. *Child's Nerv Syst*, **12**, 81-86.
- Le, T.H., Pardo, J.V. and Hu, X. (1998). 4 T-fMRI study of nonsptial shifting of selective attention: cerebellar and parietal contributions. *Journal of Neurophysiology*, 79, 1535-48.

- Leiner, H.C., Leiner, A.L. and Dow, R.S. (1993). Cognitive and Language Functions of the Human Cerebellum. *Trends in neuroscience*, **16**, 444-6.
- Lennerstrand, G. and Ygge, J. (1992). Dyslexia: Ophthalmological aspects 1991. Acta Ophthalmologica, 70, 3-13.
- Levinson, H.N. (1988). The cerebellar-vestibular basis of learning disabilities in children, adolescenets and adults: hypothesis and study. *Perceptual and Motor Skills*, **67**, 983-1006.
- Liberman, A. and Mattingly, I. (1985). The motor theory of speech perception revisited. *Cognition*, 21, 1-36.
- Light, J.G., Pennington, B.F., Gilger, J.W., and DeFries, J.C. (1995). Reading disability and hyperactivity disorder: evidence for a common genetic etiology. *Developmental Neuropsychology*, **11**, 323-335.
- Livingstone, M.S., Rosen, G.D., Drislane, F.W., and Galaburda, A.M. (1991). Physiological and anatomical evidence for a magnocellular deficit in developmental dyslexia. *Proceeding of the National Academy of Sciences of the USA*, 88, 7943-7947.
- Locke, J.L. (1983). *Phonological acquisition and change*. New York: Academic Press
- Logan, G.D. (1981). Attention, automaticity and the ability to stop a speeded choice response. In J.Long and A.Baddeley (eds.), *Attention and Performance IX*. LEA: Hillsdale, New Jersey.
- Lovegrove, W. (1994). Visual Deficits in Dyslexia: Evidence and Implications. In A.J.Fawcett and R.I. Nicolson (eds.) *Dyslexia in Children: Multidisciplinary Perspectives*, Hemel Hempstead: England, Harvester Wheatsheaf.
- Lovegrove, W., Heddle, M. and Slaghuis, W. (1980). Reading disability: spatial frequency specific effects in visual information store. *Neuropsychologia*, 18, 111-115.
- Lovegrove, W., Martin, F., and Slaghuis, W. (1986). A theoretical and experimental case for a residual deficit in specific reading disability. *Cognitive Neuropsychology*, **3**, 225-267.
- Lundberg, I., Frost, J. and Peterson, O. (1988). Effects of an extensive program for stimulating phonological awareness in preschool children. *Reading Research Quartely*, 23, 263-84.
- Luria, S.M., McKay, C.L. and Ferris, S.H.(1973). Handedness and Adaptation to Visual Distortions of Size and Distance. *Journal of Experimental Psychology*, **100**, 263-269.

- Mackworth, N.H. (195)) Researches in the measurement of human performance.
 MRC Special Report Series No. 268, H.M. Stationary Office. Cited in Broadbent (1971). Decision and Stress. London: Academic Press.
- Maclagan, F. (1999). Unpublished thesis. Dept. of Psychology. University of Sheffield.
- Manly, B. F. J. (1991). Randomization and Monte Carlo Methods in Biology, London: Chapman and Hall.
- Manly, T., Robertson, I.H., Galloway, M. and Hawkins, K. (1999). The absent mind: further investigations of sustained attention to response. *Neuropsychologia*, **37**, 661-670.
- Marr, D. (1969). A Theory of Cerebellar Cortex. Journal of Physiology, 202, 437-70.
- Marr, D. and Poggio, T. (1979). A computational theory of human stereo vision. Proceedings of the Royal Society of London B, 204, 301-328.
- Marsh, G., Friedman, M., Welch, V. and Desberg, P. (1981). A cognitive developmental theory of reading acquisition. In G.E. MacKinnon and T.G.Waller (eds), *Reading Research: Advances in theory and practice* (vol.3). New York: Academic Press.
- Mason, M., Katz, L. and Wickland, D.A. (1975). Immediate sptial order memory and item memory in sixth grade children as a fucntion of reader ability. *Journal of Educational Psychology*, **67**, 610-616.
- McLoughlin, D. (1994). Adult dyslexia : assessment, counselling and training. London : Whurr, 1994.
- McPherson, W.B., Ackerman, P.T., Holcomb, P.J., and Dykman, R.A. (1997). Auditory and visual rhyme judgements reveal differences and similarities between normal and disabled readers. *Dyslexia*, **3**, 63-77.
- Meakin, K. (1995). Unpublished dissertation from Dept. of Psychology, University of Sheffield.
- Meares, O. (1980). Figure/ background, brightness/ contrast and reading disabilities. Visible Language, 14, 13-29. cited in Wilkins, A. (1995). Visual Stress. Oxford Science Publications: Oxford.
- Meyer, D.E. and Schvaneveldt, R.W. (1971). Facilitation in recognising pairs of words: evidence of a dependence between retrieval operations. *Journal of Experimental Psychology*, **90**, 227-234.
- Michael, R.L., Klorman, R., Salzman, L.F., Borgestedt, A.D. and Dainer, K.B. (1981). Normalizing effects of methylphenidate on hyperactive children's vigilance performance and evoked potentials. *Psychophysiology*, 18, 665-677. cited in Schacher et al.

- Michel (1892). Berger: Les Maladies des Yeux dans leurs Rapports avec la Pathologie Generale. Paris, 1892. Cited in Hinshelwood (1895). Lancet, Dec.21st 1895.
- Mierzejewski (1892). Berger: Les Maladies des Yeux dans leurs Rapports avec la Pathologie Generale. Paris, 1892. Cited in Hinshelwood (1895). Lancet, Dec.21st 1895.
- Milberg, V.W., Whitman, R.D. and Galpin, R. (1981). Selective attention and laterality in good and poor readers. *Cortex*, **17**, 571-82.
- Miles, T.R. (1983). Dyslexia: The Pattern of Difficulties. London, England: Granada.
- Miles, T.R. and Miles, E. (1990). Dyslexia: A Hundred Years On. Open University Press: Buckingham.
- Miles, T.R. (1994). A proposed taxonomy and some consequences. In
 A.J.Fawcett and R.I. Nicolson (eds.) Dyslexia in Children:
 Multidisciplinary Perspectives, Hemel Hempstead: England, Harvester
 Wheatsheaf.
- Miles, T.R. (1996). Do dyslexic children have IQs? Dyslexia, 2, 175-178.
- Miles, T.R. and Wheeler, T. (1974). Responses of dyslexic and non-dyslexics to tachistoscopically presented digits. *IRCS Medical Science*, 5, 149. Cited in Thomson and Wilsher (1978). Some aspects of memory in dyslexics and controls. In M.M.Gruneberg, P.E. Morris and R.N.Sykes *Practical Aspects* of *Memory*. London: Academic Press.
- Mody, M., Studdert-Kennedy, M. and Brady, S. (1997). Speech perception deficits in poor readers: Auditory processing or phonological coding? *Journal* of Experimental Child Psychology, **64**, 199-231.
- Moores, E. (1995). Experiment to investigate auditory and visual temporal processing deficits in dyslexic children. Unpublished dissertation from University of Sheffield Psychology Dept.
- Moores, E., Frisby, J.P., Buckley, D., Reyonolds, E. and Fawcett, A. (1998).
 Vergence control across saccades in dyslexic adults, *Ophthalmic and Physiological Optics*, 18, 452-462.
- Morais, J. (1991). Metaphonological abilities. In M.Snowling and M.Thomson (eds). Dyslexia: Integrating Theory and Practice, Whurr: London.
- Närhi, V. and Ahonen, T. (1995). Reading disability with or without attention deficit hyperactivity disorder: do attentional problems make a difference? *Developmental Neuropsychology*, **11**, 337-349.
- Navon, D. (1984). Resources a theoretical soup stone? *Psychological Review*, **91**, 216-234.

- Nicolson, R.I. (1981). The relationship between memory span and processing speed. In M.P.Connor, J.P.Das and N.O'Connor, *Intelligence and Learning*. Plenum Press: New York.
- Nicolson, R.I. (1996). Developmental dyslexia: past, present and future. *Dyslexia*, **2**, 190-207.
- Nicolson, R.I. and Fawcett, A.J. (1990). Automaticity: A new framework for dyslexia research? *Cognition*, **35**, 159-182.
- Nicolson, R.I. and Fawcett, A.J. (1993a). Children with dyslexia show deficits on most primitive skills. Proceedings of the 15th Annual Conference of the Cognitive Science Society. LEA.
- Nicolson, R. I. and A. J. Fawcett (1993b). Children with Dyslexia Automatize Temporal Skills More Slowly. *Temporal Information Processing and the Nervous System* Eds. Tallal. New York, Annals New York Academy of Sciences.
- Nicolson, R.I. and Fawcett, A.J. (1993c). Children with dyslexia acquire skill more slowly. Proceedings of the 15th Annual Conference of the Cognitive Science Society. LEA.
- Nicolson, R.I. and Fawcett, A.J. (1994). Reaction times and dyslexia. Quarterly Journal of Experimental Psychology, 47A, 29-48.
- Nicolson, R.I. and Fawcett, A.J. (1994). Comparison of deficit severity across skills: towards a taxonomy of dyslexia. In A.J.Fawcett and R.I. Nicolson (eds.) Dyslexia in Children: Multidisciplinary Perspectives, Hemel Hempstead: England, Harvester Wheatsheaf.
- Nicolson, R.I. and Fawcett, A.J. (1995). Dyslexia is more than a phonological disability. *Dyslexia*, 1, 19-36.
- Nicolson, R.I., Fawcett, A.J. and Dean, P. (1995). Time estimation deficits in developmental dyslexia: Evidence of cerebellar involvement. *Proceedings of the Royal Society of London*, **259**, 43-47.
- Nicolson, R.I. and Fawcett, A.J. (1996). Dyslexia in adults: New developments in diagnosis and screening, *Journal of Research in Reading*, **20**, 77-83.
- Nicolson, R.I., Fawcett, A.J., Berry, E.L., Jenkins, H., Dean, P. and Brooks, D.J. (1999). Cerebellar function is impaired in dyslexia: A PET activation study *The Lancet.*, 353, 1162-7.
- Noelker, R.W. and Schumsky, D.A. (1973). Memory for sequence, form and position as related to the identification of reading retardates. *Journal of Educational Psychology*, **64**, 22-25.

Norman, D.A. (1982). Learning and Memory. San Francisco, CA: Freeman.

- Norman, D.A. and Bobrow, D.G. (1975). On data-limited and resource-limited processes. *Cognitive Psychology*, **7**, 44-64.
- Nuechterlein, K. (1982). Signal detection in vigilance tasks and behavioral attributes among offspring of schizophrenic mothers and among hyperactive children. *Journal of Abnormal Psychology*, **92**, 4-28.
- Nuechterlein, K., Parasuraman, R. and Jiang, Q. (1983). Visual sustained attention: image degradation produces rapid sensitivity decrement over time. *Science*, **220**, 327-329.
- Ogle, K.N. (1956). Stereoscopic acuity and the role of convergence. Journal of the Optical Society of America, 46, 269-273.
- Ogle, K.N. (1962). Spatial localization through binocular vision. In H. Davson(ed.) The Eye (vol.4). New York: Academic press.
- Ollman, R. (1977). Choice reaction time and the problem of distinguishing task effects from strategy effects. In S.Dornic (ed.) Attention and Performance VI:: Proceedings of the sixth international symposium on attention and performance. LEA: Hillsdale, New Jersey.
- Olson, R.K., Kliegl, R. and Davidson, B.J. (1983). Dyslexic and normal readers' eye movements, *Journal of Experiemntal Psychology: Human Perception* and Performance, 9, 816-825.
- Orton, S.T. 1937). Reading, Writing and Speech Problems in Children. New York, W.W. Norton. Cited in Miles and Miles (1990). Dyslexia: A Hundred Years On. Open University Press: Buckhingham.
- Orton Dyslexia Society (1995). Definition of dyslexia: report from committee of members. *Perspectives*, **21**, 16-17.
- Pardo, J.V., Fox, P.T. and Raichle, M.E. (1991). Localization of a human system for sustained attention by positron emission tomography. *Nature*, 349, 61-64.
- Park, G.E., and Burri, C. (1943). The effect of eye abnormalities on reading difficulty, *Journal of Educational Psychology*, **34**, 420-430.
- Parasuraman, R. (1979). Memory load and event rate control sensitivity decrements in sustained attention. *Science*, **205**, 924-927.
- Parasuraman (1985). Sustained attention: a multifactorial approach. In M.I.Posner and O.S.M.Marin (eds.)*Attention and Performance XI*, LEA: Hillsdale, New Jersey.
- Paulesu, E., Frith, U., Snowling, M., Gallagher, A., Morton, J., Frackowiak, R.S.J. and Frith, C.D.(1996). Is developmental dyslexia a disconnection syndrome? Evidence from PET scanning. *Brain*, 119, 143-157.

- Pavlidis, G.Th. (1981). Do eye movements hold the key to dyslexia? Neuropsychologia, 19, 57-64.
- Pavlidis, G.Th. (1985a). Eye movements in dyslexia: their significance. *Journal of Learning Disabilities*, **18**, 42-50.
- Pavlidis, G.Th. (1985b). Eye movement differences between dyslexic, normal and retarded readers while sequentially fixating digits, Amercian Journal of Optometry and Physiological Optics, 62, 820-832.
- Pavlidis, G.Th. (1985c). Erratic eye movements and dyslexia: factors determining their relationship. *Perceptual and Motor Skills*, **60**, 319-322.
- Pearson, D.A. and Lane, D.M. (1990). Reorientation in hyperactive and nonhyperactive children: Evidence for developmentally immature attention? In J.T.Enns (Ed.) *The Development of Attention: Research and Theory*. Elsevier Science: Holland.
- Pelham, W. (1979). Selective attention deficits in poor readers? Dichotic listening, speeded classification, and auditory and visual central and incidental learning tasks. *Child Development*, 50, 1050-61.
- Pennington, B.F., Groisser, D. and Welsh, M.C. (1993). Contrasting cognitive deficits in attention deficit hyperactivity disorder versus reading disability. *Developmental Psychology*, 29, 511-523.
- Perfetti, C.A. and Goldman, S.R. (1976). Discourse memory and reading comprehension skill. Journal of Verbal Learning and Verbal Behaviour, 14, 33-42.
- Posner, M.I. and Boies, S.J. (1971). Components of Attention, *Psychological Review*, **78**, 391-408.
- Posner, M.I. (1978). Chronometric Explorations of Mind. Oxford University Press: Oxford.
- Posner, M.I., Nissen, M.J. and Ogden, W.C. (1978). Attended and unattended processing modes: the role of set for spatial location. In H.J. Pick and I.J. Saltzmann (eds.), *Modes of perception*, Hillsdale, NJ: Erlbaum.
- Posner, M.I. and Snyder, C.R. (1974). Attention and cognitive control. In R.L.Solso (Ed.), Information processing and cognition: The Loyola Symposium. Hillsdale, NJ: Erlbaum.
- Posner, M.I., Snyder, C.R. and Davidson, B.J. (1980). Attention and the detection of signals, *Journal of Experimental Psychology: Human Perception* and Performance, **3**, 201-211.
- Procter, A. (1994). Unpublished dissertation. Department of Psychology, University of Sheffield.

- Rabbitt, P.M. (1979). How old and young subjects monitor and control responses for accuracy and speed. *British Journal of Psychology*, **70**, 305-311.
- Rack, J.P. (1994). Dyslexia: The Phonological Deficit Hypothesis. In A.J.Fawcett and R.I. Nicolson (eds.) Dyslexia in Children: Multidisciplinary Perspectives, Hemel Hempstead: England, Harvester Wheatsheaf.
- Reason, J.T. (1979). Actions not as planned. In G.Underwood and R.Stevens (eds.), *Aspects of Conciousness*. London: Academic Press.
- Ritchie, D. and Aten, J.L. (1976). Auditory retention of nonverbal and verbal sequential stimuli in children with reading disabilities. *Journal of Learning Disabilities*, **9**, 312-318.
- Robertson, I.H., Manly, T., Andrade, J., Baddeley, B.T. and Yiend, J. (1997).
 'Oops!': Performance correlates of everyday attentional failures in traumatic brain injured and normal subjects. *Neuropsychologia*, 35, 747-758.
- Rock, I., Goldberg, J. and Mack, A. (1966). Immediate Correction and Adaptation Based on Viewing a Prismatically Displaced Scene. *Perception and Psychophysics*, 1, 351-354.
- Rosvold, H., Mirsky, A., Sarason, I., Bransome, E. and Beck, L. (1956). A continuous performance test of brain damage. *Journal of Consulting Psychology*, 20, 343-350.
- Rovamo, J., Virsu, V. and Nasaren, R. (1978). Cortical magnification factor predicts the photopic sensitivity contrast sensitivity of peripheral vision, *Nature*, **71**, 54-56. Cited in Frisby, J.P., Catherall, C., Porrill, J., and Buckley, D. (1997). Sequential stereopsis using high pass spatial frequency filtered images, *Vision Research*, **37**, 3109-3116.
- Rudel, R.G. (1985). The definition of dyslexia: Language and motor deficits. In F.H. Duffy and N. Gsechwind (eds.) Dyslexia: a neuroscientific apporach to clinical evaluation. Boston, MA: Little Brown.
- Schacher, R., Logan, G., Wachsmuth, R. and Chajcyzk, D. (1988). Attaining and maintaining preparation: A comparison of attention in hyperactive, normal and disturbed control children. *Journal of Abnormal Child Psychology*, 16, 361-378.
- Schacher, R.J, Tannock, R., and Logan, G. (1993). Inhibitory control, impulsiveness and attention deficit hyperactivity disorder, *Clinical Psychology Review*, **13**, 721-739. cited in Barkley (1996)
- Schneider, W. and Shiffrin, R.M. (1977). Controlled and automatic human information processing:1. Detection, search and attention. *Psychological Review*, 84, 1-66.

- Senf, G.M. and Freundl, P.C. (1972). Sequential auditory and visual memory in learning disabled children. Paper presented at the meeting of the American Psychological Association, Honolulu.
- Sergeant, J. and van der Meere, J.J. (1988). What happens after a hyperactive commits an error. *Psychiatry Research*, 24, 157-64. cited in van der Meere et al (1992). A motor presetting study in hyperactive, learning disabled and control children, *Journal of Child Psychology and Psychiatry*, 33, 1347-54.
- Shankweiler, D., Liberman, I.Y., Mark, L.S., Fowler, C.A. and Fischer, F.W. (1979). The speech code and learning to read. *Journal of Experimental Psychology: Human Learning and Memory*, 5, 531-545.
- Shaywitz, S.E., Fletcher, J.M. and Shaywitz, B.A. (1994). Issues in the definition and classification of attention deficit disorder. *Topics in Language Disorders*, 14, 1-25.
- Shaywitz, B.A. and Shaywitz, S.E. (1994). Measuring and analyzing change. In G.R.Lyon, Frames of Reference for the Assessment of Learning Disabilities: New Views on Measurement Issues, Paul H. Brookes: Maryland.
- Shiffrin, R.M. and Schneider, W. (1977). Controlled and automatic human information processing II. Perceptual learning, automatic attending, and a general theory. *Psychological Review*, **84**, 127-190.
- Siegel, L.S. (1989). IQ is irrelevant in the definition of learning disabilities. Journal of Learning Disabilities, 22, 469-478.
- Siegel, L.S. (1992). An evaluation of the discrepancy definition of dyslexia. Journal of Learning Disabilities, 25, 618-629.
- Siegel, L.S. and Himel, N. (1998). Socioeconomic status, age and the classification of dyslexics and poor readers: the dangers of using IQ scores in the definition of reading disability. *Dyslexia*, **4**, 90-104.
- Simons, H.D. and Grisham, J.D. (1987). Binocular anomalies and reading problems. Journal of the American Optometric Association, 58, 578-587.
- Smith-Spark, J. (1997). Dynamic memory impairments in university students with dyslexia. Poster presented at 4th World Congress on Dyslexia, 23-26 September 1997, Greece.
- Snowling, M., Goulandris, N., Bowlby, M. and Howell, P. (1986). Segmentation and speech perception in relation to reading skill: a developmental analysis. *Journal of Experimental Child Psychology*, 41, 489-507.
- Snowling, M. (1987). Dyslexia: A Cognitive Developmental Perspective. Blackwell, Oxford.

- Spring, C. and French, L. (1990). Identifying children with specific reading disabilities from listening and reading discrepancy scores. *Journal of Learning Disabilities*, 23, 53-58.
- Stanley, G., Smith, G.A., and Howell, E.A. (1983). Eye-movements and sequential tracking in dyslexic and control chilren. *British Journal of Psychology*, 74, 181-187.
- Stanovich, K.E. (1988). Explaining the differences between the dyslexic and the garden variety poor reader: The phonological-core variable-difference model. Journal of Learning Disabilities, 21, 590-612.
- Stanovich, K.E. (1991). The theoretical and practical consequences of discrepancy definitions of dyslexia. In M.Snowling and M.Thomson (eds). *Dyslexia: Integrating Theory and Practice*, Whurr: London.
- Stanovich, K.E. (1996). Toward a more inclusive definition of dyslexia. *Dyslexia*, 2, 154-166.
- Stanovich, K.E., Siegel, L.S., and Gottardo, A. (1997). Progress in the search for dyslexia subtypes. In C.Hulme and M.Snowling (eds). *Dyslexia: Biology, Cognition and Intervention.*. Whurr: London.
- Stein, J.F. (1994). Visuospatial perception in disabled readers. In D.M.Willows, R.S.Kruk, and E.Corcos (eds) Visual Processes in Reading and Reading Disabilities. Lawrence Erlbaum Associates, Hillsdale, New Jersey, USA, pp 287-310.
- Stein, J.F. and Fowler, M.S. (1993), Unstable binocular control in dyslexic children, *Journal of Research in Reading*, 16, 30-45.
- Stein, J.F., Riddell, P.M., and Fowler, S. (1986). The Dunlop test and reading in primary school children, *British Journal of Ophthalmology*, **70**, 317-320.
- Stein, J.F., Riddell, P.M., and Fowler, S. (1987). Fine binocular control in dyslexic children, *Eye*, 1, 433-438.
- Stein, J.F., Riddell, P.M., and Fowler, S. (1988). Disordered vergence control in dyslexic children, *British Journal of Ophthalmology*, **72**, 162-166.
- Stein, J.F. and Walsh, V. (1997). To see but not to read; the magnocellular theory of dyslexia. *Trends in Neuroscience*, **20**, 147-152.
- Stevenson, J., Pennington, B.F., Gilger, J.W., DeFries, J.C. and Gillis, J.J. (1993). Hyperactivity and spelling disability: testing for shared genetic aetiology. *Journal of Child Psychology and Psychiatry*, 34, 1137-1152.
- Studdert-Kennedy, M. and Mody, M. (1995). Auditory temporal perception deficits in the reading impaired: a critical review of the evidence, *Psychonomic Bulletin and Review*, **2**, 508-514.

- Swets, J. A. (1964). Signal detection and recognition by human observers: Contemporary readings. New York; London: Wiley.
- Sykes, D.H., Douglas, V.I., and Morgenstern, G. (1973). Sustained attention in hyperactive children. Journal of Child Psychology and Psychiatry, 14, 213-220.
- Tallal, P. (1980). Auditory temporal perception, phonics and reading disabilities in children. *Brain Lang.*, **9**, 182-198.
- Tallal, P., Miller, S.L., Bedi, G., Dyma, G., Wang, X., Nagarajan, S.S., Schreiner, C., Jenkins, W.M. and Merzenich, M.M. (1996). Language comprehension in language-learning impaired children improved with acoustically modified speech. *Science*, 272, 81-84.
- Tallal, P., Miller, S. and Fitch, R.H. (1993). Neurobiological Basis of Speech: A Case for the preeminence of temporal processing. Annals of the New York Academy of Sciences, 682, 27-47.
- Tallal, P. and Piercy, M. (1973a). Defects of non-verbal auditory perception in children with developmental aphasia. *Nature*, **241**, 468-69.
- Tallal, P. and Piercy, M. (1973b). Developmental aphasia: Impaired rate of nonverbal processing as a function of sensory modality. *Neuropsychologia*, 11, 389-98.
- Tallal, P., Sainburg, R.L. and Jernigan, T. (1991). The Neuropathology of developmental dysphasia: behavioural, morphological, and physiological evidence for a pervasive temporal processing disorder. *Reading and Writing: An Interdisciplinary Journal*, 3, 363-377.
- Tallal, P. and Stark, R. (1982). Perceptual / motor profiles of reading impaired children with ot without concomitant oral language deficits. Annals of Dyslexia, 32, 163-176.
- Tallal, P., Stark, R. and Mellitus, D.(1985). The relationship between auditory temporal analysis and receptive language development: Evidence from studies of developmental language disorder. *Neuropsychologia*, 23, 527-34.
- Tarver, J.G.and Hallahan, D.P. (1974). Attention deficits in children with learning disabilities: a review. Journal of Learning Disabilities, **17**, 560-72.
- Tarver, J.G., Hallahan, D.P., Kauffman, J.M. and Ball, D.W. (1976). Verbal rehearsal and selective attention in children with learning disabilities. *Journal of Experimental Child Psychology*, **22**, 375-385.
- Tate, M.W. and Clelland, R.C. (1957). Nonparametric and Shortcut Statistics, Danville, III: Insterstate. cited in Enright, J.T. (1991). Exploring the third dimension with eye movements: better than stereopsis, Vision Research, 31, 1549-62.

- Torgesen, J.K. (1979). Performance of reading disabled children on serial memory tasks: a selective review of recent research. *Reading Research Quartely*, 1, 57-87.
- Torgesen, J. and Goldman, T. (1977). Verbal rehearsal and short-term memory in reading-disabled children. *Child Development*, **48**, 56-60.
- Treisman, A. (1991). Search, similarity and integration of features between and within dimensions, Journal of Experimental Psychology: Human Perception and Performance, 17, 652-76.
- Van der Meere, J., Vreeling, H.J. and Sergeant, J.A. (1992). A motor presetting study in hyperactives, learning disabled and control children. *Journal of Child Psychology and Psychiatry*, 34, 1347-1354.
- Vellutino, F.R. (1979). *Dyslexia: Theory and Research*. Cambridge, MA: MIT Press.
- Wagner, R.K. and Torgeson, J.K. (1987). The nature of phonological processing and its causal role in the acquisition of reading skills. *Psychological Bulletin*, 101, 192-212.
- Warren, D.H. and Platt, B.B. (1974). The Participant: A neglected factor in recombination research. *Perception*, **3**, 421-438.
- Watson, C. and Willows, D.M. (1993). Evidence for a visual-processing-deficit subtype among disabled readers. In D.M.Willows, R.S.Kruk, and E.Corcos (eds) Visual Processes in Reading and Reading Disabilities.
 Lawrence Erlbaum Associates, Hillsdale, New Jersey, USA.
- Watten, R.G. (1994). Reinvention of visual fatigue: accumulation of scientific knowledge or neglect of scientific history? Ophthalmic and Physiological Optics, 14, 428-432.
- Wechsler, D. (1976). Wechsler Intelligence Scale for Children 3rd edition(WISC III). Sidcup, UK; The Psychological Corporation, Europe.
- Wechsler (1992). Manual for the Wechsler Intelligence Scale for Children Third Edition UK. The Psychological Corporation Limited, UK.
- Weiner, M.J., Hallett, M. and Funkenstein, H.H. (1983). Adaptation to lateral displacement of vision in patients with lesions of the CNS. *Neurology*, 33, 766-72.
- Weichselgartner, E. and Sperling, G. (1987). Dynamics of automatic and controlled visual attention, *Science*, 238, 778-80.
- Welch, R.B. (1974). Research on Adaptation to rearranged vision: 1966-74. *Perception*, **3**, 367-392.
- Welch, R.B. and Goldstein, G. (1972). Prism adaptation and brain damage. Neuropsychologia, 10, 387-394.

Welch, R.B. and Rhoades, R.W. (1969). The manipulation of informational feedback and its effects upon prism adaptation. *Canadian Journal of Psychology*, 23, 415-428.

Whyte, J. (1994). Attentional Processes and Dyslexia. Cognitive Neuropsychology, 11, 99-116.

Wildsoet, C.F. and Cameron, K.D. (1985). The effect of illumination and foveal fusion lock on clinical fixation disparity measurements with the sheedy disparometer, *Ophthalmic and Physiological Optics*, 5, 171-178.

Wilkins, A. (1995). Visual Stress. Oxford Science Publications: Oxford.

Wilkinson, D.A. (1971). Visual-Motor Control Loop: A linear system? Journal of Experimental Psychology, 89, 250-257.

Williams, R.W. and Herrup, K. (1988). The control of neuron number. Annual Review of Neuroscience, 11, 423-453.

Winn, B., Gilmartin, B., Sculfor, D.L., and Bamford, J.C. (1994). Vergence Adaptation and Senescence. *Optometry and Vision Science*, **71**, 797-800.

Wolff, P., Michel, G.F., Ovrut, M. and Drake, C. (1990). Rate and timing precision of motor coordination in developmental dyslexia. *Developmental Psychology*, 26, 349-59.

World Federation of Neurology (1968). Report of research group on dyslexia and world illiteracy. Dallas: WFN.

Wright, W.D. (1951). A reply to Ogle. Proceedings of the Physical Society of London, B, 66, 514-4.

Yamaguchi, S., Tschuchiya, H. and Kobayashi, S. (1998). Visuospatial attention shift and motor response in cerebellar disorders. *Journal of Cognitive Neuroscience*, 10, 95-107.

Yap, R.L. and van der Leij, A. (1994a). Testing the automatisation deficit hypothesis of dyslexia via a dual task paradigm. *Journal of Learning Disabilities*, 27, 660-65.

- Yap, R.L. and van der Leij, A. (1994b). Automaticity deficits in word reading. In A.J.Fawcett and R.I. Nicolson (eds.) Dyslexia in Children: Multidisciplinary Perspectives, Hemel Hempstead: England, Harvester Wheatsheaf.
- Yekta, A.A., Jenkins, T. and Pickwell, D. (1987). The clinical assessment of binocular vision before and after a working day, *Ophthalmic and Physiological Optics*, 7, 349-352.
- Ygge, J., Lennerstrand, G., Wijecoon, S., Rydberg, A. and Pettersson, B.M. (1993). Oculomotor functions in a Swedish population of dyslexic and normally reading children. Acta Ophthalmologica, 71, 10-21.

Zangwill, O.L.and Blakemore, C. (1972). Dyslexia: reversal of eye-movements during reading. *Neuropsychologia*, **10**, 371-373.

Appendices

NB. The first two numbers in the appendix numbering relate to the chapter and section in which they are referred to.

Chapter 1 no appendices

Chapter 2

2.3.1	Meakin (1995) re-analysis	240
2.4.1	Straight Ahead Shift Experiment. ANOVA tables	241
2.4.2	ANOVA of consistency of measurements	247
	in the SAS expt	
2.4.3	Psychometric data of participants	250
2.5.1	Example data sheet from main prism	251
	adaptation expt	
2.5.2	Main experiment ANOVA tables	
	(i) initial accuracy	253
	(ii) adaptation	259
	(iii) intermanual transfer	261
	(iv) negative after effect	263
Chap	ter 3	
3.3.1	The method of diagnosing dyslexia in	266
	adults	
3.3.2	Expt 1 ANOVAs and RANOVAs without	267
	exclusions (including raw data for main	
	analyses)	
3.3.3	Expt 1 ANOVAs and RANOVAs with	272
	exclusions	
3.3.4	Expt 1 Simultaneous condition	276
	calculations	
3.4.1	Expt 2: ANOVAs and RANOVAs no	278
	exclusions (inc raw data for main analyses	
3.4.2	Expt 2: ANOVAs and RANOVAs with	285
	exclusions	• • •
3.4.3	Eye movement counts.(raw data and	291
251	analyses Pooled data: no exclusions	204
3.5.1	Pooled data: with exclusions	294
3.5.2	Pooled data: with exclusions	295
Char	ton A	
Chap	Shapes used	007
4.4.1	Shapes used Software differences from Akshoomoff	296
4.4.2	Soliwale unterclices nom Aksnoomon	296

	and Courchesne (1994)	
4.4.3	ANOVA tables for RT and d prime	297
	analyses (inc raw data)	

Chap	ter 5	
5.1.1	Accuracy ANOVAs (inc raw data)	303
5.1.2	Bias ANOVAs (inc raw data)	308
5.1.3	Reaction Time ANOVAs. (inc raw data)	310
5.1.4	Post-hoc analyses	314
5.1.5	Probability analyses	318
Chap	ter 6	
6.2.1	All analyses and raw data	321
6.2.2	Personal communication from Dr. Tom Manly	326
Chap	ter 7	
7.1	Comparison across 3 attention experiments	328

Appendix 2.3.1: Meakin (1995) re-analysis

AN	OVA tab	le of	poin	ting errors	made to	the init	ial thr	ee t	arge
posi	tions by	the	older	dyslexic	and contr	ol group	os onl	y.	-
r				•		0 1			
_									
•••	III Sums of Sq	uares		• • • •					
Sou			df	Sum of Squares	Mean Square		P-Value	G-G	H-F
Grou	<u> </u>		1 18	286.017 548.167	286.017 30.454	9.392	.0067		
	ject(Group)		10	346.900	173.450	8.881	.0007	.0023	.0014
_	et position et position * G	roun	2	90.633	45.317	2.320	.1128		.1217
_	et position * Su		36	703.133	19.531	2.020	.,120	.1204	.1217
	endent: target p						L		L
	······································								
Teble	of Epsilon Fac	tors for	df Adius	tment					
	ndent: target p								
		G-G Eps	ilon H-	F Epsilon					
taroe	t position		58	.859					
	· •								
	interacti Effect: ti		eition *	Group					
	Depende								
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	10 -		~				F		
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sitio	8 -						t		
Cell Means of target position	<u>[</u>						[o	one	
arge	6 -							two	
oft	4						[▲	three	
SUE			<u>ه</u>				1		
Me	2 -				Δ		F		
Test State	4				\mathbf{i}		ł		
0	o 				\rightarrow	······	+		
	4				=		ł		
	· 2		slexic		Contro				
		Uys	DIGYIC		Contro	1			
				Group					

Appendix 2.4.1: Straight Ahead Shift Experiment.

ANOVA tables of investigated effects. It will probably be necessary to read the main text in order to understand the dependent variable analysed in each case.

a) ANOVA table investigating effects of age and group on positionning of bead in dark clear condition vs. initial prisms in the dark condition.

Type III Sums of Squares

Source	đf	Sum of Squares	Mean Square	F-Value	P-Value	G-G	H-F
age	1	3.785	3.785	.647	.4291		
group	1	5.609	5.609	.959	.3373		
age * group	1	2.024	2.024	.346	.5619		
Subject(Group)	24	140.412	5.851				
cond	1	517.697	517.697	88.487	.0001	.0001	.0001
cond * age	1	3.785	3.785	.647	.4291	.4291	.4291
cond * group	1	5.609	5.609	.959	.3373	.3373	.3373
cond * age * group	1	2.024	2.024	.346	.5619	.5619	.5619
cond * Subject(Group)	24	140.412	5.851				

Dependent: condition a

Table of Epsilon Factors for df Adjustment Dependent: condition a

cond

G-G Epsilon H-F Epsilon 1.000 1.130

NOTE: Probabilities are not corrected for values of epsilon greater than 1.

241

Type III Sums of Squares	iignt	compared	ιιοιπ	the dar	:к.					
Source	df	Sum of Squares	Mean Square	F-Value	P-Value	G-G	H-F			
age	1	2.550	2.550	.345	.5625					
group	1	1.835	1.835	.248	.6229					
age * group	1	7.790	7.790	1.053	.3149					
Subject(Group)	24	177.465	7.394							
cond	1	525.242	525.242	71.033	.0001	.0001	.0001			
cond * age	1	2.550	2.550	.345	.5625	.5625	.5625			
cond * group	1	1.835	1.835	.248	.6229	.6229	.6229			
cond * age * group	1	7.790	7.790	1.053	.3149	.3149	.3149			
cond * Subject(Group)	24	177.465	7.394							
Dependent: condition b Table of Epsilon Factors for df Adjustment Dependent: condition b										
Dependent: condition b	Epsilon									

ype III Sums of Squares		0				~~	
Source	df	Sum of Squares	Mean Square	F-Value	P-Value	G-G	H-F
age	$\frac{1}{1}$	4.538	4.538	1.546	.2257		
group	1	.799	.799	.272	.6065		
age * group	1	.554	.554	.189	.6678		
Subject(Group)	24	70.436	2.935				
cond	1	4.581	4.581	1.561	.2236	.2236	.2236
cond * age	1	4.538	4.538	1.546	.2257	.2257	.2257
cond * group	1	.799	.799	.272	.6065	.6065	.6065
cond * age * group	1	.554	.554	.189	.6678	.6678	.6678
cond * Subject(Group)	24	70.436	2.935				
Dependent: condition c able of Epsilon Factors for rependent: condition c		tment					
G-G Epsilon H-	F Epsilon	l					
	1.130						

d) ANOVA table investigating effects of age and group on positionning of bead in dark clear condition vs. extra effect of prisms in light.

Type III Sums of Squares

Source	đf	Sum of Squares	Mean Square	F-Value	P-Value	G-G	H-F
age	1	6.146	6.146	2.766	.1093		
group	1	.014	.014	.006	.9366		
age * group	1	.389	.389	.175	.6793		
Subject(Group)	24	53.337	2.222				
cond	1	5.316	5.316	2.392	.1351	.1351	.1351
cond * age	1	6.146	6.146	2.766	.1093	.1093	.1093
cond * group	1	.014	.014	.006	.9366	.9366	.9366
cond * age * group	1	.389	.389	.175	.6793	.6793	.6793
cond * Subject(Group)	24	53.337	2.222				

Dependent: condition d

Table of Epsilon Factors for df AdjustmentDependent: condition d

	G-G Epsilon	H-F Epsilon
cond	1.000	1.130

NOTE: Probabilities are not corrected for values of epsilon greater than 1.

				estigating in dark		-			
	effect								
	ums of Squ								
Source	ans or oqu	QIC3	ďf	Sum of Squares	Mean Square	F-Value	P-Value	G-G	H-F
age			1	.675	.675	.150	.7015		
group			1	.568	.568	.127	.7251		
age * grou			1	.021	.021	.005	.9461		
Subject(G			24	107.677	4.487				
cond			1	30.822	30.822	6.870	.0150	.0150	.0150
cond * age	9		1	.675	.675	.150	.7015	.7015	.7015
cond * gro	oup		1	.568	.568	.127	.7251	.7251	.7251
cond * ag	e * group		1	.021	.021	.005	.9461	.9461	.9461
cond * Su	bject(Group)	24	107.677	4.487				
Table of E Dependen	t: condition psilon Fact t: conditior	ors for d 1 e		ment					
	G-G Epsilor		psilon						
cond	1.000	<u> </u>	1.130						
	babilities ar silon greate		rected fo	r values					

f) ANO	VA	table	inv	estiga	ting	effects	of	age	and	gro	up or
position	ning	g of	bead	l in	dark	clear	cond	itior	n vs.	stı	aigh
ahead	shi	ft.									
Type III Sun	ns of So	quares									
Source			df	Sum of S	quares	Mean Square	F-	Value	P-Value	G-G	H-F
age			1		.053	.053	T	.005	.9442		
group			1		.244	.244		.023	.8807		
age * group			1		6.386	6.386		.601	.4456		
Subject(Gro	up)		24	2	54.811	10.617					
cond			1	24	10.103	240.103	22	2.615	.0001	.0001	.0001
cond * age			1		.053	.053		.005	.9442	. 9 442	. 9 442
cond * group	0		1		.244	.244		.023	.8807	.8807	.8807
cond * age *	group		1		6.386	6.386		.601	.4456	.4456	.4456
cond * Subje	ect(Grou	lb)	24	25	54.811	10.617					
Dependent: Table of Eps Dependent:	ilon Fa	ctors for	df Adjus	tment							
G-	G Epsil	on H-F	Epsilon								
cond	1.00	0	1.130								
NOTE: Proba of epsi		are not co ater than 1		or values							

Appendix 2.4.2.

ANOVA of consistency of measurements in the straight ahead shift experiment.

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value	G-G	H-F
Age	1	5.925	5.925	2.472	.1290		
Group	1	6.864	6.864	2.864	.1035		
Age * Group	1	.049	.049	.021	.8870		
Subject(Group)	24	57.526	2.397				
Category 5	5	99.020	19.804	13.694	.0001	.0001	.0001
Category 5 * Age	5	7.610	1.522	1.052	.3904	.3868	.3904
Category 5 * Group	5	12.545	2.509	1.735	.1318	.1427	.1318
Category 5 * Age * Group	5	2.767	.553	.383	.8599	.8352	.8599
Category 5 * Subject(G	120	173.545	1.446				

Dependent: Compact Variable 3

Table of Epsilon Factors for df Adjustment Dependent: Compact Variable 3

	G-G Epsilon	H-F Epsilon
Category 5	.868	1.215

NOTE: Probabilities are not corrected for values of epsilon greater than 1.

Analysis	of	consistency	of	measurements	in	the	various
condition	IS						

i) In the light with clear goggles

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value
Age	1	.652	.652	.999	.3275
Group	1	.026	.026	.040	.8441
Age * Group	1	.139	.139	.213	.6485
Residual	24	15.653	.652		

Dependent: Iclear

NOTE: 2 rows have been excluded from calculations because of missing values.

ii) In the dark with clear goggles

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value
Age	1	1.357	1.357	.721	.4043
Group	1	.306	.306	.162	.6905
Age * Group	1	.077	.077	.041	.8414
Residual	24	45.179	1.882		

Dependent: dclear

NOTE: 2 rows have been excluded from calculations because of missing values.

iii) In the dark with prisms: first time

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value
Age	1	3.505	3.505	1.453	.2398
Group	1	16.414	16.414	6.804	.0154
Age * Group	1	1.818	1.818	.753	.3940
Residual	24	57.897	2.412		

Dependent: darkpr1

iv) In the light with prisms

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value
Age	1	.018	.018	.020	.8879
Group	1	2.356	2.356	2.658	.1161
Age * Group	1	.531	.531	.599	.4464
Residual	24	21.274	.886		

Dependent: lightp

NOTE: 2 rows have been excluded from calculations because of missing values.

v) In the dark with prisms: 2nd time

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value
Age	1	2.015	2.015	1.183	.2876
Group	1	.011	.011	.007	.9361
Age * Group	1	.251	.251	.147	.7044
Residual	24	40.899	1.704		

Dependent: darkpr2

NOTE: 2 rows have been excluded from calculations because of missing values.

vi) In the dark with clear goggles, 2nd time: negative after effect

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value
Age	1	5.988	5.988	2.865	.1035
Group	1	.297	.297	.142	.7097
Age * Group	1	1.476E-4	1.476E-4	7.062E-5	.9934
Residual	24	50.169	2.090		

Dependent: darknegae?

NOTE: 2 rows have been excluded from calculations because of missing values.

Annandix 2	13							
<u>Appendix 2</u>		1.4.						
Individual	Individual Psychometric data							
	CA	RA 96	IQ					
Name	years	4						
	16.31		121.00					
DG SE T	16.57		114.00					
Τ	16.72		130.00					
BD	15.53							
LC	17.27		107.00					
LA	17.22		129.00					
RD	16.95		119.00					
S	16.94		119.00					
Mean	16.69	17.00	116.88					
Name								
D14								
CJ Œ	16.60		109.00					
Œ	15.03		117.00					
MC	18.65		131.00					
RH	18.12	8.90	101.00					
SA	16.48	12.90	133.00					
ТА	15.89	14.00	120.00					
Mean	16.55	11.88	118.50					
DT	13.58	17.00	123.00					
JV	12.32	12.90	124.00					
MC	13.90		111.00					
MCh	13.11	17.00	112.00					
SS	13.68	16.00	101.00					
TW	13.21	17.00	112.00					
VS	13.20	12.90	117.00					
Mean	13.29	15.24	114.29					
JM	11.74	8.00	96.00					
LP	12.60	9.30	98.00					
MC	13.48	11.90	123.00					
PG	12.67	13.30	120.00					
STC	12.75	9.00	90.00					
JR	11.24	9.00	109.00					
JH	12.87	12.90	126.00					
Mean	12.48	10.49	108.86					

Appendix 2.5.1

Example data sheet from main prism adaptation experiment

	Target One	Target Two	Target three	Target four	Target five
Do 10 practice	s!!! (Each hand	J)			
Baseline in da	rk/ Preexposure	e Period:			
Right Hand					
Left Hand					
Check for adap					
Prisms on (RI	1):		• • • • • • • • • • • • • • • • • • • •		
1		2	4	3	
2		4		<u> </u>	· · · · · · · · · · · · · · · · · · ·
3				4	
4		S .	2		
	rmanual transfe		300000000000000000000000000000000000000	000000000000000000000000000000000000000	
Prisms on LH:		2	4	3	
(No feedback)		4			
"Recharge" RH		2	3		
1		3		*	
2		••••••• • •••• • ••		<u></u>	
Check for page	ative after effect	•			
Prisms off RH:		•			<u> </u>
(No feedback)					
(NO RECUDACK)			.	······	
2		\$		4	
3		2	2		
	ative after effe	ct intermanual	transfer (prisms	off/LH/no feed	hack):
1		4			
2		2		<u>.</u>	
3		2	<u> </u>		
					J

data sheet (contd)				
	Pointer and LE			
	1	2	3	
Light Clear goggles:				
Dark Clear Goggles:				
Dark Prisms:				
Light Prisms:				
Dark Prisms:				
Dark Clear Goggles/ Neg AE?				
Wheel and Bead:	1	2	3	4
Light Clear goggles:				
Dark Clear Goggles:				
Dark Prisms:				
Light Prisms:				
Dark Prisms:				
Dark Clear Goggles/ Neg AE?				
Score out of 10 on Randot ster	reo test:		l • •	
Dominant eye???				
Dyslexic/ Not dyslexic:				
Name:				
Age:	 		· · · · · · · · · · · · · · · · · · ·	

Appendix 2.5.2

i) Initial Accuracy ANOVA tables: Absloute errors: 5-factor ANOVA

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value	G-G	H-F
Grp	1	104.293	104.293	.351	.5593		
age	1	44.453	44.453	.149	.7025		
Grp * age	1	22.395	22.395	.075	.7862		
Subject(Group)	24	7140.452	297.519				
hand	1	400.225	400.225	3.178	.0873	.0873	.0873
hand * Grp	1	1.306	1.306	.010	.9197	.9197	.9197
hand * age	1	143.373	143.373	1.138	.2966	.2966	.2966
hand * Grp * age	1	259.405	259.405	2.060	.1641	.1641	.1641
hand * Subject(Group)	24	3022.547	125.939				
target	2	44.444	22.222	.232	.7939	.7852	.7939
target * Grp	2	182.741	91.371	.953	.3926	.3897	.3926
target * age	2	134.114	67.057	.700	.5018	.4964	.5018
target * Grp * age	2	144.298	72.149	.753	.4765	.4717	.4765
target * Subject(Group)	48	4600.547	95.845				
try	2	114.783	57.392	.699	.5021	.5000	.5021
try * Grp	2	14.694	7.347	.089	.9146	.9120	.9146
try * age	2	73.365	36.683	.447	.6423	.6393	.6423
try * Grp * age	2	692.410	346.205	4.217	.0206	.0212	.0206
try * Subject(Group)	48	3941.091	82.106				
hand * target	2	587.109	293.554	3.372	.0426	.0432	.0426
hand * target * Grp	2	190.396	95.198	1.093	.3432	.3428	.3432
hand * target * age	2	138.075	69.037	.793	.4583	.4571	.4583
hand * target * Grp * age	2	174.705	87.353	1.003	.3742	.3735	.3742
hand * target * Subject	48	4178.860	87.060				
hand * try	2	267.380	133.690	1.092	.3438	.3399	.3438
hand * try * Grp	2	64.630	32.315	.264	.7692	.7498	.7692
hand * try * age	2	175.264	87.632	.716	.4941	.4828	.4941
hand * try * Grp * age	2	26.784	13.392	.109	.8966	.8804	.8966
hand * try * Subject(Gr	48	5878.253	122.464				
target * try	4	246.281	61.570	.643	.6333	.5866	.6256
target * try * Grp	4	448.104	112.026	1.169	.3292	.3271	.3291
target * try * age	4	289.838	72.460	.756	.5562	.5195	.5501
target * try * Grp * age	4	469.047	117.262	1.224	.3057	.3072	.3063
target * try * Subject(96	9196.169	95.793				
hand * target * try	4	205.919	51.480	.626	.6452	.6182	.6452
hand * target * try * Grp	4	421.928	105.482	1.282	.2823	.2856	.2823
hand * target * try * age	4	88.538	22.134	.269	.8972	.8683	.8972
hand * target * try * G	4	637.862	159.466	1.939	.1102	.1230	.1102
hand * target * try * S	96	7896.253	82.253				

Dependent: Absolute error on initial accuracy

Table of Epsilon Factors for df Adjustment Dependent: Absolute error on initial accuracy

	G-G Epsilon	H-F Epsilon
hand	1.000	1.130
target	.960	1.172
try	.984	1.205
hand * target	.989	1.212
hand * try	.915	1.110
target * try	.733	.950
hand * target * try	.841	1.117

Signed errors - 5-factor ANOVA

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value	G-G	H-F
Grp	1	589.071	589.071	.737	.3992		
age	1	141.346	141.346	.177	.6779		
Grp * age	1	21.222	21.222	.027	.8719		
Subject(Group)	24	19189.825	799.576				
hand	1	427.415	427.415	.596	.4476	.4476	.4476
hand * Grp	1	83.162	83.162	.116	.7364	.7364	.7364
hand * age	1	892.601	892.601	1.245	.2755	.2755	.2755
hand * Grp * age	1	80.556	80.556	.112	.7404	.7404	.7404
hand * Subject(Group)	24	17204.754	716.865				
target	2	5153.672	2576.836	10.746	.0001	.0001	.0001
target * Grp	2	1207.868	603.934	2.519	.0912	.0919	.0912
target * age	2	1074.630	537.315	2.241	.1174	.1180	.1174
target * Grp * age	2	1086.057	543.029	2.265	.1149	.1155	.1149
target * Subject(Group)	48	11509.916	239.790				
try	2	1066.530	533.265	2.166	.1257	.1272	.1257
try * Grp	2	143.429	71.715	.291	.7486	.7428	.7486
try * age	2	422.013	211.007	.857	.4308	.4283	.4308
try * Grp * age	2	561.526	280.763	1.140	.3282	.3273	.3282
try * Subject(Group)	48	11816.868	246.185				
hand * target	2	322.903	161.451	1.154	.3240	.3219	.3240
hand * target * Grp	2	715.778	357.889	2.558	.0880	.0919	.0880
hand * target * age	2	1973.404	986.702	7.052	.0021	.0026	.0021
hand * target * Grp * age	2	302.491	151.245	1.081	.3474	.3445	.3474
hand * target * Subject	48	6716.087	139.918				
hand * try	2	248.527	124.264	.642	.5307	.5218	.5307
hand * try * Grp	2	99.688	49.844	.257	.7740	.7607	.7740
hand * try * age	2	453.328	226.664	1.171	.3188	.3170	.3188
hand * try * Grp * age	2	273.682	136.841	.707	.4982	.4903	.4982
hand * try * Subject(Gr	48	9291.626	193.576				
target * try	4	348.280	87.070	.498	.7371	.6801	.7274
target * try * Grp	4	940.278	235.070	1.345	.2589	.2670	.2607
target * try * age	4	426.636	106.659	.610	.6562	.6065	.6476
target * try * Grp * age	4	375.503	93.876	.537	.7088	.6539	.6994
target * try * Subject(96	16777.578	174.766				
hand * target * try	4	1555.536	388.884	2.698	.0352	.0532	.0377
hand * target * try * Grp	4	292.230	73.057	.507	.7308	.6757	.7228
hand * target * try * age	4	585.483	146.371	1.015	.4035	.3902	.4017
hand * target * try * G	4	175.158	43.790	.304	.8748	.8192	.8672
hand * target * try * S	96	13839.519	144.162				

Dependent: real errors on initial accuracy

Table of Epsilon Factors for df Adjustment Dependent: real errors on initial accuracy

	G-G Epsilon	H-F Epsilon
hand	1.000	1.130
target	.989	1.212
try	.974	1.191
hand * target	.938	1.142
hand * try	.940	1.145
target * try	.731	.948
hand * target * try	.737	.956

Independent analyses of each hand: preferred hand a) absolute errors

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value	G-G	H-F
Grp	1	64.471	64.471	.435	.5158		
age	1	14.080	14.080	.095	.7606		
Grp * age	1	217.118	217.118	1.465	.2379		
Subject(Group)	24	3557.105	148.213				
target	2	377.770	188.885	2.144	.1282	.1291	.1282
target * Grp	2	102.660	51.330	.583	.5623	.5598	.5623
target * age	2	4.282	2.141	.024	.9760	.9748	.9760
target * Grp * age	2	311.611	155.806	1.769	.1815	.1821	.1815
target * Subject(Group)	48	4228.147	88.086				
try	2	98.217	49.108	.456	.6365	.6213	.6365
try * Grp	2	60.625	30.313	.281	.7559	.7386	.7559
try * age	2	15.416	7.708	.072	.9310	.9189	.9310
try * Grp * age	2	227.248	113.624	1.055	.3561	.3521	.3561
try * Subject(Group)	48	5169.147	107.691				
target * try	4	269.434	67.359	.790	.5344	.4966	.5239
target * try * Grp	4	82.548	20.637	.242	.9138	.8552	.8991
target * try * age	4	132.238	33.060	.388	.8169	.7498	.7989
target * try * Grp * age	4	133.475	33.369	.391	.8143	.7472	.7963
target * try * Subject(96	8183.940	85.249				

Dependent: preferred Absolute error on initial accuracy

Table of Epsilon Factors for df Adjustment Dependent: preferred Absolute error on initial accuracy

	G-G Epsilon	H-F Epsilon
target	.985	1.206
try	.923	1.122
target * try	.705	.908

b) signed errors

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value	G-G	H-F
Grp	1	114.784	114.784	.170	.6836		
age	1	161.775	161.775	.240	.6287		
Grp * age	1	9.542	9.542	.014	.9063		
Subject(Group)	24	16181.936	674.247				
target	2	1590.035	7 9 5.018	4.552	.0155	.0183	.0155
target * Grp	2	209.968	104.984	.601	.5523	.5390	.5523
target * age	2	2840.268	1420.134	8.131	.0009	.0013	.0009
target * Grp * age	2	1006.131	503.066	2.880	.0659	.0710	.0659
target * Subject(Group)	48	8383.983	174.666				
try	2	951.259	475.630	2.690	.0781	.0791	.0781
try * Grp	2	226.179	113.090	.640	.5320	.5296	.5320
try * age	2	518.087	259.044	1.465	.2412	.2414	.2412
try * Grp * age	2	28.689	14.345	.081	.9222	.9197	.9222
try * Subject(Group)	48	8487.403	176.821				
target * try	4	389.079	97.270	.708	.5886	.5569	.5886
target * try * Grp	4	560.864	140.216	1.020	.4009	.3910	.4009
target * try * age	4	194.344	48.586	.354	.8410	.7962	.8410
target * try * Grp * age	4	443.883	110.971	.807	.5234	.4988	.5234
target * try * Subject(96	13193.898	137.436				

Dependent: preferred real errors on initial accuracy

Table of Epsilon Factors for df Adjustment Dependent: preferred real errors on initial accuracy

	G-G Epsilon	H-F Epsilon
target	.918	1.115
try	.984	1.205
target * try	.787	1.033

Independent analyses of each hand: non-preferred hand a) absolute errors

Type III Sums of Squares

df	Sum of Squares	Mean Square	F-Value	P-Value	G-G	H-F
1	41.128	41.128	.149	.7025		
1	173.746	173.746	.631	.4347		
1	64.681	64.681	.235	.6322		
24	6605.894	275.246				
2	253.784	126.892	1.338	.2719	.2716	.2719
2	270.477	135.239	1.426	.2502	.2507	.2502
2	267.907	133.953	1.413	.2534	.2538	.2534
2	7.392	3.696	.039	.9618	.9542	.9618
48	4551.261	94.818				
2	283.946	141.973	1.465	.2411	.2413	.2411
2	18.699	9.349	.097	.9082	.9057	.9082
2	233.214	116.607	1.204	.3090	.3087	.3090
2	491.946	245.973	2.539	.0895	.0904	.0895
48	4650.197	96.879				
4	182.766	45.692	.492	.7413	.6998	.7413
4	787.485	196.871	2.122	.0840	.1007	.0840
4	246.138	61.534	.663	.6191	.5861	.6191
4	973.433	243.358	2.622	.0395	.0533	.0395
96	8908.481	92.797				
	1 1 1 24 2 2 2 2 2 2 2 2 2 2 2 2 2	1 41.128 1 173.746 1 64.681 24 6605.894 2 253.784 2 253.784 2 270.477 2 267.907 2 7.392 48 4551.261 2 283.946 2 18.699 2 233.214 2 491.946 48 4650.197 4 182.766 4 787.485 4 246.138 4 973.433	1 41.128 41.128 1 173.746 173.746 1 64.681 64.681 24 6605.894 275.246 2 253.784 126.892 2 270.477 135.239 2 267.907 133.953 2 7.392 3.696 48 4551.261 94.818 2 283.946 141.973 2 18.699 9.349 2 233.214 116.607 2 491.946 245.973 48 4650.197 96.879 4 182.766 45.692 4 787.485 196.871 4 246.138 61.534 4 973.433 243.358 96 8908.481 92.797	1 41.128 41.128 .149 1 173.746 173.746 .631 1 64.681 64.681 .235 24 6605.894 275.246	1 41.128 41.128 .149 .7025 1 173.746 173.746 .631 .4347 1 64.681 64.681 .235 .6322 24 6605.894 275.246	1 41.128 41.128 .149 .7025 1 173.746 173.746 .631 .4347 1 64.681 64.681 .235 .6322 24 6605.894 275.246

Dependent: nonpreferred Absolute error on initial accuracy

Table of Epsilon Factors for df Adjustment Dependent: nonpreferred Absolute error on initial accuracy

	G-G Epsilon	H-F Epsilon
target	.932	1.134
try	.985	1.207
target * try	.797	1.048

b) signed errors

Type III Sums of Squares

Source d		Sum of Squares	Mean Square	F-Value	P-Value	G-G	H-F
Grp	1	557.449	557.449	.662	.4239		
age	1	872.171	872.171	1.036	.3190		
Grp * age	1	92.236	92.236	.110	.7436		
Subject(Group)	24	20212.643	842.193				
target	2	3886.540	1943.270	9.477	.0003	.0005	.0003
target * Grp	2	1713.677	856.839	4.179	.0212	.0245	.0212
target * age	2	207.767	103.883	.507	.6057	.5910	.6057
target * Grp * age	2	382.417	191.208	.933	.4006	.3943	.4006
target * Subject(Group)	48	9842.020	205.042				
try	2	363.797	181.899	.692	.5056	.5040	.5056
try * Grp	2	16.938	8.469	.032	.9683	.9672	.9683
try * age	2	357.254	178.627	.679	.5118	.5101	.5118
try * Grp * age	2	806.519	403.259	1.534	.2261	.2264	.2261
try * Subject(Group)	48	12621.091	262.939				
target * try	4	1514.738	378.684	2.087	.0885	.1091	.0898
target * try * Grp	4	671.644	167.911	.925	.4527	.4336	.4516
target * try * age	4	817.775	204.444	1.126	.3487	.3443	.3485
target * try * Grp * age	4	106.779	26.695	.147	.9639	.9321	.9623
target * try * Subject(96	17423.198	181.492				

Dependent: nonpreferred real errors on initial accuracy

Table of Epsilon Factors for df Adjustment Dependent: nonpreferred real errors on initial accuracy

	G-G Epsilon	H-F Epsilon
target	.921	1.119
try	.988	1.211
target * try	.755	.983

ii) Adaptation

ANOVAs of regression line slope and intercept coefficeents

Unadjusted coefficients for all targets

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value
age	1	2.201	2.201	.885	.3561
group	1	.441	.441	.178	.6772
age * group	1	.708	.708	.285	.5985
Residual	24	59.666	2.486		

Dependent: slopeall

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value
age	1	2772.320	2772.320	3.851	.0614
group	1	8.808	8.808	.012	.9128
age * group	1	939.198	939.198	1.305	.2647
Residual	24	17279.059	719.961		

Dependent: interceptall

Adjusted coefficient for all targets

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value
age	1	33.093	33.093	10.075	.0041
group	1	.819	.819	.249	.6221
age * group	1	2.701	2.701	.822	.3735
Residual	24	78.833	3.285		

Dependent: Allthro60

Target One coefficients

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value
age	1	26.532	26.532	1.022	.3222
group	1	10.910	10.910	.420	.5230
age * group	1	27.905	27.905	1.075	.3102
Residual	24	623.231	25.968		

Dependent: t1slope

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value
age	1	2117.754	2117.754	3.342	.0800
group	1	20.629	20.629	.033	.8583
age * group	1	796.967	796.967	1.258	.2732
Residual	24	15207.594	633.650		

Dependent: t1intercept

Target Two coefficients

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value
age	1	35.666	35.666	1.254	.2739
group	1	.580	.580	.020	.8876
age * group	1	43.331	43.331	1.523	.2291
Residual	24	682.774	28.449		

Dependent: t2slope

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value
age	1	3471.617	3471.617	3.702	.0663
group	1	103.313	103.313	.110	.7428
age * group	1	2575.087	2575.087	2.746	.1105
Residual	24	22504.622	937.693		

Dependent: t2intercept

Target Three coefficients

Dependent: t3slopeType III

Sums of Squares Source	df	Sum of Squares	Mean Square	F-Value	P-Value
age	1	7.195	7.195	.134	.7178
gr oup	1	.358	.358	.007	.9357
age * group	1	17.169	17.169	.3 19	.5774
Residual	24	1291.318	53.805		

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value
age	1	2207.168	2207.168	1.315	.2628
group	1	34.751	34.751	.021	.8868
age * group	1	64.614	64.614	.039	.8461
Residual	24	40275.919	1678.163		

Dependent: t3intercept

iii) Intermanual Transfer

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value	G-G	H-F
age	1	3798.081	3798.081	.933	.3436		
group	1	499.160	499.160	.123	.7292		
age * group	1	2605.082	2605.082	.640	.4315		
Subject(Group)	24	97650.891	4068.787				
target position	2	69.219	34.610	.510	.6038	.5642	.5957
target position * age	2	420.771	210.386	3.099	.0542	.0669	.0568
target position * group	2	92.172	46.086	.679	.5120	.4817	.5058
target position * age *	2	380.578	190.289	2.803	.0706	.0838	.0733
target position * Subjec	48	3258.174	67.879				

Dependent: intermanual transfer

Table of Epsilon Factors for df Adjustment Dependent: intermanual transfer

	G-G Epsilon	H-F Epsilon					
target position	.800	.956					
non-preferre	d han	d target	one	vs	initial	displacement	of

prisms

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value	G-G	H-F
age	1	5077.238	5077.238	4.168	.0523		
group	1	106.446	106.446	.087	.7701		1
age * group	1	831.759	831.759	.683	.4168		
Subject(Group)	24	29238.333	1218.264				
initial	1	2405.402	2405.402	3.509	.0733	.0733	.0733
initial * age	1	878.272	878.272	1.281	.2689	.2689	.2689
initial * group	1	10.630	10.630	.016	.9019	.9019	.9019
initial * age * group	1	301.092	301.092	.439	.5138	.5138	.5138
initial * Subject(Group)	24	16453.495	685.562		_		

Dependent: im transfer?

Table of Epsilon Factors for df Adjustment Dependent: im transfer?

	G-G Epsilon	H-F Epsilon
initial	1.000	1.130

non-preferred hand target two vs initial displacement of prisms

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value	G-G	H-F
age	1	7624.684	7624.684	6.012	.0219		
group	1	89.509	89.509	.071	.7928		
age * group	1	1225.528	1225.528	.966	.3354		
Subject(Group)	24	30435.461	1268.144		:		
initial	1	3281.694	3281.694	4.234	.0506	.0506	.0506
initial * age	1	184.171	184.171	.238	.6304	.6304	.6304
initial * group	1	5.779	5.779	.007	.9319	.9319	.9319
initial * age * group	1	553.158	553.158	.714	.4066	.4066	.4066
initial * Subject(Group)	24	18602.068	775.086				

Dependent: im transfer?

Table of Epsilon Factors for df Adjustment Dependent: im transfer?

H-F Epsilon G-G Epsilon ini

NOTE: Probabilities are not corrected for values of epsilon greater than 1.

hand target three vs initial displacement non-preferred of

prisms

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value	G-G	H-F
age	1	4656.793	4656.793	3.226	.0851		
group	1	330.160	330.160	.229	.6368		
age * group	1	252.669	252.669	.175	.6794		
Subject(Group)	24	34641.813	1443.409				
initial	1	2722.451	2722.451	3.212	.0857	.0857	.0857
initial * age	1	1066.003	1066.003	1.258	.2732	.2732	.2732
initial * group	1	123.508	123.508	.146	.7060	.7060	.7060
initial * age * group	1	19.425	19.425	.023	.8809	.8809	.8809
initial * Subject(Group)	24	20341.662	847.569				

Dependent: im transfer?

Table of Epsilon Factors for df Adjustment Dependent: im transfer?

	G-G Epsilon	H-F Epsilon
initial	1.000	1.130

iv) Negative after effect

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value	G-G	H-F
age	1	17946.064	17946.064	3.378	.0785		
group	1	14432.936	14432.936	2.717	.1123		
age * group	1	3224.406	3224.406	.607	.4436		
Subject(Group)	24	127506.526	5312.772				
hand	1	10040.009	10040.009	8.839	.0066	.0066	.0066
hand * age	1	9.852	9.852	.009	.9266	.9266	.9266
hand * group	1	1516.638	1516.638	1.335	.2592	.2592	.2592
hand * age * group	1	2145.063	2145.063	1.889	.1821	.1821	.1821
hand * Subject(Group)	24	27260.332	1135.847				
target position	2	13746.704	6873.352	8.319	.0008	.0026	.0014
target position * age	2	2288.886	1144.443	1.385	.2601	.2590	.2602
target position * group	2	496.014	248.007	.300	.7421	.6781	.7154
target position * age *	2	2911.504	1455.752	1.762	.1826	.1919	.1871
target position * Subjec	48	39658.908	826.227				
trial	2	594.390	297.195	.907	.4105	.3993	.4105
trial * age	2	86.845	43.423	.133	.8762	.8491	.8762
trial * group	2	2484.577	1242.289	3.792	.0296	.0360	.0296
trial * age * group	2	1384.241	692.120	2.112	.1321	.1393	.1321
trial * Subject(Group)	48	15727.037	327.647				
hand * target position	2	5955.608	2977.804	6.175	.0041	.0064	.0041
hand * target position *	2	49.427	24.714	.051	.9501	.9299	.9501
hand * target position *	2	914.209	457.104	.948	.3947	.3837	.3947
hand * target position *	2	215.083	107.542	.223	.8009	.7682	.8009
hand * target position *	48	23146.114	482.211				
hand * trial	2	516.253	258.126	.708	.4975	.4907	.4975
hand * trial * age	2	2448.758	1224.379	3.361	.0431	.0458	.0431
hand * trial * group	2	1697.794	848.897	2.330	.1082	.1113	.1082
hand * trial * age * group	2	870.553	435.276	1.195	.3116	.3103	.3116
hand * trial * Subject(G	48	17487.886	364.331				
target position * trial	4	2862.169	715.542	2.848	.0280	.0451	.0308
target position * trial *	4	599.058	149.765	.596	.6663	.6150	.6569
target position * trial *	4	2112.914	528.228	2.103	.0864	.1094	.0907
target position * trial *	4	620.002	155.000	.617	.6515	6018	.6424
target position * trial *	96	24117.761	251.227				
hand * target position *	4	2802.812	700.703	2.960	.0236	.0276	.0236
hand * target position *	4	838.759	209.690	.886	.4755	.4687	.4755
hand * target position *	4	2322.961	580.740	2.453	.0510	.0567	.0510
hand * target position *	4	903.839	225.960	.955	.4362	.4311	.4362
hand * target position *	96	22724.983	236.719				

Dependent: Exact negative after effects

Table of Epsilon Factors for df Adjustment Dependent: Exact negative after effects

	G-G Epsilon	H-F Epsilon
hand	1.000	1.130
target position	.746	.884
trial	.872	1.053
hand * target position	.861	1.037
hand * trial	.949	1.157
target position * trial	.729	.944
hand * target position * trial	.917	1.237

Type III Sums of Squares

Source	df	Sum of Sq	Mean Sq	F-Va	P-V	G-G	H-F
age	1	3507.194	3507.194	3.308	.0814		
group	1	3190.665	3190.665	3.010	.0956		
age * group	1	1332.179	1332.179	1.257	.2734		
Subject(Group)	24	25443.055	1060.127				
target	2	103.178	51.589	.800	.4554	.3891	.4037
target * age	2	63.488	31.744	.492	.6144	.5041	.5276
target * group	2	246.400	123.200	1.910	.1592	.1787	.1763
target * age * group	2	305.918	152.959	2.371	.1043	.1341	.1291
target *	48	3096.918	64.519				

Dependent: Negative After Effect-rh

Table of Epsilon Factors for df Adjustment Dependent: Negative After Effect-rh

	G-G Epsilon	H-F Epsilon
target	.542	.619

Type III Sums of Squares

Source	df	Sum of Sq	Mean Sq	F-Va	P-V	G-G	H-F
age	1	1192.318	1192.318	.642	.4310		
group	1	1211.640	1211.640	.652	.4273		
age * group	1	7658.109	7658.109	4.121	.0536		
Subject(Group)	24	44596.416	1858.184				
target	2	381.541	190.770	.335	.7167	.6945	.7167
target * age	2	1367.807	683.904	1.203	.3093	.3067	.3093
target * group	2	1356.407	678.204	1.193	.3123	.3095	.3123
target * age * grou	2	900.066	450.033	.791	.4590	.4477	.4590
target * Subject(48	27295.754	568.662				

Dependent: Adjusted right hand negative after effect

Table of Epsilon Factors for df Adjustment Dependent: Adj. right hand neg.ae

	G-G Epsilon	H-F Epsilon
target	.901	1.091

Type III Sums of Squares

Source	df	Sum of	Mean Sq	F-Va	P-V	G-G	H-F
age	1	3245.487	3245.487	2.963	.0981		
group	1	1238.636	1238.636	1.131	.2982		
age * group	1	1.528	1.528	.001	.9705		
Subject(Group)	24	2.629E4	1095.453				
target	2	91.939	45.969	3.588	.0353	.0375	.0353
target * age	2	61.445	30.723	2.398	.1017	.1044	.1017
target* group	2	29.554	14.777	1.153	.3242	.3228	.3242
target * age *group	2	107.736	53.868	4.204	.0208	.0224	.0208
target * Subj	48	615.017	12.813				

Dependent: Negative after effect - Ih

Table of Epsilon Factors for df Adjustment Dependent: Negative after effect - Ih

	G-G Epsilon	H-F Epsilon
target	.958	1.169

Appendix 3.3.1: The method of diagnosing dyslexia in adults

The diagnosis of dyslexia in adults is not straightforward, in that adult skills vary considerably as a result of experience (Nicolson, Fawcett and Miles, 1992). The normal method for diagnosing dyslexia in adults is to proceed by analogy with testing children for dyslexia, and this is probably the best starting point for an explanation of the method we use.

For children the conventional definition of dyslexia is "a disorder in children who, despite conventional classroom experience, fail to attain the language skills of reading, writing and spelling commensurate with their intellectual abilities" (from the definition by the World Federation of Neurology, 1968). Simplifying considerably, conservative operational approach to diagnosing dyslexia in children would therefore be to take as criterion an IQ of 90 or more, coupled with a reading age of at least 18 months behind chronological age. Furthermore, a distinctive pattern of functioning (the ACID profile) is frequently recorded, which involves normal or good scores on the most of the subtests of the Wechsler Intelligence Scale for Children, coupled with unexpectedly low scores on two or more of the Arithmetic, Coding, Information and Digit span sub-tests.

Unfortunately the discrepancy between reading age and chronological age is of little value in diagnosing adults, since they may well have effectively caught up with their reading over a period. Furthermore, a certain spikiness is reasonably common in the profiles for normal students on psychometric tests, based on the overlay of learning styles on the natural abilities of the student. In view of these difficulties we have developed a composite Adult Dyslexia Diagnostic Test, which involves administration of the Wechsler Adult Intelligence Scale (WAIS), together with a test of nonsense passage reading known to be useful for diagnosing adult dyslexia, a spelling test (spelling difficulties usually persist longer than reading difficulties in adult dyslexia), and also evidence of dyslexia in childhood.

Measure	ADI Scoring criterion					
Previous diagnosis of dyslexia	1 for Psychologist's report, 0 otherwise					
BAS/Word spelling scale	0.5 for 16-17 1 for <16					
Nonsense word passage						
Error score	0.5 for >7 errors					
Completion time	0.5 for >59 seconds					
WAIS profile	0.5 for a shortfall (>3 points					
Arithmetic	compared with non-ACID					
Digit Symbol	mean) in one of the ACID					
Digit Span	subtests					
Information	1 for two or more shortfalls					
Overall ADI Score	>2.5 points—dyslexic >1 point —non-dyslexic					

Dr. R.I. Nicolson Dr. A.J.Fawcett December 1994

Appendix 3.3.2: ANOVAs without exclusions

Raw Data (main analyses only)

000110		errors		classical	M	ean SDs		mage also	modat	٨٩٩	Х2-Х5 п	ieans not u	eber!	classic	fatigue on	classical
group	UNF	Medium	Hi	UIA33IUAI	UF	Med	Hi	mean clas	randot	Age	UF	Med	Hi	CIA33IC	beginning	end
control	.710	.871	.843	.975	70.765	77.874	1.2E2	47.220	10	21	104.637	118.154	1.77E2	81.876	58.631	35.809
control	.901	1.173	1.303	.453	35.637	37.334	8.1E1	43.311	10	20	60.738	57.553	1.34E2	63.624	52.253	35.899
control	.485	.707	1.880	.334	43.767	98.217	3.4E2	54.250	10	21	44.477	123.842	6.23E2	79.070	58.565	49.934
control	.289	.393	.406	.489	50.705	49.883	8E1	31.365	10	20	66.514	82.412	1.25E2	45.203	30.003	32.726
control	.682	.806	1.063	1.188	55.573	58.802	8.5E1	49.740	10	21	54.476	86.808	1.15E2	74.475	66.097	33.384
dyslexic	.144	.223	.310	.591	30.397	28.156	5.8E1	101.766	10	26	47.923	24.025	91.356	133.058	101.713	101.818
dyslexic	.391	.508	2.155	1.666	50.209	73.880	3.9E2	99.740	10	27	75.238	69.318	423.34	185.037	74.196	125.284
dyslexic	1.376	1.435	5.441	.988	278.747	402.134	7.9E2	131.649	Ô	18	448.646	194.016	1.06E3	232.000	123.036	140.263
dyslexic	.240	.260	1.930	.260	38.341	30.090	2.9E2	32.753	5	25	69.308	49.228	4.92E2	5.700	25.560	39.948
control	1.042	.723	1.294	1.401	49.792	43.918	1.7E2	61.819	10	22	62.179	56.009	2.14E2	79.869	75.268	48.371
control	.450	.248	.711	.709	49.953	32.774	1.3E2	60.988	10	22	39.615	39.425	165.85	110.961	50.956	71.019
control	.273	.277	1.316	.293	54.457	58.026	2E2	56.150	8	20	76.141	83.797	2.91E2	88.442	57.876	54.425
dyslexic	.236	.575	.504	.623	38.724	113.820	9.7E1	67.725	8	18	43.275	63.177	1.58E2	114.910	51.835	83.616
dyslexic	.387	.324	.837	.968	48.497	45.780	1.6E2	72.694	8	22	83.517	72.188	1.92E2	127.091	81.043	64.345
dyslexic	1.189	1.377	1.185	1.484	51.264	52.625	1.3E2	47.141	8	31	84.860	46.085	1.79E2	73.210	38.085	56.198

a) thresholds in sequential condition

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value	G-G	H-F
group	1	48351.495	48351.495	1.384	.2605		
Subject(Group)	13	454202.159	34938.628				
Mean SDs	2	192459.708	96229.854	16.865	.0001	.0008	.0005
Mean SDs * group	2	19043.432	9521.716	1.669	.2080	.2190	.2186
Mean SDs * Subject(Gro	26	148352.172	5705.853				

Dependent: Mean SDs

Table of Epsilon Factors for df Adjustment Dependent: Mean SDs

	G-G Epsilon	H-F Epsilon
Mean SDs	.545	.603

b) absolute % errors

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value	G-G	H-F
group	1	.523	.523	.467	.5062		
Subject(Group)	13	14.551	1.119				
Sequential Stereopsis	2	6.592	3.296	8.697	.0013	.0103	.0083
Sequential Stereopsis *	2	1.130	.565	1.491	.2438	.2444	.2456
Sequential Stereopsis *	26	9.853	.379				

Dependent: errors

Table of Epsilon Factors for df Adjustment Dependent: errors

	G-G Epsilon	H-F Epsilon
Sequential Stereopsis	.520	.569

c) using unbiased estimator method

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value	G-G	H-F
group	1	45169.559	45169.559	.932	.3519		
Subject(Group)	13	629784.200	48444.938				
Textures	2	462347.881	231173.940	14.945	.0001	.0013	.0008
Textures * group	2	40098.797	20049.398	1.296	.2907	.2791	.2823
Textures * Subject(Gro	26	402175.733	15468.297				

Dependent: X2-X5 means not weber!

Table of Epsilon Factors for df Adjustment Dependent: X2-X5 means not weber!

	G-G Epsilon	H-F Epsilon
Textures	.556	.618

d) RANOVAs

Thresholds: ***** Analysis of data in input file: ex1thresholds This is output file: exlthresholds output ***** STATISTICS FROM OBSERVED DATA ***** 2 No. of groups: Group sizes: 1 8 7 2 ****** Means over within-subjs factor (cols) for each group (row): Gp 1 73.73725 172.5766 * 61.62275 Gp 2 320.7248 * 81.95013 140.0968 ****** F for Groups: 1.463377 F for Within-subjs: 12.67897 F for Interaction: 1.619287 STATISTICS FROM RANDOMIZED DATA ***** No. of randomizations: 10000 2.633333 mins Duration: ***** Significance of F ratios: No. of randomization F ratios that equal or exceed obtained F, & p-values: .2584 2584 Groups: .0001 Withn subjs: 1 .224 2240 Interaction: *****

Errors: ***** Analysis of data in input file: exlerrors This is output file: exlerrors output ***** STATISTICS FROM OBSERVED DATA ***** 2 No. of groups: Group sizes: 8 1 7 2 ****** Means over within-subjs factor (cols) for each group (row): Gp 1 .604 .6497499 1.102 Gp 2 1.766 * .5661429 .6717142 ****** F for Groups: .4669734 F for Within-subjs: 8.260505 F for Interaction: 1.491418 ****** STATISTICS FROM RANDOMIZED DATA ***** No. of randomizations: 10000 2.716667 mins Duration: ****** Significance of F ratios: No. of randomization F ratios that equal or exceed obtained F, & p-values: 6269 .6269 Groups: Withn subjs: 2 .0002 Interaction: 2479 .2479 *****

Appendix 3.3.3. Expt 1 ANOVAs with exclusions

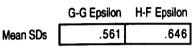
a) Thresholds

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value	G-G	H-F
group	1	206.245	206.245	.130	.7258		
Subject(Group)	10	15854.110	1585.411				
Mean SDs	2	55791.457	27895.728	16.007	.0001	.0016	.0009
Mean SDs * group	2	1667.154	833.577	.478	.6267	.5247	.5492
Mean SDs * Subject(Gro	20	34855.068	1742.753				

Dependent: Mean SDs

Table of Epsilon Factors for df Adjustment Dependent: Mean SDs



b) Absolute % errors

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value	G-G	H-F
group	1	.093	.093	.281	.6077		
Subject(Group)	10	3.303	.330				
Sequential Stereopsis	2	1.324	.662	6.683	.0060	.0210	.0156
Sequential Stereopsis *	2	.031	.016	.157	.8559	.7382	.7740
Sequential Stereopsis *	20	1.982	.099				

Dependent: errors

Table of Epsilon Factors for df Adjustment Dependent: errors

	G-G Epsilon	H-F Epsilon
Sequential Stereopsis	.585	.682

c) Unbiased estimator method

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value	G-G	H-F
group	1	518.293	518.293	.103	.7551		
Subject(Group)	10	50435.674	5043.567				
Textures	2	139225.430	69612.715	17.661	.0001	.0016	.0010
Textures * group	2	7756.979	3878.489	.984	.3912	.3473	.3558
Textures * Subject(Gro	20	78834.237	3941.712				

Dependent: X2-X5 means not weber!

Table of Epsilon Factors for df Adjustment Dependent: X2-X5 means not weber!

	G-G Epsilon	H-F Epsilon
Textures	.518	.582

d) RANOVAs **Errors:** ***** Analysis of data in input file: exlerrors excl This is output file: ex1 errors excl ****** STATISTICS FROM OBSERVED DATA ***** No. of groups: 2 Group sizes: 1 7 2 5 ****** Means over within-subjs factor (cols) for each group (row): Gp 1 .621 .6415714 .9908571 Gp 2 .4392 .5518 .9531999 * ***** F for Groups: .2814433 F for Within-subjs: 6.577976 F for Interaction: .1567826 ****** STATISTICS FROM RANDOMIZED DATA ***** No. of randomizations: 10000 2.166667 Duration: mins ***** Significance of F ratios: No. of randomization F ratios that equal or exceed obtained F. & p-values: .6053 6053 Groups: .0012 Withn subjs: 12 Interaction: 8753 .8753

Thresholds:

***** Analysis of data in input file: ex1 nwthresholds excl This is output file: ex1 nw excl out ***** STATISTICS FROM OBSERVED DATA ****** No. of groups: 2 Group sizes: 7 1 5 2 ****** Means over within-subjs factor (cols) for each group (row): Gp 1 77,70457 75.95185 182.3724 * Gp 2 61.445 80.199 215.9794 * ****** F for Groups: .1301002 F for Within-subjs: 15.59092 F for Interaction: .4783162 ***** STATISTICS FROM RANDOMIZED DATA ***** No. of randomizations: 10000 Duration: 2.15 mins ****** Significance of F ratios: No. of randomization F ratios that equal or exceed obtained F, & p-values: 7283 .7283 Groups: .0002 Withn subjs: 2 6452 .6452 Interaction:

Appendix 3.3.4: Expt 1 Simultaneous condition

a)Mann-Whitney U test (no exclusions) of thresholds:

Mann-Whitney U X 1 : Column 1 Y 1 : Column 2

	Number:	<u>Σ</u> Rank:		Mean Rank:
dyslexi	7	71		10.143
dyslexio contro	1 8	49		6.125
г	U		13	,
Γ	U-prime		43	
	Z		-1.736	p = .0826

b) F test/ Variance ratio test

$$F(6,7) = \frac{2579.4}{223.95} = 11.51 \quad \text{p<0.05}$$

c) Fatigue Effect RANOVA

***** Analysis of data in input file: fatigue This is output file: fatigue out ***** STATISTICS FROM OBSERVED DATA ***** 2 No. of groups: Group sizes: 1 8 2 7 ******* Means over within-subjs factor (cols) for each group (row): Gp 1 45.19587 56.20613 Gp 2 87.35285 70.78114 * ***** F for Groups: 5.043093 F for Within-subjs: .1374063 F for Interaction: 7.509558 ***** STATISTICS FROM RANDOMIZED DATA ****** No. of randomizations: 10000 1.85 mins Duration: ****** Significance of F ratios: No. of randomization F ratios that equal or exceed obtained F, & p-values: .0406 406 Groups: 7197 .7197 Withn subjs: 179 Interaction: .0179 *****

Appendices: Chapter 3

<u>Appendix 3.4.1</u> Expt 2: no exclusions Raw data for main analyses

	Errors						NOT x 0.6745!					
group		Single			Dual			Single			Dual	
	UF	MED	HI	UF	MED	HI	UF	MED	HI	UF	MED	HI
Category	Real	Real	Real	Real	Real	Real	Real	Real	Real	Real	Real	Real
User E	User E	User E	User	User	User	User	User	User E	User E	User E	User	User E
Nominal	Continu	Continu	Conti	Cont	Cont	Conti	Contin	Contin	Contin	Contin	Contin	Continu.
•	Free Fo	Free F	Free	Free	Free	Free	Free F	Free F	Free F	Free F	Free F	Free Fo.
•	3	3	3	3	3	3	3	3	3	3	3	3
control	.698	.506	6.444	.579	.787	4.042	250.368	73.156	1611.957	131.182	142.197	1104.53
control	.208	.357	1.231	.233	.219	1.628	52.772	92.372	204.068	67.419	56.520	410.83
control	.271	.293	3.172	.413	.585	.689	67.792	61.231	944.817	77.109	135.613	172.77
dyslexic	.793	1.025	1.560	1.088	1.146	.918	82.233	92.489	200.537	104.537	150.694	206.952
control	1.252	1.386	3.067	1.980	2.188	2.794	250.003	268.519	825.658	354.648	256.787	582.534
control	.786	.790	1.140	.777	.617	.504	120.511	77.278	222.426	98.983	104.759	130.44
control	.617	.968	2.085	.371	1.290	3.211	64.814	152.417	287.999	93.566	155.779	693.714
dyslexic	3.481	9.691	5.576	5.869	10.332	7.821	273.446	1057.385	563.299	474.018	1.353E3	694.130
control	.247	.273	.957	.333	.459	1.045	70.790	58.292	265.616	62.394	96.946	153.810
dyslexic	1.098	1.282	1.139	1.208	1.366	2.190	94.867	74.587	165.121	138.113	96.175	302.314
control	.670	.972	.620	.753	.899	1.163	63.742	45.493	162.503	56.836	42.796	277.481
dyslexic	1.651	2.345	2.819	2.423	2.567	3.203	313.503	540.271	834.095	363.386	565.534	714.068
dyslexic	.347	.285	.322	.407	.374	.147	42.514	41.726	77.798	34.692	84.626	55.023
dyslexic	.480	.931	.954	.806	1.113	.732	97.787	58.936	244.649	99.878	141.107	195.63
dyslexic	.322	.137	.567	.190	.289	.574	59.852	30.099	171.735	56.182	89.749	119.784
dyslexic	.468	1.553	3.879	.596	1.249	2.077	105.143	221.213	595.125	84.206	137.297	218.43
dyslexic	1.051	.513	1.520	.862	.617	2.686	40.669	66.981	628.510	74.581	89.219	674.17
control	2.307	2.041	2.899	1.846	2.170	2.194	834.983	557.228	855.724	556.245	666.181	613.33
dyslexic	.319	.157	.632	.303	.348	.523	90.855	81.094	178.114	69.213	90.838	127.56
control	.295	.274	.809	.269	.340	.894	81.238	63.148	157.902	79.076	94.259	232.30

a) thresholds

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value	G-G	H-F
group	1	27498.425	27498.425	.099	.7563		
Subject(Group)	18	4983972.493	276887.361				
Task	1	3670.377	3670.377	.223	.6423	.6423	.6423
Task * group	1	21973.358	21973.358	1.336	.2628	.2628	.2628
Task * Subject(Group)	18	295970.714	16442.817				
Texture	2	1617951.115	808975.558	11.606	.0001	.0006	.0003
Texture * group	2	289229.321	144614.661	2.075	.1403	.1535	.1477
Texture * Subject(Group)	36	2509308.079	69703.002				
Task * Texture	2	71392.582	35696.291	2.805	.0738	.1019	.0972
Task * Texture * group	2	4336.565	2168.282	.170	.8440	.7347	.7563
Task * Texture * Subje	36	458171.393	12726.983				

Dependent: NOT x 0.6745!

Table of Epsilon Factors for df Adjustment Dependent: NOT x 0.6745!

	G-G Epsilon	H-F Epsilon
Task	1.000	1.059
Texture	.767	.871
Task * Texture	.614	.672

b) Absolute % errors

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value	G-G	H-F
group	1	6.539	6.539	.471	.5012		
Subject(Group)	18	249.767	13.876				
Task	1	.189	.189	.450	.5108	.5108	.5108
Task * group	1	.752	.752	1.789	.1977	.1977	.1977
Task * Subject(Group)	18	7.560	.420				
Texture	2	22.184	11.092	7.462	.0019	.0023	.0019
Texture * group	2	5.343	2.671	1.797	.1803	.1821	.1803
Texture * Subject(Group)	36	53.514	1.486				
Task * Texture	2	.591	.296	1.331	.2769	.2709	.2730
Task * Texture * group	2	.513	.257	1.156	.3262	.3091	.3135
Task * Texture * Subje	36	7.992	.222				

Dependent: Errors

Table of Epsilon Factors for df Adjustment Dependent: Errors

	G-G Epsilon	H-F Epsilon
Task	1.000	1.059
Texture	.957	1.128
Task * Texture	.640	.706

c) RANOVAs ***** Analysis of data in input file: dual data This is output file: dual data out ****** STATISTICS FROM OBSERVED DATA ***** 2 No. of groups: Group sizes: 1 10 10 2 ****** Means over within-subjs factor (cols) for each group (row): Gp 1 118.1615 294.8763 * 106.3995 Gp 2 223.1301 * 101.0945 188.7134 ****** F for Groups: 1.092879E-03 F for Within-subjs: 8.716195 F for Interaction: 1.757292 ***** STATISTICS FROM RANDOMIZED DATA ***** No. of randomizations: 10000 3.483333 mins Duration: ****** Significance of F ratios: No. of randomization F ratios that equal or exceed obtained F, & p-values: .973 9730 Groups: .0005 5 Withn subjs: .1897 1897 Interaction: *****

***** Analysis of data in input file: dual errors This is output file: dual errors output ****** STATISTICS FROM OBSERVED DATA ***** 2 No. of groups: Group sizes: 1 10 2 10 ****** Means over within-subjs factor (cols) for each group (row): Gp 1 1.9401 2.0871 1.3752 * Gp 2 .7554 .9554 1.8164 * ****** F for Groups: .6762314 F for Within-subjs: 7.182094 F for Interaction: 1.157808 ****** STATISTICS FROM RANDOMIZED DATA ***** No. of randomizations: 10000 3.433333 mins Duration: ***** Significance of F ratios: No. of randomization F ratios that equal or exceed obtained F, & p-values: .5785 5785 Groups: .0009 9 Withn subjs: 3424 .3424 Interaction: ******

```
*****
Analysis of data in input file: single thresholds
This is output file: single thresholds
*****
        STATISTICS FROM OBSERVED DATA
******
                 2
No. of groups:
Group sizes:
       10
1
       10
2
******
Means over within-subjs factor (cols) for each group (row):
Gp 1
       152.7595
               246.7984
80.9986
Gp 2
               373.5832
125.2556
        97.7439
*****
F for Groups: .3250186
F for Within-subjs: 11.20406
F for Interaction: 1.795329
*****
       STATISTICS FROM RANDOMIZED DATA
*****
No. of randomizations: 10000
        3.5
           mins
Duration:
*****
Significance of F ratios:
No. of randomization F ratios that equal or exceed obtained F,
& p-values:
                  .5728
         5728
Groups:
                  .0001
Withn subjs:
         1
                  .1777
Interaction:
         1777
*****
```

****** Analysis of data in input file: single errors This is output file: single errors ****** STATISTICS FROM OBSERVED DATA ****** 2 No. of groups: Group sizes: 10 1 2 10 ****** Means over within-subjs factor (cols) for each group (row): Gp 1 1.7919 1.8968 1.001 Gp 2 .7351 .786 2.2424 * ****** F for Groups: .2539323 F for Within-subis: 6.419899 F for Interaction: 1.97759 ****** STATISTICS FROM RANDOMIZED DATA ****** No. of randomizations: 10000 3.483333 mins Duration: ****** Significance of F ratios: No. of randomization F ratios that equal or exceed obtained F. & p-values: .7407 7407 Groups: .0011 11 Withn subjs: .1484 1484 Interaction:

<u>Appendix 3.4.2: with exclusions</u> thresholds

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value	G-G	H-F
group	1	17176.041	17176.041	.889	.3644		
Subject(Group)	12	231927.212	19327.268				
Task	1	614.848	614.848	.044	.8381	.8381	.8381
Task * group	1	384.299	384.299	.027	.8716	.8716	.8716
Task * Subject(Group)	12	169223.463	14101.955				
Texture	2	540924.122	270462.061	27.522	.0001	.0001	.0001
Texture * group	2	59958.564	29979.282	3.051	.0660	.1029	.0974
Texture * Subject(Group)	24	235852.219	9827.176				
Task * Texture	2	15295.460	7647.730	.543	.5879	.4805	.4957
Task * Texture * group	2	1665.277	832.638	.059	.9427	.8198	.8417
Task * Texture * Subje	24	337972.614	14082.192				

Dependent: NOT x 0.6745!

Table of Epsilon Factors for df Adjustment Dependent: NOT x 0.6745!

	G-G Epsilon	H-F Epsilon
Task	1.000	1.091
Texture	.535	.594
Task * Texture	.517	.568

absolute % errors

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value	G-G	H-F
group	1	.056	.056	.048	.8296		
Subject(Group)	12	13.854	1.154				
Task	1	.011	.011	.063	.8068	.8068	.8068
Task * group	1	.001	.001	.005	.9467	.9467	.9467
Task * Subject(Group)	12	2.062	.172				
Texture	2	8.349	4.175	11.275	.0004	.0043	.0030
Texture * group	2	.727	.363	.982	.3892	.3487	.3569
Texture * Subject(Group)	24	8.886	.370				
Task * Texture	2	.323	.162	.853	.4387	.3812	.3914
Task * Texture * group	2	.054	.027	.143	.8676	.7306	.7562
Task * Texture * Subje	24	4.548	.189				

Dependent: Errors

Table of Epsilon Factors for df Adjustment Dependent: Errors

	G-G Epsilon	H-F Epsilon
Task	1.000	1.091
Texture	.551	.616
Task * Texture	.539	.600

```
*****
Analysis of data in input file: ex2 single thresho
This is output file: ex2 single thresh out
******
        STATISTICS FROM OBSERVED DATA
*****
                 2
No. of groups:
Group sizes:
       7
1
       7
2
*****
Means over within-subjs factor (cols) for each group (row):
Gp 1
        78.60442
                320.7616
                        *
74.52271
Gp 2
81.893 85.73486 233.297 *
*******
F for Groups: .2757001
F for Within-subjs: 10.74932
F for Interaction: .6220124
*****
       STATISTICS FROM RANDOMIZED DATA
******
No. of randomizations: 10000
        2.466667
                mins
Duration:
****
Significance of F ratios:
No. of randomization F ratios that equal or exceed obtained F,
& p-values:
Groups:
         5407
                  .5407
                  .0001
Withn subjs:
         1
         6150
                  .615
Interaction:
*****
```

```
******
Analysis of data in input file: ex2 single errors
This is output file: ex2 single errors out
*****
        STATISTICS FROM OBSERVED DATA
******
No. of groups:
                 2
Group sizes:
       7
1
       7
2
******
Means over within-subjs factor (cols) for each group (row):
Gp 1
    .561 1.430571
.442
Gp 2
        .7671428
                1.293286
                        *
.5467142
*******
F for Groups: 4.947044E-02
F for Within-subjs: 8.224869
F for Interaction: .3027945
******
       STATISTICS FROM RANDOMIZED DATA
*****
No. of randomizations: 10000
Duration:
       2.45
            mins
*****
Significance of F ratios:
No. of randomization F ratios that equal or exceed obtained F,
& p-values:
                  .8358
         8358
Groups:
                  .0002
Withn subjs:
         2
         7608
                  .7608
Interaction:
*****
```

```
*****
Analysis of data in input file: ex2 dual thresholds
This is output file: ex2 dual thresholds out
*****
        STATISTICS FROM OBSERVED DATA
*****
                2
No. of groups:
Group sizes:
       7
1
       7
2
*****
Means over within-subjs factor (cols) for each group (row):
Gp 1
76.48328 98.096 295.9096
                     *
Gp 2
               175.1003
                       *
83.83158 112.9266
******
F for Groups: 1.041113
F for Within-subjs: 13.72829
F for Interaction: 2.872428
*****
       STATISTICS FROM RANDOMIZED DATA
*****
No. of randomizations: 10000
       2.466667
                mins
Duration:
******
Significance of F ratios:
No. of randomization F ratios that equal or exceed obtained F,
& p-values:
                 .367
         3670
Groups:
Withn subjs:
         1
                 .0001
         591
Interaction:
                  .0591
```

***** Analysis of data in input file: dual errorstx This is output file: dual errors 98 out ****** STATISTICS FROM OBSERVED DATA ******* 2 No. of groups: Group sizes: 7 1 7 2 ****** Means over within-subjs factor (cols) for each group (row): Gp 1 1.304857 .4498571 .6298572 Gp 2 .8407143 1.023 * .6568571 ****** F for Groups: 3.502133E-02 F for Within-subjs: 6.883419 F for Interaction: 1.406042 ****** STATISTICS FROM RANDOMIZED DATA ***** No. of randomizations: 10000 2.5 mins Duration: ****** Significance of F ratios: No. of randomization F ratios that equal or exceed obtained F. & p-values: .8612 8612 Groups: .0016 16 Withn subjs: .2776 2776 Interaction:

			no.eye mo	vements			
group	u	F	med hi			<u>ר</u>	
	single	dual	single	dual	single	dual	
Category	Real	Real	Real	Real	Real	Real	
User Entered	User Entered	User Entered	User Entered	User Entered	User Entered	User Entered	
Nominal	Continuous	Continuous	Continuous	Continuous	Continuous	Continuous	
•	Free Format Fix	Free Format Fi					
•	3	3	3	3	3	3	
control	2.000	7.583	10.500	10.167	18.000	14.833	
control	13.000	13.667	14.167	13.917	20.333	17.583	
control	14.000	15.583	17.750	16.667	21.250	20.333	
dyslexic	8.000	17.250	16.167	19.083	16.667	16.750	
control	15.000	12.167	14.833	16.000	17.500	12.250	
control	20.000	19.833	19.250	17.500	25.333	28.417	
control	19.000	21.583	25.667	24.833	27.917	25.917	
dyslexic	18.000	26.583	21.667	23.417	22.583	27.750	
control	7.000	11.500	11.500	10.667	13.250	14.583	
dy s lexic	6.000	6.000	8.583	6.750	14.250	11.000	
control	17.000	21.917	17.750	23.500	25.667	19.583	
dyslexic	4.000	8.917	8.583	11.333	12.167	9.000	
dyslexic	3.000	10.500	9.417	9.167	12.750	10.833	
dy slexic	5.000	13.750	13.000	15.167	19.583	21.000	
dyslexic	16.000	24.083	15.083	21.083	20.417	24.250	
dyslexic	9.000	8.917	11.250	11.000	15.917	10.583	
dyslexic	11.000	14.833	13.833	14.583	19.750	20.833	
control	10.000	17.333	12.667	16.917	12.417	14.167	
dyslexic	1.000	11.000	12.083	10.583	18.000	11.500	
control	12.000	16.000	19.500	17.167	20.167	19.583	

Appendix 3.4.3 Eye movement counts: Raw data

No exclusions

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value	G-G	H-F
group	1	257.889	257.889	1.913	.1836		
Subject(Group)	18	2427.093	134.839				
texture	2	584.707	292.354	41.149	.0001	.0001	.0001
texture * group	2	1.216	.608	.086	.9182	.8995	.9182
texture * Subject(Group)	36	255.769	7.105				
task	1	56.147	56.147	7.210	.0151	.0151	.0151
task * group	1	18.736	18.736	2.406	.1383	.1383	.1383
task * Subject(Group)	18	140.178	7.788				
texture * task	2	161.897	80.948	22.859	.0001	.0001	.0001
texture * task * group	2	10.756	5.378	1.519	.2327	.2335	.2327
texture * task * Subjec	36	127.484	3.541				

Dependent: no.eye movements

Table of Epsilon Factors for df Adjustment Dependent: no.eye movements

	G-G Epsilon	H-F Epsilon
texture	.894	1.041
task	1.000	1.059
texture * task	.959	1.130

Exclusions

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value	G-G	H-F
group	1	632.046	632.046	5.996	.0307		
Subject(Group)	12	1265.034	105.420				
texture	2	472.514	236.257	42.391	.0001	.0001	.0001
texture * group	2	2.235	1.117	.200	.8197	.8191	.8197
texture * Subject(Group)	24	133.758	5.573				
task	1	27.333	27.333	3.655	.0801	.0801	.0801
task * group	1	10.894	10.894	1.457	.2507	.2507	.2507
task * Subject(Group)	12	89.739	7.478				
texture * task	2	123.060	61.530	16.145	.0001	.0001	.0001
texture * task * group	2	15.312	7.656	2.009	.1561	.1605	.1561
texture * task * Subjec	24	91.464	3.811				

Dependent: no.eye movements

Table of Epsilon Factors for df Adjustment Dependent: no.eye movements

	G-G Epsilon	H-F Epsilon
texture	.997	1.295
task	1.000	1.091
texture * task	.921	1.171

NOTE: Probabilities are not corrected for values of epsilon greater than 1.

Gul C

<u>Appendix 3.5.1</u> Pooled data: no exclusions a) Comparison of thresholds over the 2 experiments

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value	G-G	H-F
Group	1	7379.715	7379.715	.055	.8155		
expt no.	1	90201.573	90201.573	.677	.4167		
Subject(Group)	32	4264097.226	133253.038				
Textures	2	1558192.605	779096.302	19.471	.0001	.0001	.0001
Textures * Group	2	68381.271	34190.636	.854	.4303	.3926	.4014
Textures * expt no.	2	25291.850	12645.925	.316	.7302	.6445	.6643
Textures * Subject(Gro	64	2560836.954	40013.077				

Dependent: Not x 0.6745

Table of Epsilon Factors for df Adjustment Dependent: Not x 0.6745

	G-G Epsilon	H-F Epsilon
Textures	.676	.739

b) Comparison of absolute % errors

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value	G-G	H-F
Group	1	1.897	1.897	.524	.4746		
expt no.	1	6.780	6.780	1.871	.1809		
Subject(Group)	32	115.946	3.623				
Textures	2	19.007	9.503	10.874	.0001	.0002	.0001
Textures * Group	2	1.321	.660	.756	.4739	.4551	.4681
Textures * expt no.	2	.728	.364	.417	.6611	.6292	.6513
Textures * Subject(Gro	64	55.932	.874				

Dependent: errors

Table of Epsilon Factors for df Adjustment Dependent: errors

	G-G Epsilon	H-F Epsilon
Textures	.853	.952

c) comparison of randot scores

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value
Group	1	2.925	2.925	1.705	.2007
Residual	33	56.618	1.716		

Dependent: Randot Score

<u>Appendix 3.5.2</u>: comparison of 2 expts with exclusions a) thresholds

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value	G-G	H-F
Group	1	42.800	42.800	.002	.9637		
expt no.	1	5132.185	5132.185	.254	.6187		
Subject(Group)	25	505195.333	20207.813				
Textures	2	704486.924	352243.462	21.390	.0001	.0001	.0001
Textures * Group	2	885.023	442.511	.027	.9735	.8923	.9096
Textures * expt no.	2	2802.805	1401.402	.085	.9186	.7984	.8203
Textures * Subject(Gro	50	823365.404	16467.308				

Dependent: Not x 0.6745

Table of Epsilon Factors for df Adjustment

Dependent: Not x 0.6745

	G-G Epsilon	H-F Epsilon
Textures	.554	.608

b) absolute % error

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value	G-G	H-F
Group	1	.001	.001	.003	.9585		
expt no.	1	.161	.161	.323	.5747		
Subject(Group)	25	12.457	.498				
Textures	2	8.501	4.251	17.321	.0001	.0002	.0001
Textures * Group	2	.038	.019	.078	.9253	.8127	.8346
Textures * expt no.	2	.273	.136	.556	.5773	.4829	.4983
Textures * Subject(Gro	50	12.270	.245				

Dependent: errors

Table of Epsilon Factors for df Adjustment Dependent: errors

	G-G Epsilon	H-F Epsilon
Textures	.565	.621

c) randot score

Type III Sums of Squares

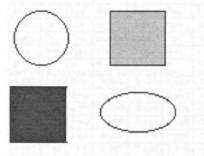
Source	df	Sum of Squares	Mean Square	F-Value	P-Value
Group	1	3.405	3.405	2.344	.1383
expt no.	1	.060	.060	.041	.8406
Residual	25	36.309	1.452		

Dependent: Randot Score

Appendices: Chapter 4

Appendix 4.4.1.

Shapes used in order to replicate Akshoomoff and Courchesne procedure. Actual size.



Appendix 4.4.2

The software was a close replication of Akshoomoff and Courchesne (1994). Differences included:

- 1. Increased probability of a target stimulus (15% instead of 12.5%).
- 2. Increased duration of all stimuli (250 milliseconds instead of 50 milliseconds).
- 3. Different ISIs.
- 4. 350 stimuli were given in a single block in the focus conditions and 700 in the shift condition, rather than splitting conditions into either 5 or 10 blocks of 80 stimuli.
- 5. Main experiment trials were not dependent on any success rate in the practice trials.
- 6. A short tone indicated a correct hit and a reminder to switch to the other target.
- 7. The focus conditions were not counterbalanced.
- 8. Responses counted as hits if they came within 1000 milliseconds (instead of 200 to 1400 msecs) of the target.
- 9. Akshoomoff and Courchesne's analysis used percentage correct hits rather than a d' measure of accuracy.

Appendices: Chapter 4

Appendix 4.4.3 Raw data for analysis

		-		s- realist	1	1					d prim	ies - realis	stic zs					_	-	e - reali				grp	agegr
ero-1	one-2	two-3	three-4	four-5	five-6	six-7	seven+	zero-1	one-2	two-3	three	four-5	five-6	six-7	seven+	zero-1	one-2	two-3	three-4	four-5	five-6	six-7	seven+	2.1	
leal	Real	Real	Real	Real	Real	Real	Real	Real	Real	Real	Real	Real	Real	Real	Real	Real	Real	Real	Real	Real	Real	Real	Real	Catego	Catego
lser	Us	Use	User	User	User	User	User	User	User	User	Use	User E	User	User	User	User E	User	Use	User	User	Use	User	User	User E	User B
ont	Co	Con	Conti	Conti	Cont	Conti	Conti	Conti	Conti	Conti	Con	Contin	Conti	Conti	Conti	Contin	Conti	Con	Cont	Cont	Con	Conti	Conti	Nominal	Nomin
ree	Fre	Fre	Free	Free	Free	Free	Free	Free	Free	Free	Fre	Free F	Free	Free	Free	Free F	Free	Fre	Free	Free	Fre	Free	Free	•	
5	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3	•	
.439	7.44	5.71	5.596	7.439	7.439	7.439	7.439	2.830	7.439	4.787	7.44	4.787	7.439	7.439	4.740	7.439	3.642	7.44	4.870	7.439	7.439	7.439	2.549	control	0
.439	2.74	7.44	4.285	5.506	7.439	7.439	4.607	7.439	3.899	4.150	7.44	4.870	3.719	5.254	5.185	2.158	2.092	5.15	3.039	2.684	2.827	4.394	2.308	dyslexic	0
439	5.49	4.29	5.609	7.439	7.439	-2.155	3.041	4.561	2.401	1.926	4.15	7.439	7.439	7.439	5.146	4.150	2.192	2.8	2.477	1.950	2.703	7.439	2.200	dyslexic	0
.439	2.36	7.44	7.439	7.439	7.439	7.439	5.185	4.561	5.001	4.687	4.79	7.439	7.439	7.439	7.439	3.899	2.486	3.3	2.869	5.055	5.765	5.001	5.488	control	(
.000	5.45	1.64	4.607	1.134	0.000	-2.185	1.186	0.000	3.401	3.045	4.69	4.787	7.439	4.561	4.787	3.154	2.942	1.71	2.481	1.666	-2.07	0.000	1.203	dyslexic	you
.439	7.44	1.9	4.787	7.439	3.719	-2.126	4.324	7.439	4.787	3.719	4.79	4.870	7.439	7.439	7.439	7.439	4.324	3.16	5.867	2.552	5.146	7.439	4.729	dyslexic	you
.439	4.56	4.69	4.394	7.439	2.612	7.439	4.906	7.439	4.394	2.213	4.29	7.439	7.439	7.439	5.185	1.923	2.284	5.95	5.403	5.303	5.451	4.940	2.760	control	you
.596	4.39	4.79	2.265	4.561	4.687	0.000	3.481	3.973	4.394	3.899	7.44	4.787	7.439	4.687	7.439	3.623	4.394	2.93	4.787	4.940	5.001	4.940	4.749	control	you
.439	3.12	7.44	7.439	7.439	3.466	7.439	3.232	7.439	4.285	2.878	7.44	7.439	7.439	4.561	3.375	2.434	4.940	5.05	7.439	5.001	4.561	4.150	2.898	control	
000	3.72	5.41	3.973	7.439	3.045	-2.293	2.264	3.719	4.150	3.719	4.29	4.484	7.439	4.150	4.870	3.719	1.942	4.15	3.719	2.406	4.787	4.285	2.465	dyslexic	you
.553	2.61	1.89	7.439	7.439	4.687	7.439	3.056	7.439	4.285	3.289	2.92	4.687	7.439	4.687	5.146	1.755	1.965	3.07	2.141	4.150	4.456	4.281	3.027	dyslexic	you
.622	4.79	2.52	5.609	5.166	2.636	4.906	3.127	7.439	4.940	7.439	5.55	5.451	7.439	7.439	5.961	5.221	3.274	3.29	5.892	5.322	5.756	7.439	5.237	dyslexic	you
.439	7.44	3.05	4.870	7.439	7.439	7.439	7.439	7.439	7.439	4.150	7.44	4.787	7.439	7.439	7.439	3.649	5.055	7.44	7.439	7.439	7.439	7.439	5.203	control	(
.439	4.79	7.44	7.439	7.439	7.439	7.439	7.439	7.439	4.940	4.150	7.44	7.439	7.439	7.439	7.439	3.084	4.628	7.44	2.627	2.893	7.439	7.439	3.241	dyslexic	0
.439	7.44	7.44	4.786	7.439	7.439	7.439	3.327	4.787	7.439	4.787	7.44	4.787	5.451	7.439	5.102	3.690	4.687	5.93	7.439	5.805	7.439	7.439	6.192	control	0
.582	2.37	1.83	2.237	5.506	2.510	5.339	3.170	4.561	4.870	3.719	7.44	7.439	3.719	4.687	5.146	2.862	3.581	3.13	4.561	3.973	7.439	4.394	4.581	control	you
.439	4.39	4.29	4.038	7.439	4.561	0.000	4.972	7.439	4.150	1.349	2.41	4.394	7.439	4.394	3.547	3.003	2.369	2.35	4.285	2.209	3.262	7.439	1.735	dyslexic	you
.439	5.45	4.29	4.394	7.439	5.388	0.000	3.268	4.150	4.940	4.787	7.44	5.451	7.439	7.439	5.102	2.031	3.536	3.55	3.304	5.001	4.740	7.439	4.000	control	you
.439	7.44	7.44	7.439	7.439	4.687	0.000	7.439	7.439	7.439	4.285	7.44	7.439	7.439	7.439	7.439	2.521	2.356	5.1	5.867	4.940	7.439	4.787	3.136	dyslexic	0
.439	7.44	5.69	7.439	7.439	7.439	7.439	5.185	5.634	7.439	4.687	7.44	7.439	7.439	7.439	7.439	5.185	3.724	7.44	7.439	7.439	7.439	7.439	5.538	control	0
.269	2.71	5.68	2.801	5.506	2.612	0.000	3.223	4.687	5.748	2.636	7.44	7.439	7.439	7.439	5.364	1.579	2.252	2.51	2.191	1.636	3.262	2.599	1.370	dyslexic	0
.582	4.69	3.03	4.787	7.439	3.719	5.339	5.284	7.439	4.870	4.687	7.44	4.687	7.439	4.394	5.102	4.150	2.680	3.69	5.861	4.244	2.904	2.538	2.202	control	you
.439	4.56	3.29	4.687	7.439	4.285	0.000	2.831	7.439	4.285	3.289	7.44	4.285	7.439	4.687	4.740	4.013	1.853	2.8	1.957	2.719	4.648	7.439	3.015	control	you
.439	2.94	7.44	7.439	7.439	7.439	5.221	7.439	4.787	4.787	2.151	7.44	7.439	7.439	7.439	5.970	5.221	6.053	7.44	7.439	7.439	7.439	7.439	3.720	control	(
.439	3.04	2.03	7.439	7.439	7.439	7.439	3.347	7.439	7.439	4.687	4.79	5.488	7.439	7.439	7.439	5.861	2.614	5	3.139	5.044	5.254	4.787	2.511	dyslexic	you
.439	7.44	3.47	7.439	5.166	7.439	0.000	4.687	4.687	4.940	3.973	4.79	4.787	7.439	7.439	4.285	2.024	3.207	3.63	3.321	2.912	2.775	5.055	2.250	control	(
.439	7.44	4.79	3.013	7.439	7.439	-2.126	4.906	4.687	7.439	4.150	7.44	4.787	5.451	7.439	3.690	3.667	6.057	5.93	5.403	2.956	5.773	7.439	2.919	dyslexic	
.439	4.94	5.69	7.439	7.439	4.687	7.439	4.648	7.439	4.870	4.150	7.44	7.439	7.439	7.439	5.102	3.094	2.029	5.63	5.602	3.146	7.439	4.285	2.841	dyslexic	0

20-

			foc 1 fas			foc 2 fas	5		switch	FAs	
Group	age grp	circle	dark blues	light blue	oval	circle	light sq	oval	circle	darksq	1ightsq
Categ	Catego	Real	Real	Rea1	Real	Real	Real	Real	Real	Real	Real
User	User E	User E	User E	User Ent	User	User	User E	User E	User	User E	Use
Nominal	Nominal	Contin	Contin	Continuous	Cont	Contin	Contin	Continu	Conti	Contin	Con
٠	•	Free F	Free F	Free For	Free	Free F	Free F	Free Fo	Free	Free F	Fre
•	٠	3	3	3	3	3	3	3	3	3	3
control	old	2.000	0.000	0.000	0.04	0.000	1.000	2.000	0.000	2.000	0.000
dysle	old	1.000	0.000	1.000	0.000	1.000	0.000	9.000	1.000	10.000	0.000
dy sle	old	5.000	0.000	0.000	0.000	0.000	2.000	12.000	5.000	13.000	5.000
control	old	0.000	0.000	0.000	0.000	0.000	0.000	5.000	1.000	3.000	2.000
dysle	young	17.000	0.000	0.000	0.000	0.000	0.000	4.000	7.000	8.000	3.000
dysle	young	2.000	0.000	0.000	0.000	0.000	0.000	0.000	1.000	2.000	0.000
control	young	1.000	0.000	0.000	1.000	0.000	0.000	5.000	2.000	8.000	5.000
control	young	3.000	0.000	0.000	0.000	0.000	0.000	1.000	0.000	1.000	0.000
control	old	1.000	0.000	1.000	0.000	1.000	0.000	2.000	0.000	2.000	0.000
dysle	young	3.000	0.000	0.000	0.000	0.000	0.000	2.000	1.000	1.000	1.000
dysle	young	4.000	0.000	1.000	0.000	0.000	1.000	4.000	5.000	3.000	1.000
dysle	young	14.000	1.000	0.000	0.000	2.000	1.000	1.000	4.000	0.000	3.000
control	old	1.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	1.000	0.000
dysle	old	0.000	0.000	0.000	0.000	0.000	0.000	4.000	0.000	6.000	1.000
control	old	1.000	0.000	0.000	0.000	1.000	0.000	3.000	0.000	1.000	0.000
control	young	14.000	2.000	2.000	0.000	0.000	0.000	1.000	2.000	1.000	0.000
dysle	young	0.000	0.000	0.000	0.000	1.000	3.000	4.000	1.000	14.000	1.000
control	young	4.000	0.000	0.000	0.000	0.000	1.000	2.000	0.000	2.000	1.000
dysle	old	0.000	0.000	0.000	0.000	0.000	0.000	0.000	1.000	6.000	1.000
control	old	1.000	0.000	0.000	0.000	0.000	1.000	1.000	0.000	0.000	0.000
dysle	old	5.000	3.000	2.000	5.000	2.000	0.000	9.000	11	13.000	9.000
control	young	2.000	0.000	1.000	0.000	0.000	0.000	6.000	3.000	4.000	1.000
control	young	1.000	0.000	0.000	0.000	0.000	0.000	8.000	2.000	0.000	0.000
control	old	2.000	0.000	1.000	1.000	0.000	1.000	0.000	0.000	0.000	1.000
dysle	young	4.000	0.000	1.000	0.000	0.000	1.000	5.000	1.000	9.000	4.000
control	old	2.000	0.000	0.000	0.000	0.000	0.000	7.000	3.000	6.000	0.000
dysle	old	2.000	0.000	0.000	0.000	1.000	1.000	5.000	2.000	4.000	0.000
dysle	old	1.000	0.000	0.000	0.000	0.000	0.000	8.000	1.000	5.000	2.000

Ē			fo	cus 1 H	lit laten	cu					f(ocus 2 Hit	latenci				[switch H	lit latenc						Γ
	zero-1	one-2	*wo-3	thre	four-5	five-6	six-7	seven+	zero-1	one-2	two-3	three-4	four-5	five-6	six-7	sevent	zero-1	one-2		three-4		five-6	six-7	seven+	Group	age grp	5
e:	Real	Real	Real	Real	Real	Real	Real	Real	Real	Real	Real	Real	Real	Real	Real	Real	Real	Real	Real	Real	Real	Real	Real	Real	Categ	Catego	Re
e:	User	User	User	Use	Use	User	Use	User E	User	Use	User	User E	Use	User	Use	Use	Use	User	Use	User	User	User	Us	User	User	User E	Us
s :	Cont	Conti	Conti	Con	Con	Cont	Con	Contin	Conti	Con	Conti	Contin	Con	Conti	Cont	Con	Cont	Conti	Con	Conti	Conti	Conti	Co	Contin	Nominal	Nominal	Cc
t:	Free	Free	Free	Fre	Fre	Free	Fre	Free F	Free	Fre	Free	Free F	Fre	Free	Free	Fre	Free	Free	Fre	Free	Free	Free	Fre	Free F	•	•	Fr
s :	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3	•	•	3
1	.592	.538	.531	.564	.529	.567	.533	.540	.560	.588	.622	.588	.614	.542	.571	.655	.553	.572	.608	.555	.585	.570	.556	.587	control	old	\Box
2	1.000	.700	.639	.633	.622	.728	.733	.681	.636	.617	.596	.569	.664	.733	.667	.705	.704	.619	.626	.606	.617	.617	.617	.676	dysle	bĩo	Γ
3	.700	.650	.623	.650	.650	.673	•	.688	.579	.564	.644	.588	.603	.650	.630	.628	.775	.614	.627	.573	.575	.593	.613	.637	dysle	old	
4	.608	.636	.564	.571	.583	.531	.533	.540	.717	.631	.637	.611	.676	.639	.558	.689	.790	.731	.732	.600	.637	.642	.641	.702	control	old	1
5	•	.742	.900	.633	.767	•	٠	.676	٠	.617	.650	.603	.639	.683	.542	.638	.575	.562	.597	.546	.681	.561	•	.635	dysle	young	1
6	.700	.714	.817	.636	.658	.667	٠	.634	.560	.625	.639	.592	.605	.550	.587	612	.570	.612	.628	.567	.583	.576	.572	.616	dysle	young	Ľ
4	.892	.592	.583	.533	.544	.627	.450	.576	.594	.631	.689	.610	.669	.633	.679	.672	.685	.675	.627	.619	.641	.656	.646	.701	control	young	L
8	.867	.817	.581	.539	.579	.487	•	.535	.633	.614	.588	.606	.558	.683	.583	.625	.500	.578	.518	.506	.527	.537	.548	.571	control	young	Ľ
2	.525	.550	.510	.622	.608	.608	.608	.586	.550	.507	.683	.578	.602	.658	.5%	608	.578	.562	.595	.588	.587	.567	.586	.626	control	old	L
힉	0/7	.725	.775	.717	.756	.800	•	.778	.775	.542	.600	.833	.695	.675	.675	.665	.756	.650	.714	.629	.689	.792	.760	.826	dysle	young	<u> </u> -'
 2	.867	.627	.656	.672	.672	.670	.650	.723	.561	.597	.583	.700	.673	.628	.603	.672	.654	.638	.665	.683	.625	.700	.682	.692	dysle	young	H-
싉	1.017 .808	.633 .540	.697 .608	.594 .531	.600 .511	.617	.633 .467	.712	.574	.558	.536	.508	.542 .575	.592	.529 .537	.582 .542	.586 .532	.570	.592 .550	.567 .512	.583 .518	.556	.607 .531	.597	dysie	young old	Ľ
-	.558	.525	.554	.538	.550	.567	.567	.556	.476	.511	.558	.508	.512	.308	.507	.592	.550	.545	.583	.540	.510	.475	.557	.557	control dysle	oid	F.
╡	.600	.510	.525	.530	.531	.531	.500	.510	.611	.565	.514	.486	.481	.533	.507	.526	.569	.505	.483	.483	.485	.498	.553	.531	control	old	F
6	.808.	.653	.713	.672	.653	.621	.583	.686	.604	.602	.572	.603	.627	.550	.630	.603	.619	.620	.630	.542	.533	.588	.597	.561	control	uouna	1
7	.733	.700	.663	.623	.683	.696	•	.645	.547	.579	.667	.607	.697	.728	.639	.6%	.604	.642	.625	.580	.578	.619	.643		dysie	young	H
8	.650	.550	.617	.581	.567	.510	•	.502	.5%	.556	.503	.490	.533	.506	.479	.508	.572	.581	.512	.486	.498	.533	.508	.520	control	young	F.
9	.658	.600	.594	.595	.619	.643	•	.592	.545	.562	.613	.531	.517	.533	.530	.604	.611	.5%	.600	.593	.581	.593	.600	.585	dysle	old	F
20	.500	.465	.438	.478	.448	.450	.417	.438	.431	.456	.430	.450	.424	.617	.430	.450	.445	.439	.433	.423	.430	.430	.440	.471	control	old	
21	.908	.600	.603	.550	.786	.630	•	.646	.583	.525	.653	.638	.507	.558	.550	.600	.590	1.486	.560	.546	4.033	.733	.607	.640	dysie	old	
22	.467	.600	.456	.625	.560	.483	.450	.486	.538	.614	.570	.611	.600	.758	.611	.602	.675	.662	.551	.4%	.574	.590	.667	.560	control	young	
23	.783	.654	.650	.713	.693	.663	٠	.679	.543	.597	.617	.652	.627	.672	.643	.674	.581	.5%	.608	.614	.602	.574	.585	.651	control	young	
24	.583	.569	.545	.538	.578		.600	.549	.525	.592	.550	.583	.585	.542	.527	.562	.515	.518	.554	.490	.525	.531	.521	.550	control	oid	
25	.600	.597	.703	.604	.556		.583	.608	.748	.689	.747	.692	.742	.683	.792	.722	.681	.640	.704	.700	.589	.640	.631	.622	dysie	young	Ē
<u>`6</u>	.617	.514	.575	.517	.531	.533	•		.617	.481	.494	.583	.567	.517	.550	.527	.587	.569	.541	.521	.502	.543	.510	.592	control	bfo	Ŀ
?7	.700	.680	.706	.643	.644	+	•	.663	.683		.621	.553	.603	.575	.613	.655	.613	.643	.605	.571	.575	.612	-	.615	dysle	old	
28	.517	.567	.570	.564	.567	.553	.483	.633	.562	.579	.575	.579	.608	.683	.593	.609	.640	.588	.633	.583	.600	.592	.590	.613	dysle	old	
	2.2 ⁶ .77	100	2.20	1.00	125	75		1.54	1945 C.	l Pro	2.5	- 39.47		12.4	10-50¢	192	1.00	1000	1.00	1.2.5	100	1.00	1.00	1.37	1000		

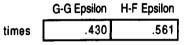
a) Focus Attention Condition 1: d prime analysis

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value	G-G	H-F
Group	1	23.408	23.408	2.795	.1076		
age grp	1	164.257	164.257	19.611	.0002		
Group * age grp	1	3.681	3.681	.440	.5137		
Subject(Group)	24	201.022	8.376				
times	7	224.551	32.079	8.779	.0001	.0001	.0001
times * Group	7	30.252	4.322	1.183	.3152	.3224	.3234
times * age grp	7	27.975	3.996	1.094	.3695	.3575	.3638
times * Group * age grp	7	27.891	3.984	1.090	.3716	.3589	.3653
times * Subject(Group)	168	613.871	3.654				

Dependent: focus1 Accuracy - realistic z's used!

Table of Epsilon Factors for df Adjustment Dependent: focus1 Accuracy - realistic z's used!



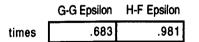
b) Focus Attention Condition 2: d prime analysis

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value	G-G	H-F
Group	1	1.269	1.269	.324	.5746		
age grp	1	19.338	19.338	4.933	.0360		
Group * age grp	1	1.068	1.068	.272	.6065		
Subject(Group)	24	94.086	3.920				
times	7	173.801	24.829	15.530	.0001	.0001	.0001
times * Group	7	12.593	1.799	1.125	.3495	.3505	.3497
times * age grp	7	23.466	3.352	2.097	.0464	.0737	.0478
times * Group * age grp	7	23.022	3.289	2.057	.0508	.0789	.0522
times * Subject(Group)	168	268.585	1.599				

Dependent: focus2 Accuracy - realistic z's used!

Table of Epsilon Factors for df Adjustment Dependent: focus2 Accuracy - realistic z's used!



c) Shift attention condition: d prime analysis

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value	G-G	H-F
Group	1	50.609	50.609	5.197	.0318		[
age grp	1	34.285	34.285	3.521	.0728		
Group * age grp	1	19.578	19.578	2.010	.1691		
Subject(Group)	24	233.718	9.738				
times	7	131.752	18.822	11.912	.0001	.0001	.0001
times * Group	7	17.734	2.533	1.603	.1376	.1623	.1376
times * age grp	7	21.687	3.098	1.961	.0632	.0872	.0632
times * Group * age grp	7	9.739	1.391	.880	.5232	.4987	.5232
times * Subject(Group)	168	265.458	1.580				

Dependent: switch Accuracy - realistic z's used!

Table of Epsilon Factors for df Adjustment Dependent: switch Accuracy - realistic z's used!

G-G Epsilon H-F Epsilon times .734 1.075

NOTE: Probabilities are not corrected for values of epsilon greater than 1.

d) Focus Attention condition 1: Reaction Time Analysis

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value	G-G	H-F
Group	1	.087	.087	2.709	.1280		
age grp	1	.055	.055	1.718	.2167		
Group * age grp	1	.001	.001	.028	.8706		
Subject(Group)	11	.354	.032				
times	7	.124	.018	4.382	.0004	.0300	.0136
times * Group	7	.017	.002	.618	.7391	.5320	.5925
times * age grp	7	.014	.002	.506	.8273	.5908	.6626
times * Group * age grp	7	.010	.001	.355	.9254	.6830	.7658
times * Subject(Group)	77	.310	.004				

Dependent: one Focus Hit Latencies

Table of Epsilon Factors for df Adjustment Dependent: one Focus Hit Latencies

	G-G Epsilon	H-F Epsilon
times	.256	.386

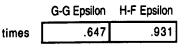
e) Focus Attention Condition 2: Reaction Time Analysis

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value	G-G	H-F
Group	1	.061	.061	2.872	.1036		
age grp	1	.123	.123	5.763	.0249		
Group * age grp	1	7.445E-5	7.445E-5	.003	.9534		
Subject(Group)	23	.491	.021				
times	7	.030	.004	2.001	.0580	.0913	.0633
times * Group	7	.008	.001	.530	.8109	.7360	.7989
times * age grp	7	.010	.001	.679	.6897	.6261	.6791
times * Group * age grp	7	.012	.002	.778	.6067	.5564	.5982
times * Subject(Group)	161	.345	.002				

Dependent: two Focus Hit Latencies

Table of Epsilon Factors for df Adjustment Dependent: two Focus Hit Latencies



f) Shift attention condition: reaction time analysis

Type III Sums of Squares

Source df		df Sum of Squares Me		F-Value	P-Value	G-G	H-F
Group	1	.156	.156	6.668	.0170		
age grp	1	.062	.062	2.639	.1185		
Group * age grp	1	8.000E-7	8.000E-7	3.411E-5	.9954		
Subject(Group)	22	.516	.023				
times	7	.056	.008	8.217	.0001	.0001	.0001
times * Group	7	.007	.001	.984	.4454	.4219	.4378
times * age grp	7	.008	.001	1.149	.3353	.3388	.3378
times * Group * age grp	7	.010	.001	1.441	.1927	.2262	.2058
times * Subject(Group)	154	.149	.001				

Dependent: switch Hit latency

Table of Epsilon Factors for df Adjustment Dependent: switch Hit latency

	G-G Epsilon	H-F Epsilon
times	.584	.831

Appendix 5.1.1: Accuracy ANOVAs

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		accu	iracy				┝
group	norn	nal	degr	aded	age	which first?	—
	focus	shift	focus	shift			
Category	Real	Real	Real	Real	Category	Real	Re
User Entered	User Entered	User Ente	User Entered	User Entered	User Entered	User Entered	Us
Nominal	Continuous	Continuous	Continuous	Continuous	Nominal	Continuous	Co
•	Free Format	Free For	Free Forma	Free Forma	•	Free Format Fi	Fri
•	8	8	8	8	•	0	3
Dyslexic	3.33529897	3.3879769	3.10047199	3.08891003	old	2	Γ
Control	5.77321771	3.8702319	3.43556167	3.45443368	old	1	
Dyslexic	3.04028163	3.4291497	3.30545845	2.95305199	oid	2	
Dyslexic	2.82594101	2.5018198	2.34848812	2.18079322	young	2	
Dyslexic	3.56259989	2.7600117	3.03056368	2.55538225	young	1	
Control	4.10749635	4.556241	3.16234036	3.29573140	young	1	
Control	2.87038574	3.0796969	3.10047199	2.91668584	young	1	
Control	3.92077709	3.7817631	3.24151188	3.43039574	old	1	
Dyslexic	2.42765964	2.2239681	2.37038421	2.28761564	young	1	
Dyslexic	3.03056368	2.6574935	2.81014763	2.17319325	young	2	
Dyslexic	2.39115707	2.5328541	2.72108082	2.81176654	young	2	
Dyslexic	2.87974217	2.7558099	3.28586339	2.93070570	old	2	
Control	3.16234036	2.9819898	2.79565484	2.23294137	young	2	
Control	2.82594101	3.2440516	2.79615506	2.58142563	y oung	1	
Dyslexic	2.58006139	2.1827918	2.61076366	2.36529331	old	1	
Control	3.50137270	2.4001815	2.63509264	3.08891003	old	2	_
Dyslexic	2.53701955	2.9615148	3.56259989	2.61173682	old	1	
Control	3.33529897	3.1029231	3.36503490	2.71113208	young	2	
Control	3.05577714	2.9063835	2.80158247	2.58457476	young	2	
Dyslexic	3.71064743	2.7060241	3.10047199	2.83866257	old	1	
Control	3.56259989	5.1274219	5.53138307	3.23685072	old	2	
Dyslexic	2.54719225	2.5642908	3.34887773	2.44449893	young	1	
Control	6.04581146	3.5015955	4.01370926	2.84933776	old	2	
Control	3.57029194	3.1257082	3.28586339	2.89483523	young	2	
Dyslexic	3.50137270	3.2039134	2.87038574	2.51096253	young	1	
Dyslexic	2.35115522	2.6120824	2.96111011	2.23389634	young	2	
Control	3.15576017	3.4852201	3.19962510	2.89668833	young	1	

Source	df	Sum of Squares	Mean Square	F-Value	P-Value	G-G	H-F
group	1	10.681	10.681	21.227	.0001		
age	1	6.781	6.781	13.476	.0013		
group * age	1	1.137	1.137	2.259	.1464		
Subject(Group)	23	11.573	.503				
visibility	1	2.089	2.089	12.278	.0019	.0019	.0019
visibility * group	1	1.378	1.378	8.100	.0091	.0091	.0091
visibility * age	1	.031	.031	.180	.6753	.6753	.6753
visibility * group * age	1	.304	.304	1.788	.1943	.1943	.1943
visibility * Subject(Gro	23	3.912	.170				
condition	1	2.981	2.981	20.383	.0002	.0002	.0002
condition * group	1	.125	.125	.853	.3652	.3652	.3652
condition * age	1	.407	.407	2.784	.1088	.1088	.1088
condition * group * age	1	.651	.651	4.453	.0459	.0459	.0459
condition * Subject(Gro	23	3.364	.146				
visibility * condition	1	.121	.121	.406	.5301	.5301	.5301
visibility * condition *	1	.058	.058	.195	.6627	.6627	.6627
visibility * condition *	1	.132	.132	.443	.5122	.5122	.5122
visibility * condition *	1	.175	.175	.586	.4519	.4519	.4519
visibility * condition *	23	6.866	.299				

Accuracy analysis: 4 factor ANOVA

Type III Sums of Squares

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Dependent: accuracy

Table of Epsilon Factors for df Adjustment Dependent: accuracy

	G-G Epsilon	H-F Epsilon
visibility	1.000	1.136
condition	1.000	1.136
visibility * condition	1.000	1.136

NOTE: Probabilities are not corrected for values of epsilon greater than 1.

Accuracy analysis: normal focus condition

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value
group	1	6.342	6.342	14.385	.0009
age	1	3.574	3.574	8.105	.0091
group * age	1	2.020	2.020	4.582	.0431
Residual	23	10.141	.441		

Dependent: focus 1 d'

NOTE: One row has been excluded from calculations because of missing values.

Accuracy analysis: normal shift condition

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value
group	1	3.700	3.700	11.468	.0025
age	1	.790	.790	2.447	.1314
group * age	1	.039	.039	.119	.7328
Residual	23	7.421	.323		

Dependent: shift1d'

NOTE: One row has been excluded from calculations because of missing values.

Accuracy analysis: degraded focus condition

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value
group	1	1.217	1.217	4.299	.0495
age	1	1.827	1.827	6.454	.0183
group * age	1	.204	.204	.721	.4045
Residual	23	6.510	.283		

Dependent: focus 2 d'

NOTE: One row has been excluded from calculations because of missing values.

Accuracy analysis: degraded shift condition

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value
group	1	.983	.983	13.760	.0012
age	1	1.161	1.161	16.254	.0005
group * age	1	.004	.004	.056	.8158
Residual	23	1.642	.071		

Dependent: shift 2 d'

NOTE: One row has been excluded from calculations because of missing values.

Accuracy analysis: normal conditions

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value	G-G	H-F
group	1	9.866	9.866	20.919	.0001		
age	1	3.861	3.861	8.188	.0088		
group * age	1	1.308	1.308	2.774	.1093		
Subject(Group)	23	10.847	.472				
condition	1	.950	.950	3.253	.0844	.0844	.0844
condition * group	1	.177	.177	.606	.4443	.4443	.4443
condition * age	1	.502	.502	1.719	.2028	.2028	.2028
condition * group * age	1	.750	.750	2.571	.1225	.1225	.1225
condition * Subject(Gro	23	6.715	.292				

Dependent: normal

Table of Epsilon Factors for df Adjustment Dependent: normal

	G-G Epsilon	H-F Epsilon
condition	1.000	1.136

NOTE: Probabilities are not corrected for values of epsilon greater than 1.

Accuracy analysis: degraded conditions

Type III Sums of Squares

df	Sum of Squares	Mean Square	F-Value	P-Value	G-G	H-F
1	2.193	2.193	10.875	.0031		
1	2.950	2.950	14.628	.0009		
1	.132	.132	.657	.4259		
23	4.638	.202				
1	2.152	2.152	14.086	.0010	.0010	.0010
1	.006	.006	.041	.8415	.8415	.8415
1	.038	.038	.246	.6245	.6245	.6245
1	.076	.076	.495	.4889	.4889	.4889
23	3.514	.153				
	1 1 23 1 1 1 1	1 2.193 1 2.950 1 132 23 4.638 1 2.152 1 .006 1 .038 1 .076	1 2.193 2.193 1 2.950 2.950 1 1.132 1.132 23 4.638 .202 1 2.152 2.152 1 .006 .006 1 .038 .038 1 .076 .076	1 2.193 2.193 10.875 1 2.950 2.950 14.628 1 .132 .132 .657 23 4.638 .202 1 1 2.152 2.152 14.086 1 .006 .006 .041 1 .038 .038 .246 1 .076 .076 .495	1 2.193 2.193 10.875 .0031 1 2.950 2.950 14.628 .0009 1 .132 .132 .657 .4259 23 4.638 .202	1 2.193 2.193 10.875 .0031 1 2.950 2.950 14.628 .0009 1 .132 .132 .657 .4259 23 4.638 .202

Dependent: degraded

Table of Epsilon Factors for df Adjustment Dependent: degraded

	G-G Epsilon	H-F Epsilon
condition	1.000	1.136

Accuracy analysis: focus conditions

Type III Sums of Squares

df	Sum of Squares	Mean Square	F-Value	P-Value	G-G	H-F
1	6.557	6.557	17.589	.0003		
1	5.255	5.255	14.097	.0010		
1	1.754	1.754	4.706	.0406		
23	8.575	.373				
1	.602	.602	1.713	.2035	.2035	.2035
1	1.002	1.002	2.852	.1048	.1048	.1048
1	.145	.145	.413	.5267	.5267	.5267
1	.470	.470	1.339	.2591	.2591	.2591
23	8.076	.351				
	1 1 23 1 1 1 1	1 6.557 1 5.255 1 1.754 23 8.575 1 .602 1 1.002 1 .145 1 .470	1 6.557 6.557 1 5.255 5.255 1 1.754 1.754 23 8.575 .373 1 .602 .602 1 1.002 1.002 1 .145 .145 1 .470 .470	1 6.557 6.557 17.589 1 5.255 5.255 14.097 1 1.754 1.754 4.706 23 8.575 .373 1 1 6.602 1.713 1 1 1.002 1.002 2.852 1 .145 .145 .413 1 .470 .470 1.339	1 6.557 6.557 17.589 .0003 1 5.255 5.255 14.097 .0010 1 1.754 1.754 4.706 .0406 23 8.575 .373	1 6.557 6.557 17.589 .0003 1 5.255 5.255 14.097 .0010 1 1.754 1.754 4.706 .0406 23 8.575 .373

Dependent: focus

Table of Epsilon Factors for df Adjustment Dependent: focus

	G-G Epsilon	H-F Epsilon
visibility	1.000	1.136

NOTE: Probabilities are not corrected for values of epsilon greater than 1.

Accuracy analysis: shift conditions

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value	G-G	H-F
group	1	4.248	4.248	15.359	.0007		
age	1	1.932	1.932	6.986	.0145		
group * age	1	.034	.034	.121	.7306		
Subject(Group)	23	6.362	.277				
visibility	1	1.608	1.608	13.692	.0012	.0012	.0012
visibility * group	1	.435	.435	3.700	.0669	.0669	.0669
visibility * age	1	.018	.018	.152	.7005	.7005	.7005
visibility * group * age	1	.009	.009	.076	.7857	.7857	.7857
visibility * Subject(Gro	23	2.702	.117				

Dependent: shift

Table of Epsilon Factors for df Adjustment Dependent: shift

	G-G Epsilon	H-F Epsilon
visibility	1.000	1.136

Appendix 5.1.2: Bias ANOVAs

	╟	i	bi	as			f
group	E	nor	mal	degr	aded		
	1	focus	shift	focus	shift	square	
Category	1	Real	Real	Real	Real	Real	Rea
User Entered	1	User Entered	Use				
Nominal		Continuous	Continuous	Continuous	Continuous	Continuous	Cor
•]i	Free Format Fi	Fre				
•		3	3	3	3	3	3
Dyslexic	16	386	155	145	376	1.000	\square
Control	19	.833	310	163	328	1.000	
Dyslexic	19	440	.157	098	147	0.000	
Dyslexic	9	641	444	707	611	1.000	
Dyslexic	8	031	319	.039	398	2.000	
Control	1	0.000	.033	300	162	1.000	
Control	19	260	466	145	320	1.000	
Control	8	210	688	339	397	2.000	
Dyslexic	57	746	844	413	730	3.000	
Dyslexic	2	.039	091	0.000	333	4.000	
Dyslexic	3	280	292	079	405	5.000	
Dyslexic	53	158	197	238	379	2.000	
Control	2	300	322	116	522	0.000	
Control	19	641	710	483	586	1.000	
Dyslexic	53	115	300	390	242	4.000	
Control	8	0.000	372	237	376	1.000	
Dyslexic	80	427	337	031	414	1.000	
Control	1	386	299	277	521	0.000	
Control	F	353	430	559	618	1.000	
Dyslexic	81	105	317	145	305	0.000	
Control	98	031	245	.954	343	1.000	
Dyslexic	2	099	090	.076	161	5.000	
Control	1	.697	421	.047	244	0.000	
Contro)	6	791	360	238	467	0.000	
Dyslexic	18	0.000	213	260	392	1.000	
Dyslexic	9	469	358	400	504	3.000	
Control	2	173	173	045	334	2.000	

Raw data: age group as above (order same)

Source	df	Sum of Squares	Mean Square	F-Value	P-Value	G-G	H-F
group	1	.054	.054	.413	.5267		
age	1	.686	.686	5.224	.0318		
group * age	1	.177	.177	1.349	.2574		
Subject(Group)	23	3.020	.131				
visibility	1	.019	.019	.495	.4888	.4888	.4888
visibility * group	1	.002	.002	.052	.8218	.8218	.8218
visibility * age	1	.004	.004	.102	.7522	.7522	.7522
visibility * group * age	1	.010	.010	.260	.6147	.6147	.6147
visibility * Subject(Gro	23	.883	.038				
condition	1	.955	.955	34.445	.0001	.0001	.0001
condition * group	1	.309	.309	11.137	.0029	.0029	.0029
condition * age	1	.191	.191	6.880	.0152	.0152	.0152
condition * group * age	1	.514	.514	18.545	.0003	.0003	.0003
condition * Subject(Gro	23	.638	.028				
visibility * condition	1	.038	.038	.881	.3576	.3576	.3576
visibility * condition *	1	.090	.090	2.097	.1611	.1611	.1611
visibility * condition *	1	.073	.073	1.694	.2060	.2060	.2060
visibility * condition *	1	.107	.107	2.482	.1288	.1288	.1288
visibility * condition *	23	.989	.043				

Dependent: bias

Table of Epsilon Factors for df Adjustment Dependent: blas

	G-G Epsilon	H-F Epsilon
visibility	1.000	1.136
condition	1.000	1.136
visibility * condition	1.000	1.136

NOTE: Probabilities are not corrected for values of epsilon greater than 1.

Means Table Effect: visibility * condition * group Dependent: bias

normal, focus, Dyslexic normal, focus, Control normal, shift, Dyslexic normal, shift, Control degraded, focus, Dyslexic degraded, focus, Control degraded, shift, Dyslexic degraded, shift, Control

Count	Mean	Std. Dev.	Std. Error
14	275	.246	.066
13	124	.458	.127
14	271	.224	.060
13	366	.196	.054
14	199	.218	.058
13	146	.369	.102
14	385	.157	.042
13	401	.134	.037

		Rts correct FAs											correct		
	group	st?	normal			degraded		normal		aded					
			focus	shift	focus	shift	focus	shift	focus	shift					
1	Category		Real	Real											
ī	User Entere	₽d	User Entered	User Entered											
ſ	Nominal		Continuous	Continuous											
	•	t Fi	Free Format Fi	Free Format Fi	Free Format Fi	Free Format Fi	Free Format Fi	Free Format Fi	Free Format Fi	Free Format Fi					
•	•		3	3	3	3	3	3	3	3					
	Dyslexid	2	.559	.571	.655	.598	.279	.538	.263	.406					
	Contro	1	.535	.550	.615	.610	.492	.480	.278	.429					
	Dyslexio	2	.639	.583	.634	.624	.403	.576	.379	.559					
	Dyslexi	2	.606	.618	.625	.701	.271	.462	.314	.469					
	Dyslexio	1	.534	.531	.564	.544	.488	.636	.552	.518					
	Contro	1	.577	.519	.531	.548	.467	.467	.539	.601					
	Contro	1	.575	.547	.572	.588	.359	.431	.426	.459					
	Contro	1	.557	.530	.578	.588	.394	.500	.197	.448					
	Dyslexi	1	.673	.668	.667	.666	.380	.587	.361	.457					
	Dyslexi	2	.631	.592	.688	.635	.445	.475	.298	.482					
	Dyslexio	2	.688	.621	.696	.672	.345	.533	.422	.563					
	Dyslexio	2	.643	.601	.622	.600	.412	.496	.372	.533					
	Contro	2	.609	.578	.697	.647	.244	.486	.291	.472					
	Contro	1	.544	.500	.554	.509	.175	.450	.247	.639					
	Dyslexio	1	.660	.602	.701	.657	.524	.648	.339	.533					
	Contro	2	.593	.842	.633	.635	.298	.878	.404	.398					
	Dyslexio	1	.561	.493	.578	.555	.457	.518	.205	.480					
	Contro	2	.667	.675	.636	.685	.492	.922	.520	.621					
	Contro	2	.618	.598	.691	.618	.264	.636	.237	.541					
	Dyslexic	1	.578	.569	.638	.692	.383	.524	.337	.581					
	Contro	2	.562	.545	.576	.608	.419	.167	.398	.608					
	Dyslexia	1	.592	.596	.642	.635	.394	.544	.297	.462					
	Contro	2	.528	.563	.577	.619	.342	.375	.263	.511					
	Contro	2	.567	.566	.600	.575	.150	.427	.261	.436					
	Dyslexic		.560	.565	.655	.691	.358	.567	.328	.678					
	Dyslexia	2		.658	.720		.330	.488	.342	.529					
	Contro	1	.611	.587	.604	.606	.358	.461	.482						
									a seguration of the second	and the state of the state of the					

Appendix 5.1.3: Reaction Times - raw data - age group as above

3,c

Source	df	Sum of Squares	Mean Square	F-Value	P-Value	G-G	H-F
group	1	.035	.035	2.078	.1629		
age	1	.010	.010	.597	.4476		
group * age	1	2.296E-5	2.296E-5	.001	.9708		
Subject(Group)	23	.385	.017				
RTs	1	1.506	1.506	144.156	.0001	.0001	.0001
RTs * group	1	.001	.001	.072	.7904	.7904	.7904
RTs * age	1	2.324E-5	2.324E-5	.002	.9628	.9628	.9628
RTs * group * age	1	.008	.008	.758	.3929	.3929	.3929
RTs * Subject(Group)	23	.240	.010				
visibility	1	.001	.001	.148	.7042	.7042	.7042
visibility * group	1	.001	.001	.121	.7309	.7309	.7309
visibility * age	1	.011	.011	1.767	.1968	.1968	.1968
visibility * group * age	1	3.048E-4	3.048E-4	.051	.8239	.8239	.8239
visibility * Subject(Group)	23	.138	.006				
condition	1	.315	.315	54.883	.0001	.0001	.0001
condition * group	1	.002	.002	.289	.5961	.5961	.5961
condition * age	1	4.949E-5	4.949E-5	.009	.9268	.9268	.9268
condition * group * age	1	3.880E-6	3.880E-6	.001	.9795	.9795	.9795
condition * Subject(Group)	23	.132	.006				
RTs * visibility	1	.037	.037	8.201	.0088	.0088	.0088
RTs * visibility * group	1	.009	.009	1.901	.1812	.1812	.1812
RTs * visibility * age	1	.013	.013	2.803	.1076	.1076	.1076
RTs * visibility * group * age	1	3.683E-4	3.683E-4	.081	.7786	.7786	.7786
RTs * visibility * Subject(Gro	23	.105	.005				
RTs * condition	1	.366	.366	88.616	.0001	.0001	.0001
RTs * condition * group	1	.003	.003	.742	.3979	.3979	.3979
RTs * condition * age	1	.006	.006	1.371	.2537	.2537	.2537
RTs * condition * group * age	1	.015	.015	3.717	.0663	.0663	.0663
RTs * condition * Subject(Gro	23	.095	.004				
visibility * condition	1	.001	.001	.190	.6672	.6672	.6672
visibility * condition * group	1	.002	.002	.282	.6004	.6004	.6004
visibility * condition * age	1	.005	.005	.714	.4069	.4069	.4069
visibility * condition * group	1	2.465E-4	2.465E-4	.032	.8587	.8587	.8587
visibility * condition * Subjec	23	.175	.008				
RTs * visibility * condition	1	.001	.001	.187	.6690	.6690	.6690
RTs * visibility * condition *	1	1.039E-4	1.039E-4	.034	.8564	.8564	.8564
RTs * visibility * condition *	1	.013	.013	4.227	.0513	.0513	.0513
RTs * visibility * condition *	1	.003	.003	1.069	.3119	.3119	.3119
RTs * visibility * condition *	23	.071	.003				

Dependent: Rts

Table of Epsilon Factors for df Adjustment Dependent: Rts

	G-G Epsilon	H-F Epsilon
RTs	1.000	1.136
visibility	1.000	1.136
condition	1.000	1.136
RTs * visibility	1.000	1.136
RTs * condition	1.000	1.136
visibility * condition	1.000	1.136
RTs * visibility * condition	1.000	1.136

Correct	reaction	time	analysis	only
---------	----------	------	----------	------

Source	df	Sum of Squares	Mean Square	F-Value	P-Value	G-G	H-F
group	1	.023	.023	2.789	.1085		
age	1	.005	.005	.669	.4219		
group * age	1	.004	.004	.536	.4714		
Subject(Group)	23	.189	.008				
visibility	1	.025	.025	15.926	.0006	.0006	.0006
visibility * group	1	.002	.002	1.396	.2495	.2495	.2495
visibility * age	1	4.839E-5	4.839E-5	.031	.8618	.8618	.8618
visibility * group * age	1	1.502E-6	1.502E-6	.001	.9755	.9755	.9755
visibility * Subject(Gro	23	.036	.002				
condition	1	.001	.001	.802	.3798	.3798	.3798
condition * group	1	.005	.005	3.798	.0636	.0636	.0636
condition * age	1	.002	.002	1.915	.1797	.1797	.1797
condition * group * age	1	.007	.007	6.121	.0212	.0212	.0212
condition * Subject(Gro	23	.028	.001				
visibility * condition	1	9.625E-5	9.625E-5	.098	.7572	.7572	.7572
visibility * condition *	1	.002	.002	1.624	.2152	.2152	.2152
visibility * condition *	1	.001	.001	.848	.3667	.3667	.3667
visibility * condition *	1	.001	.001	.892	.3547	.3547	.3547
visibility * condition *	23	.023	.001				

Dependent: correct Rts

Table of Epsilon Factors for df Adjustment Dependent: correct Rts

	G-G Epsilon	H-F Epsilon
visibility	1.000	1.136
condition	1.000	1.136
visibility * condition	1.000	1.136

False Alarm Reaction Times

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value	G-G	H-F
group	1	.013	.013	.666	.4228		r
age	1	.005	.005	.238	.6300		
group * age	1	.004	.004	.187	.6695		
Subject(Group)	23	.436	.019				
visibility	1	.013	.013	1.482	.2358	.2358	.2358
visibility * group	1	.007	.007	.800	.3804	.3804	.3804
visibility * age	1	.023	.023	2.592	.1211	.1211	.1211
visibility * group * age	1	.001	.001	.075	.7872	.7872	.7872
visibility * Subject(Gro	23	.207	.009				
condition	1	.679	.679	78.592	.0001	.0001	.0001
condition * group	1	1.075E-4	1.075E-4	.012	.9122	.9122	.9122
condition * age	1	.003	.003	.391	.5377	.5377	.5377
condition * group * age	1	.008	.008	.916	.3484	.3484	.3484
condition * Subject(Gro	23	.199	.009				
visibility * condition	1	.002	.002	.198	.6602	.6602	.6602
visibility * condition *	1	.001	.001	.067	.7979	.7979	.7979
visibility * condition *	1	.018	.018	1.821	.1903	.1903	.1903
visibility * condition *	1	.003	.003	.276	.6042	.6042	.6042
visibility * condition *	23	.224	.010				

Dependent: FAs Rts

Table of Epsilon Factors for df Adjustment Dependent: FAs Rts

	G-G Epsilon	H-F Epsilon
visibility	1.000	1.136
condition	1.000	1.136
visibility * condition	1.000	1.136

Appendix 5.1.4: Post-hoc analyses

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value	G-G	H-F
group	1	.008	.008	7.551	.0115		
age	1	4.511E-4	4.511E-4	.442	.5129		
group * age	1	6.023E-5	6.023E-5	.059	.8103		
Subject(Group)	23	.023	.001				
focus 1 FAs	2	.027	.014	37.264	.0001	.0001	.0001
focus 1 FAs * group	2	4.277E-4	2.139E-4	.588	.5593	.5128	.5406
focus 1 FAs * age	2	2.215E-4	1.108E-4	.305	.7388	.6746	.7133
focus 1 FAs * group * age	2	.001	.001	1.715	.1912	.1996	.1951
focus 1 FAs * Subject(G	46	.017	3.634E-4				

Dependent: Compact Variable 1Foc1%FAs

Table of Epsilon Factors for df Adjustment Dependent: Compact Variable 1Foc1%FAs

	G-G Epsilon	H-F Epsilon
focus 1 FAs	.745	.889

Means Table Effect: focus 1 FAs * group Dependent: Compact Variable 1Foc1%FAs

	Count	Mean	Std. Dev.	Std. Error
square, Dyslexic	14	.030	.023	.006
square, Control	13	.011	.009	.003
diamond, Dyslexic	14	.073	.036	.010
diamond, Control	13	.047	.025	.007
circle, Dyslexic	14	.029	.022	.006
circle, Control	13	.014	.017	.005

Source	df	Sum of Squares	Mean Square	F-Value	P-Value	G-G	H-F
group	1	.017	.017	18.703	.0003		
age	1	2.249E-4	2.249E-4	.247	.6239		
group * age	1	7.147E-5	7.147E-5	.079	.7818		
Subject(Group)	23	.021	.001				
shift FAs	3	.005	.002	5.007	.0034	.0079	.0040
shift FAs * group	3	.002	.001	1.817	.1522	.1681	.1552
shift FAs * age	3	.001	1.783E-4	.554	.6474	.5999	.6391
shift FAs * group * age	3	4.192E-4	1.397E-4	.434	.7294	.6753	.7201
shift FAs * Subject(Gro	69	.022	3.220E-4				

Dependent: %shiftFAs-normal v

Table of Epsilon Factors for df Adjustment Dependent: %shiftFAs-normal v

	G-G Epsilon	H-F Epsilon
shift FAs	.756	.953

Means Table Effect: shift FAs * group Dependent: %shiftFAs-normal v

square, Dyslexic square, Control diamond, Dyslexic diamond, Control circle, Dyslexic circle, Control triangle, Dyslexic triangle, Control

	Count	Mean	Std. Dev.	Std. Error
Γ	14	.025	.012	.003
Γ	13	.013	.011	.003
	14	.048	.027	.007
	13	.018	.013	.004
	14	.056	.027	.007
Γ	13	.020	.023	.006
Γ	14	.045	.029	.008
C	13	.022	.016	.004

df	Sum of Squares	Mean Square	F-Value	P-Value	G-G	H-F
1	.004	.004	5.030	.0348		
1	.001	.001	1.764	.1971		
1	.001	.001	1.381	.2519		
23	.017	.001				
2	.035	.018	29.995	.0001	.0001	.0001
2	.004	.002	3.696	.0325	.0347	.0325
2	2.019E-4	1.009E-4	.172	.8425	.8329	.8425
2	.001	4.618E-4	.787	.4612	.4561	.4612
46	.027	.001				
	1 1 23 2 2 2 2 2 2	1 .004 1 .001 1 .001 23 .017 2 .035 2 .004 2 2.019E-4 2 .001	1 .004 .004 1 .001 .001 1 .001 .001 23 .017 .001 2 .035 .018 2 .004 .002 2 2.019E-4 1.009E-4 2 .001 4.618E-4	1 .004 .004 5.030 1 .001 .001 1.764 1 .001 .001 1.381 23 .017 .001 . 2 .035 .018 29.995 2 .004 .002 3.696 2 2.019E-4 1.009E-4 .172 2 .001 4.618E-4 .787	1 .004 .004 5.030 .0348 1 .001 .001 1.764 .1971 1 .001 .001 1.381 .2519 23 .017 .001 . . 2 .035 .018 29.995 .0001 2 .004 .002 3.696 .0325 2 2.019E-4 1.009E-4 .172 .8425 2 .001 4.618E-4 .787 .4612	1 .004 .004 5.030 .0348 1 .001 .001 1.764 .1971 1 .001 .001 1.381 .2519 23 .017 .001 .001 .001 2 .035 .018 29.995 .0001 .001 2 .004 .002 3.696 .0325 .0347 2 2.019E-4 1.009E-4 .172 .8425 .8329 2 .001 4.618E-4 .787 .4612 .4561

Dependent: %FAs-degfoc

Table of Epsilon Factors for df Adjustment Dependent: %FAs-degfoc

	G-G Epsilon	H-F Epsilon
focus 2 FAs	.955	1.175

NOTE: Probabilities are not corrected for values of epsilon greater than 1.

Means Table Effect: focus 2 FAs * group Dependent: %FAs-degfoc

	Count	Mean	Std. Dev.	Std. Error
triangle, Dyslexic	14	.032	.015	.004
triangle, Control	13	.027	.014	.004
diamond, Dyslexic	14	.094	.035	.009
diamond, Control	13	.057	.026	.007
circle, Dyslexic	14	.034	.023	.006
circle, Control	13	.033	.032	.009

Source	df	Sum of Squares	Mean Square	F-Value	P-Value	G-G	H-F
group	1	.005	.005	9.458	.0054		
age	1	.001	.001	1.583	.2209		
group * age	1	6.219E-6	6.219E-6	.012	.9148		
Subject(Group)	23	.012	.001				
shift FAs	3	.003	.001	4.941	.0036	.0048	.0036
shift FAs * group	3	.001	3.027E-4	1.321	.2748	.2759	.2748
shift FAs * age	3	.001	3.865E-4	1.686	.1780	.1827	.1780
shift FAs * group * age	3	.001	1.732E-4	.756	.5228	.5126	.5228
shift FAs * Subject(Gro	69	.016	2.292E-4				

Dependent: %shiftdegFAs

Table of Epsilon Factors for df Adjustment Dependent: %shiftdegFAs

	G-G Epsilon	H-F Epsilon
shift FAs	.917	1.189

NOTE: Probabilities are not corrected for values of epsilon greater than 1.

Means Table Effect: shift FAs * group Dependent: %shiftdegFAs

	Count	Mean	Std. Dev.	Std. Error
square, Dyslexic	14	.034	.026	.007
square, Control	13	.022	.010	.003
diamond, Dyslexic	14	.029	.015	.004
diamond, Control	13	.022	.011	.003
circle, Dyslexic	14	.038	.019	.005
circle, Control	13	.026	.015	.004
triangle, Dyslexic	14	.052	.020	.005
triangle, Control	13	.029	.017	.005

Appendix 5.1.5: Probability analysis Probability of a hit

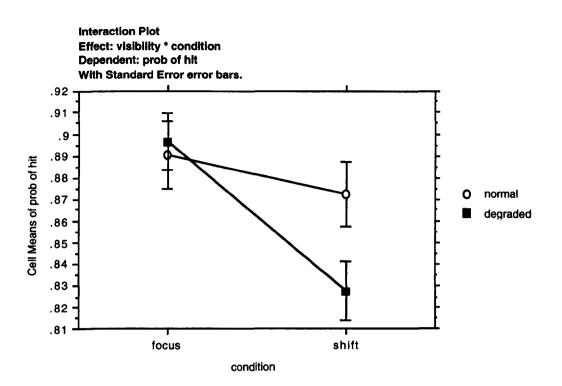
Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value	G-G	H-F
group	1	.049	.049	3.726	.0660	-	
age	1	.087	.087	6.580	.0173		
group * age	1	3.020E-6	3.020E-6	2.292E-4	.9881		
Subject(Group)	23	.303	.013				
visibility	1	.009	.009	4.251	.0507	.0507	.0507
visibility * group	1	.003	.003	1.359	.2556	.2556	.2556
visibility * age	1	.002	.002	.775	.3877	.3877	.3877
visibility * group * age	1	2.811E-4	2.811E-4	.135	.7164	.7164	.7164
visibility * Subject(Gro	23	.048	.002				
condition	1	.049	.049	33.905	.0001	.0001	.0001
condition * group	1	2.397E-5	2.397E-5	.017	.8988	.8988	.8988
condition * age	1	1.638E-4	1.638E-4	.113	.7397	.7397	.7397
condition * group * age	1	.004	.004	2.966	.0984	.0984	.0984
condition * Subject(Gro	23	.033	.001				
visibility * condition	1	.012	.012	6.189	.0205	.0205	.0205
visibility * condition *	1	.003	.003	1.380	.2521	.2521	.2521
visibility * condition *	1	.007	.007	3.404	.0779	.0779	.0779
visibility * condition *	1	.003	.003	1.633	.2141	.2141	.2141
visibility * condition *	23	.045	.002				

Dependent: prob of hit

Table of Epsilon Factors for df Adjustment Dependent: prob of hit

	G-G Epsilon	H-F Epsilon
visibility	1.000	1.136
condition	1.000	1.136
visibility * condition	1.000	1.136



Source	df	Sum of Squares	Mean Square	F-Value	P-Value	G-G	H-F
group	1	.011	.011	14.889	.0008		
age	1	.001	.001	1.314	.2635		
group * age	1	5.824E-5	5.824E-5	.082	.7770		
Subject(Group)	23	.016	.001				
visibility	1	.001	.001	4.226	.0513	.0513	.0513
visibility * group	1	.001	.001	5.541	.0275	.0275	.0275
visibility * age	1	9.250E-5	9.250E-5	.651	.4279	.4279	.4279
visibility * group * age	1	6.078E-5	6.078E-5	.428	.5194	.5194	.5194
visibility * Subject(Gro	23	.003	1.420E-4				
condition	1	3.413E-8	3.413E-8	2.369E-4	.9879	.9879	.9879
condition * group	1	4.910E-4	4.910E-4	3.408	.0778	.0778	.0778
condition * age	1	1.973E-5	1.973E-5	.137	.7148	.7148	.7148
condition * group * age	1	2.322E-4	2.322E-4	1.611	.2170	.2170	.2170
condition * Subject(Gro	23	.003	1.441E-4				
visibility * condition	1	7.878E-5	7.878E-5	.609	.4431	.4431	.4431
visibility * condition *	1	2.264E-5	2.264E-5	.175	.6796	.6796	.6796
visibility * condition *	1	1.889E-10	1.889E-10	1.461E-6	.9990	.9990	.9990
visibility * condition *	1	1.370E-6	1.370E-6	.011	.9189	.9189	.9189
visibility * condition *	23	.003	1.294E-4				

Dependent: prob of FA

Table of Epsilon Factors for df Adjustment Dependent: prob of FA

	G-G Epsilon	H-F Epsilon
visibility	1.000	1.136
condition	1.000	1.136
visibility * condition	1.000	1.136

Appendices: Chapter 6

<u>Appendix 6.2.1.</u> Raw <u>data for</u> main analyses

	R.		-			befor	e RTs	after	r RTs		FAR	T	CR F	Rts	1010 -	DT CD
,	group	FAs	FARTS	corre	correct	FA	correct	FÅ	correct	age group	before	after	before	after	adhid s	RT SD
ry	Category	Int	Real	integ	Real	Real	Real	Real	Real	Category	Real	Real	Real	Real	Real	Real F
	User En	Use	Use	User	User E	User Enter	User Entered	User Ent	User Ente	User Ent	User Ente	User En	User Ente	User Ent	User E	User Ent L
3]	Nominal	Con	Con	Conti	Contin	Continuous	Continuous	Continuous	Continuous	Nomina1	Continuous	Continu	Continuous	Continuo	Contin	Continuous (
	•	•	Fre	•	Free F	Free Form	Free Forma	Free For	Free For	•	Free Form	Free Fo	Free For	Free For	Free F	Free For F
	•	•	0	۲	0	6	6	3	3	•	3	3	3	3	0	3 3
۲	Dyslexic	17	216	195	245	215.068627	269.812500	215.005	258.719	young	215.069	215.005	•	•	2	104.980
۲	Dyslexic	13	315	200	373	288.282051	443.750000	365.487	435.924	young	288.282	365.487	•	٠	6	143.865
•	Dyslexic	15	288	198	359	323.688889	365.375000	351.511	373.700	young	323.689	351.511	365.375	373.700	0	92.829
nr	Dyslexic	14	321	196	330	325.244048	319.742424	329.179	336.523	young	325.244	329.179	319.742	336.523	0	143.665
ер	Dyslexic	16	313	153	359	361.755208	377.527778	350.516	358.769	young	361.755	350.516	377.528	358.769	0	120.230
ke	Dyslexic	18	261	195	293	277.500000	291.678571	281.861	269.488	young	277.500	281.861	291.679	269.488	0	120.130
eu	Dyslexic	16	210	182	274	254.140625	270.333333	290.661	248.111	young	254.141	290.661	270.333	248.111	0	150.910
•	Dyslexic	9	302	200		376.296000	381.703125	391.583	375.510	young	376.296	391.583	381.703	375.510	0	116.400
id	Dyslexic	18	223	195	247	247.185185	234.500000	266.144	238.679	old	247.185	266.144	234.500	238.679	0	67.430
·	Dyslexic	11	283	200	313	298.916667	319.660714	327.682	309.232	old	298.917	327.682	319.661	309.232	0	58.150
sj	Dyslexic	12	332	197	375	370.868056	399.000000	397.639	377.250	old	370.868	397.639	399.000	377.250	0	98.140
m	Dyslexic	14	282	198		318.011905	346.250000	361.494	299.167	old	318.012	361.494	346.250	299.167	1	96.630
an	Dyslexic	17	303	198	328	305.828431	326.031250	333.417	315.125	old	305.828	333.417	326.031	315.125	0	88.669
rd	Dyslexic	10	345	198	406	369.891667	424.650000	418.808	392.550	old	369.892	418.808	424.650	392.550	1	133.610
l	Control	5	307	187	443	472.466667	468.325000	425.000	446.346	young	472.467	425.000	468.325	446.346	0	154.840
ch	Control	11	327	199	334	307.909091	341.250000	310.962	320.196	young	307.909	310.962	341.250	320.196	0	98.540
хy	Control	15	275	200	311	301.088889	310.825000	312.978	300.950	young	301.089	312.978	310.825	300.950	0	66.580
١W	Control	5	377	199	587	449.033333	607.300000	474.683	594.963	young	449.033	474.683	607.300	594.963	0	121.300
an	Control	11	344	197	339	316.598485	363.410714	351.667	361.625	young	316.598	351.667	363.411	361.625	0	85.272
ra	Control	12	290	199		299.479167	318.846154	329.521	288.385	young	299.479	329.521	318.846	288.385	0	78.641
en	Control	4	267	200	395	324.625000	416.261905	351.188	390.944	young	324.625	351.188	416.262	390.944	0	102.022
ah	Control	16	280	189	378	320.937500	489.388889	316.495	486.528	young	320.938	316.495	489.389	486.528	1	187.305
ЪŶ	Control	14	210	195	256	230.880952	259.909091	221.851	261.288	young	230.881	221.851	259.909	261.288	0	74.824
na	Control	8	306	200	327	299.166667	335.500000	322.719	333.059	old	299.167	322.719	335.500	333.059	0	49.050
ac	Control	13	231	195	272	268.532051	270.229167	260.750	269.326	old	268.532	260.750	270.229	269.326	0	69.608
٠	Control	10	244	200	281	247.350000	287.216667	244.175	244.467	old	247.350	244.175	287.217	244.467	0	96.000
<u>y</u>	Control	6	280	199	300	272.291667	297.118421	276.986	313.487	old	272.292	276.986	297.118	313.487	0	78.314
id	Control	18	207	196	228	209.634259	260.750000	228.347	226.976	old	209.634	228.347	260.750	226.976	0	98.850
	1975 B.	1.1		100			1		2	1.00 m 2.		C	1999 - Star (1997)	a and a state	and the state of the	- 73 C

321

a) Number of FAs made

Type III Sums of Squares

df	Sum of Squares	Mean Square	F-Value	P-Value
1	83.237	83.237	5.476	.0279
1	.288	.288	.019	.8917
1	5.081	5.081	.334	.5686
24	364.833	15.201		
	1 1 1 1	1 83.237 1 .288 1 5.081	1 83.237 83.237 1 .288 .288 1 5.081 5.081	1 83.237 83.237 5.476 1 .288 .288 .019 1 5.081 5.081 .334

Dependent: FAs

b)FA reaction times

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value
group	1	822.002	822.002	.407	.5294
age group	1	1262.556	1262.556	.625	.4368
group * age group	1	6044.510	6044.510	2.994	.0964
Residual	24	48450.489	2018.770		

Dependent: FARTS

c) Number of correct hits made

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value
group	1	71.598	71.598	.855	.3643
age group	1	155.469	155.469	1.857	.1856
group * age group	1	57.804	57.804	.691	.4142
Residual	24	2009.097	83.712		

Dependent: correct hits

d) RT of correct responses

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value
group	1	54.189	54.189	.012	.9153
age group	1	11506.552	11506.552	2.455	.1303
group * age group	1	16387.903	16387.903	3.496	.0738
Residual	24	112505.011	4687.709		

Dependent: correct RTs

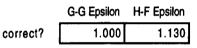
e) RTs before target digit

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value	G-G	H-F
group	1	336.497	336.497	.040	.8436		
age group	1	22917.539	22917.539	2.710	.1127		
group * age group	1	33514.988	33514.988	3.964	.0580		
Subject(Group)	24	202933.410	8455.559				
correct?	1	19324.217	19324.217	16.904	.0004	.0004	.0004
correct? * group	1	832.575	832.575	.728	.4019	.4019	.4019
correct? * age group	1	1652.396	1652.396	1.445	.2410	.2410	.2410
correct? * group * age	1	228.822	228.822	.200	.6586	.6586	.6586
correct? * Subject(Gro	24	27436.779	1143.199				

Dependent: before RTs

Table of Epsilon Factors for df Adjustment Dependent: before RTs



NOTE: Probabilities are not corrected for values of epsilon greater than 1.

f) RTs after target digit

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value	G-G	H-F
group	1	2563.227	2563.227	.291	.5944		
age group	1	22427.478	22427.478	2.548	.1235		
group * age group	1	33854.335	33854.335	3.846	.0616		
Subject(Group)	24	211256.644	8802.360				
correct?	1	837.495	837.495	.873	.3596	.3596	.3596
correct? * group	1	3979.830	3979.830	4.146	.0529	.0529	.0529
correct? * age group	1	3809.310	3809.310	3.969	.0579	.0579	.0579
correct? * group * age	1	86.219	86.219	.090	.7670	.7670	.7670
correct? * Subject(Gro	24	23037.031	959.876				

Dependent: after RTs

Table of Epsilon Factors for df Adjustment Dependent: after RTs

	G-G Epsilon	H-F Epsilon
correct?	1.000	1.130

g) RTs before and after a FA

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value	G-G	H-F
group	1	6473.208	6473.208	.965	.3357		
age group	1	9877.798	9877.798	1.473	.2367		
group * age group	1	32620.867	32620.867	4.864	.0372		
Subject(Group)	24	160955.200	6706.467				
time	1	3697.796	3697.796	14.403	.0009	.0009	.0009
time * group	1	1106.091	1106.091	4.308	.0488	.0488	.0488
time * age group	1	128.793	128.793	.502	.4856	.4856	.4856
time * group * age group	1	160.489	160.489	.625	.4369	.4369	.4369
time * Subject(Group)	24	6161.660	256.736				

Dependent: FA RT

Table of Epsilon Factors for df Adjustment Dependent: FA RT

G-G Epsilon H-F Epsilon

time 1.000 1.130

NOTE: Probabilities are not corrected for values of epsilon greater than 1.

h) RTs after a correctly withheld response

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value	G-G	H-F
group	1	420.568	420.568	.035	.8527		
age group	1	34493.383	34493.383	2.895	.1029		
group * age group	1	35996.259	35996.259	3.022	.0961		
Subject(Group)	22	262088.676	11913.122				
time	1	2226.383	2226.383	15.024	.0008	.0008	.0008
time * group	1	.233	.233	.002	.9687	.9687	.9687
time * age group	1	97.231	97.231	.656	.4266	.4266	.4266
time * group * age group	1	139.635	139.635	.942	.3422	.3422	.3422
time * Subject(Group)	22	3260.201	148.191				

Dependent: CR Rts

Table of Epsilon Factors for df Adjustment Dependent: CR Rts

G-G Epsilon H-F Epsilon time 1.000 1.143

i) Standard deviation of reaction times

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value
group	1	1347.265	1347.265	1.532	.2278
age group	1	6589.977	6589.977	7.493	.0115
group * age group	1	31.387	31.387	.036	.8518
Residual	24	21108.278	879.512		

Dependent: RT SD

j) ANCOVA

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value
group	1	88.608	88.608	10.851	.0032
age group	1	20.829	20.829	2.551	.1239
group * age group	1	6.960	6.960	.852	.3655
correct RTs	1	177.013	177.013	21.676	.0001
Residual	23	187.821	8.166		

Dependent: FAs

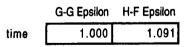
k) dyslexic group only: RTs either side of a FA

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value	G-G	H-F
age group	1	3408.739	3408.739	.619	.4468		
Subject(Group)	12	66118.159	5509.847				
time	1	4571.821	4571.821	17.843	.0012	.0012	.0012
time * age group	1	298.024	298.024	1.163	.3020	.3020	.3020
time * Subject(Group)	12	3074.730	256.227				

Dependent: FA RT

Table of Epsilon Factors for df Adjustment Dependent: FA RT



1) control group only: RTs either side of a FA

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value	G-G	H-F
age group	1	37974.889	37974.889	4.805	.0488		
Subject(Group)	12	94837.040	7903.087				
time	1	367.683	367.683	1.429	.2550	.2550	.2550
time * age group	1	.844	.844	.003	. 9 553	.9553	.9553
time * Subject(Group)	12	3086.930	257.244				

Dependent: FA RT

Table of Epsilon Factors for df Adjustment Dependent: FA RT

G-G Epsilon H-F Epsilon time 1.000 1.091

NOTE: Probabilities are not corrected for values of epsilon greater than 1.

Appendix 6.2.2 Dear Liz and Jackie

Thanks for the paper - a rollicking good read. Sorry if you sent it a while ago - I have just returned from a culurally stimulating stay in Las Vegas.

I enlosse a draft of the latest series of studies with normals which is currently under review at neuropsychologia. This may help a little with the thorny question of response inhibition per se vs sustained attention to action. Broadly these may be terminological differences rather than substantive but what we have shown is that the capacity to withold a response is (unsurprisingly) determined by the task context. That is that the capacity to withhold a response to targets increases with target probabilty. Because group differences apparent on the SART disappear under

these conditions, it suggests that sustaining a readiness to withhold maintaining attentive control over actions over longer periods is the primary cause of poor SART performance and terms like 'response inhibition' as putative unitary capacity may have little meaning outside of the contraints of a particular task. This is also relevent to the question of speed-accuracy trade-off as this manipulation also slows people down. For me the whole question of 'speed-accuracy' trade-off is a tricky one. Clearly there is a strong relationship within (but not between) groups between speed and capacity to withhold a response. The term 'trade off' suggests that there is some strategic, attentionally demanding titration going on - which is certainly in keeping with the instructions to the task. But are they always 'hypothesis testing' (How fast can I go?) or does the speeding result from a lapse in attention to their own responding? Clearly people do reach a stage where they are initiating responses before they have fully processed the relevance of the digit for their actions. We have some data awaiting analysis which will hopefully clarify this issue somewhat.

Anyway - enough of the ramble. I would be grateful for any comments on the ms.

Tom

Attachment converted: Liz's HD:the_absent_mind_(new1).doc (WDBN/MSWD) (000027ED) Tom Manly

Appendix 7.1

Results from 18 participants who took part in all 3 attention expts for focus an shift normal visibility conditions and SART FAs.

i) correlations controls only: n=10 therefore critical values (p<0.05 = 0.6, p<0.01=0.73) Correlation Matrix for Variables: X₁ ... X₇

	focus_1	focus 2	switch	focus no	<u>switch n.</u>	<u> SART F</u>	A group	
focus 1	1							
focus 2	.589	1						
switch	.747	.76	1					
focus nor	.8	.478	.757	1				
switch no	.598	.692	.702	.241	1			
SART FA	379	882	616	275	576	1		
group	•	•	•	•	•	•	1	

dyslexics only:

```
n= 8, p<0.05=0.66, p<0.01=0.8
```

Correlation Matrix for Variables: X1 ... X7

	focus 1	focus 2	switch	focus no	. switch n.	SART F	A group
focus 1	1						
focus 2	.529	1					
switch	.568	.664	1				
focus nor	057	.476	142	1			
switch no	.194	.402	.291	.578	1		
SART FA	096	558	117	647	036	1	
group	•	•	•	•	•	•	1

both:

Correlation Matrix for Variables: X1 ... X7

	focus 1	focus 2	switch	focus no	. switch n	<u>. SART FA</u>	group
focus 1	1						
focus 2	.549	1					
switch	.715	.652	1				
focus nor	.608	.378	.551	1			
switch no	.585	.441	.62	.474	1		
SART FA	344	54	462	413	5	1	
group	.527	.277	.396	.47	.545	264	1

) Raw dat				.h 		
focus 1	focus 2	switch	focus normal	switch normal	SART FA	group
4.823	5.063	3.239	3.040	3.429	17.000	dyslexi
1.480	4.088	1.386	2.826	2.502	13.000	dyslexi
5.042	5.635	5.017	2.880	2.756	11.000	dyslexi
5.861	5.244	3.081	2.580	2.183	12.000	dyslexi
2.945	4.602	3.434	2.428	2.224	16.000	dyslexi
4.297	6.458	5.178	3.501	3.204	9.000	dyslexi
3.475	6.024	2.174	3.711	2.706	10.000	dyslexi
4.365	5.990	5.082	2.351	2.612	15.000	dyslexi
4.983	5.757	3.533	3.156	3.485	11.000	contra
5.877	5.607	4.560	3.921	3.782	13.000	contro
5.435	5.729	4.251	3.570	3.126	11.000	contro
5.384	5.292	3.147	3.501	2.400	18.000	contro
3.721	5.507	4.420	2.870	3.080	12.000	contro
6.600	5.931	6.524	6.046	3.502	10.000	contro
4.708	5.843	4.200	2.830	3.240	5.000	contro
6.593	5.904	6.077	3.563	5.127	8.000	contro
6.992	5.862	6.032	5.773	3.870	6.000	contro
4.316	5.450	3.556	3.162	2.982	16.000	contro

ii) Raw data

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