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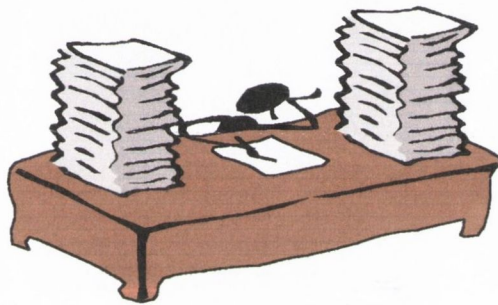
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The Role of Persistent Primitive Reflexes in Learning Difficulties

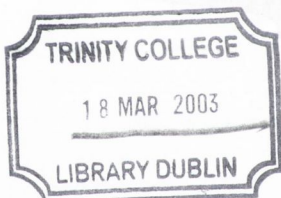


An evaluation of a new causal theory of dyslexia

By
Maria McEvoy

Submitted for the fulfilment for the requirement of the award of Masters of Science by Research, Trinity College Dublin, 2002.

The Role of Perseus
Primitive Reflexes in
Learning Difficulties



An evaluation of a new theory of dyslexia
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Submitted for the Fellowship for the Department of the Faculty of
Science in Research Trinity College Dublin 2003

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Abstract

In a recent study in Belfast by McPhillips, Hepper and Mulhern (2000) the persistence of an early motor reflex, the asymmetric tonic neck reflex (ATNR) was linked to dyslexia. A 100% prevalence of the ATNR was reported in a sample of children with specific reading difficulties. A movement programme involving repetition of reflex movements was reported to have significantly reduced the persistent primitive reflex. This, in turn, led to an improvement of performance in reading. If the persistent ATNR causes both a maturational lag and is reliably associated with dyslexia, then it may constitute both a neurological cause and indicate a definite treatment. However, the McPhillip's study did not use a control group who did not have reading difficulties when testing for the prevalence of the ATNR and so it may be that the ATNR is present in most or all of the population and not just a sub-population of dyslexic children as the work of other research has shown (e.g., Parmenter, 1975).

This research attempted to assess both the normal and dyslexic population for prevalence of the ATNR. A second aim of the study was to assess the prevalence of one other motor reflex, the tonic labyrinthine reflex (TLR) because of its link in the literature with reading achievement. A third aim of the study was to investigate the finding by Parmenter (1975) that the ATNR manifested more powerfully when the child's head was turned to the right, in contrast with the left. A fourth aim of the study was to contrast the ATNR measurement method used by McPhillips, the Schilder test, with another method, the Ayres II test, which was used by Parmenter, to determine the reliability of the measurement procedure. Finally, the relationship between the TLR and ATNR was analysed in order to test the theory that the TLR predates the ATNR and must be integrated fully, before the ATNR can be integrated.

The Ayres test for the ATNR was the only reflex measure that significantly discriminated between dyslexic and non-dyslexic children. Both of the tests for the ATNR found a difference between sides to which the head was turned but no interaction effect. The Schilder test was found to be a very unreliable measure for measuring the ATNR when compared to the Ayres II test and both measures were found to have a high

degree of subjectivity making it difficult to provide conclusive evidence regarding the validity of the hypothesis that persistent reflexes are related to developmental dyslexia.

Future research must attempt to standardise reflex measurement methods so that studies can be reliably compared and evaluated. Finally, the ATNR and TLR were not significantly correlated which questions the theory of the evolution of postural reflexes. The theory of persistent reflexes as a causal explanation for dyslexia is not supported by this research. Future research might consider the role of the cerebellum in learning difficulties as this structure has a role in reflex control and tonus and so persistent reflexes may be only a part of a larger cerebellar system dysfunction rather than the primary cause of dyslexia.

Index

	<u>Page</u>
Declaration	-
Acknowledgements	-
Abstract	-
Index	-
1 1. Introduction	1
1.1 General Introduction: A Definition of Dyslexia	1
1.2 The Importance and Difficulty of Diagnosis	3
1.3 A Diagnosis of Dyslexia	4
1.4 The Symptoms of Dyslexia	5
1.4a Difficulties of Reading, Writing and Arithmetic	5
1.4b Spatial and Temporal Difficulties	7
1.4c Memory Problems	7
1.4d Difficulties of Balance and Co-ordination	9
1.4e Neurological Differences	9
1.4f Emotional Problems	11
1.5 The Gender Difference in Dyslexia	12
1.6 Causal Theories of Dyslexia	15
1.6a Socio-Cultural Theories of Dyslexia	16
1.6b Genetic Theories of Dyslexia	18
1.6c Theory of Magnocellular Deficit	19
1.6d Theory of Phonological deficit	24
1.6e Theory of Automatisatation Deficit & Cerebellar Dysfunction	26
1.7 Theory of Persistent Primary Reflexes	33
1.7a Definition of Postural and Primitive Reflexes	33

1.7b	The Evolution of Postural Reflexes	35
1.7c	Function of Primitive and Postural Reflexes	38
1.7d	The Embryology of Behaviour	40
1.7e	The Role of Persistent Reflexes in Maturational Lag	42
1.7f	The Asymmetric Tonic Neck Reflex and Learning Disabilities	43
1.7g	The Asymmetric Tonic Neck Reflex in the Normal Population	46
1.7h	Other Reflexes as Predictors for Developmental Lag	48
1.8	The Aim of the Present Study	51
2.	Method	52
2.1	Overall Design of the Study	52
2.2	Participants	52
2.3	Materials	53
2.3a	The Wechsler Intelligence Scale for Children (WISC-R)	53
2.3b	The Wechsler Objective Reading Dimensions (WORD)	54
2.3c	The Schilder Test for ATNR	55
2.3d	The Ayers II Test for ATNR	56
2.3e	A Test for the Tonic Labyrinthine Reflex	57
2.4	Procedure	57
2.4a	Before the Testing Session: Preparation	57
2.4b	During the Testing Session: Administering the tests	58
2.4c	After the testing session: Collating and analyzing the data	58
3.	Results	60
3.1	Inter-rater reliability for scoring reflexes	60
3.2	Identification of specific reading, spelling or comprehension difficulties	64
3.3	Diagnosis of dyslexics based on specific sub-test difficulties	65

3.4	Do higher reflex scores significantly correlate with a diagnosis of dyslexia?	67
3.5	Do any of the reflex tests show significant differences between the groups and is there a difference between the sides to which the head is turned?	69
3.6	Do the Schilder and Ayers tests for the ATNR correlate?	73
3.7	Do any of the dyslexic children not score at all on measures of persistent reflexes?	73
3.8	Is there a correlation between the presence of the TLR and the ATNR?	76
4.	Discussion	77
4.1	An analysis of scoring inter-rater reliability for scoring reflexes	77
4.2	Do higher reflex scores significantly correlate with a diagnosis of dyslexia?	79
4.3	Do any of the tests show significant differences between the groups on reflex scores and is there a difference between the sides to which the head is turned?	80
4.4	A comparison of tests for the ATNR	82
4.5	Do any of the children not score at all on measures of persistent reflexes?	82
4.6	Is there a correlation between the presence of the TLR and ATNR?	83
4.7	Implications of the findings of this study for the theory of persistent reflexes as a primary cause of dyslexia	83
4.8	Suggestions for future research	84
4.8a	The standardization of methodology	84
4.8b	The use of modern technology	85
4.8c	Scientific evaluation of the evolution of reflexes	87
4.8d	Scientific comparisons with other causal theories of dyslexia	87
4.8e	Could there be a role for the theory of persistent reflexes within a framework of cerebellar dysfunction theory?	88
5.	Conclusion	91
6.	References	92

Graphs

Figure 1: Nicolson and Fawcett’s Model of Cerebellar Abnormality	30
Figure 2: The Persistence of Primary and Postural Reflexes	34
Figure 3: The Evolution of Postural Reflexes to Voluntary Movement	37
Figure 4: Inter-rater Observation Graph: Schilder Right	61
Figure 5: Inter-rater Observation Graph: Schilder Left	61
Figure 6: Inter-rater Observation Graph: Ayers Right	62
Figure 7: Inter-rater Observation Graph: Ayers Left	62
Figure 8: Inter-rater Observation Graph: TLR Up	63
Figure 9: Inter-rater Observation Graph: TLR Down	63
Figure 10: The Percentage of Specific Difficulties with the Word Sub-tests	65
Figure 11: Average Scores of Dyslexics and Non-Dyslexics on the Schilder Tests with a Comparison of Sides	70
Figure 12: Average Scores of Dyslexics and Non-Dyslexics on the Ayers Tests with a Comparison of Sides	71
Figure 13: Average Scores of Dyslexics and Non-Dyslexics on the TLR Tests with a Comparison of Direction	72

Tables

Table 1: Inter-rater Reliability between Observers for Reflex Scores	60
Table 2: Categories of Difficulties Experienced by Children	64
Table 3: The Specific Sub-Test Difficulties of the Dyslexic and Non-Dyslexic Children	66
Table 4: Statistical Relationship Between Diagnosis and Scoring on Reflex Tests: The Pearson Correlation Coefficient	67
Table 5: Statistical Relationship Between Diagnosis and Scoring on Reflex Tests: The Biserial Correlation Between Diagnosis and Reflex Scores.	68
Table 6: A Comparison of Dyslexic and Non-Dyslexic Reflex Scores	69
Table 7: A Comparison of Zero Scores on Reflex Tests According to Diagnosis	73
Table 8: A Comparison of Low Scores (0,1) on Reflex Tests According to Diagnosis	75
Table 9: The Relationship Between the Presence of the ATNR and TLR	76

Appendices

Appendix A: Letter of Permission to Schools

Appendix B: Letter of Permission to Parents Regarding Educational Assessment

Appendix C: Letter of Permission to Parents Regarding Reflex Tests

Appendix D: Criteria and Scoring Sheets for Recording Reflexes

1. Introduction

1.1 General Introduction: A Definition of Dyslexia.

Dyslexia has been defined in many different ways but few definitions have come close to encompassing all of the facets of this disorder. Dyslexia has been called many different names throughout its history. At the beginning of the last century, Hinshelwood called the disorder “word blindness” and Orton called it “strephosymbolia,” literally symbol twisting rather than blindness, stating that it was a cognitive rather than a visual deficit in nature (Fawcett, 1994). Because the effects of dyslexia primarily manifest in reading, most definitions have focused on this aspect. For example, the British Dyslexia Association (1989) defined dyslexia as a specific difficulty in learning, which was constitutional in origin in one or more of reading, writing and spelling tasks. The most frequently quoted definition is the Federation of Neurology (1968) definition of what they called “specific developmental dyslexia.” This is defined as a disorder manifested by difficulty in learning to read despite conventional instruction, adequate intelligence and socio-cultural opportunity. However, this definition has been criticised heavily for its failure to define what is meant by “conventional” instruction, “adequate” intelligence and socio-cultural “opportunity.” This definition does not address the wide variety of symptoms with which dyslexics present and treats dyslexics as a homogenous group with difficulties in learning to read only. There is no reason why children of below average intelligence, from poor homes, who may have missed school through illness, who have been inadequately taught, speak English as a second language and have emotional problems could not also be dyslexic (Turner, 1997). This definition, in addition, means that dyslexia cannot be diagnosed until after beginning to learn to read (Beech, 1994) and this can be detrimental to the child who, at this stage, may already have experienced failure and frustration.

A discrepancy definition of dyslexia is more favourable than a definition based on exclusionary criteria, that is a comparison of the degree to which dyslexic individuals differ from the normal population rather than the complete absence or presence of particular traits (Snowling, 1996). Turner (1997) has stated that with a shift in thinking towards a developmental framework, it is possible to move from a discrepancy definition

to an explanatory one. A good example of this shift in thinking is illustrated by the following definition:

“ Learning disabilities is a generic term that refers to a heterogeneous group of disorders manifested by significant difficulties in the acquisition and use of listening, speaking, reading, writing, reasoning and mathematical abilities. These disorders are intrinsic to the individual and presumed to be due to central nervous system dysfunction. Even though a learning disorder may occur concomitantly with other handicapping conditions (e.g. sensory impairment, mental retardation or social and emotional disturbances) or environmental influences (e.g. cultural differences, insufficient or inappropriate instruction or psychogenetic factors) it is not the direct result of these conditions or influences.”

(Duane, 1985, pp.10)

This definition recognises, first of all, that a learning disability, such as dyslexia, manifests in each individual in a unique pattern and may be influenced, but not caused by, an interaction of impairments such as sensory or socio-emotional and environmental influences such as culture and teaching methods. The only reservation about this definition is the use of the term (specific) learning difficulties rather than dyslexia. In a survey, ninety percent of parents said they would use the term “dyslexia” to describe their children’s learning deficit (Riddell, Brown and Duffield, 1990) whereas the term “specific learning disability” meant nothing but a vague construct to them. The causal factor, as stated in this definition, is a central nervous system dysfunction and the parents interviewed also stated unequivocally that their children did have distinct constitutional problems that could not be merged within the general continuum of learning disabilities. It is perhaps, the wide-ranging symptoms of dyslexia beyond difficulties in reading and writing that sets it apart from other learning difficulties and a definition of dyslexia must encompass the range of problems and an explanatory model must identify a causal factor or factors that account for all of them. The above definition (Duane, 1985) appears to be the most useful definition because it does recognise that dyslexia is more than just a

problem with learning and that a theory of the causes of dyslexia must take the underlying neurology into account.

1.2 The Importance and Difficulty of Diagnosis

Schiffman and Clemmens (1966) have estimated that if a potential learning disability in children was diagnosed in the second grade, eighty-two percent of them could be brought back to grade level in a two year period by appropriate teaching methods but that if a child was not identified until after grade four, only fifteen percent of them could be successfully helped and after grade nine only six percent would benefit from intervention (cited in Goldberg and Schiffman, 1972). Satz (1978) conducted a six year follow up of students identified as being dyslexic and found that only 6.1% of students classified as having severe reading retardation in grade two had improved by the end of grade five. Thirty percent of the average readers in grade three had become problem readers in grade five and virtually no improvement was seen in the performance of problem readers between grade two and five (cited in Duane, 1985). By middle childhood, the brain reaches ninety five percent of its adult size and so the earlier the intervention, the more the plasticity of the brain to learn (Scarborough, 1990).

As dyslexic children grow older, they tend to compensate for their learning disability making it even more difficult to identify. Most of the symptoms of dyslexia occur most frequently in younger age groups. Dyslexic students often learn to compensate and some manage to do very well academically thus masking obvious difficulties in later life. For example, a case study of a university student R. E. who was dyslexic found that she associated the appearance of new words with their meanings and pronunciations on a whole-word basis instead of phonetically deciphering them (Campbell and Butterworth, 1985, cited in Ellis, 1993). This condition is called developmental phonological dyslexia. Other dyslexics, described as having developmental surface dyslexia, try to read and spell phonologically using sub-lexical letter-sound correspondences, e.g. writing 'shoo' for 'shoe' (Patterson, Marshall and Colheart, 1985). Ellis (1993) has suggested that these strategies form a phonological-surface continuum with dyslexics using these strategies to a greater or lesser extent to compensate for their difficulties, producing an individual pattern of variation in each dyslexic person. However, dyslexia is a lifelong disability and makes

life more difficult for those who have to expend their resources using compensatory strategies to overcome their difficulties. The Institute for Neurophysiological Psychology in Chester has shown that dyslexics tend not to grow out of their reading and spelling problems and they frequently grow further from the normal group with age (Goddard-Blythe and Hyland, 1988, cited in Blythe, 2000). Crichley (1981) has listed an early diagnosis as one of the best predictors of a positive outcome along with a high I.Q., a sympathetic, informed and encouraging attitude of parents, the availability of skilled, sympathetic and intensive tuition and high determination of the dyslexic. The early diagnosis of dyslexia is, therefore, of paramount importance.

1.3 A Diagnosis of Dyslexia

Of course, not all children with reading difficulties have dyslexia. A diagnosis of dyslexia is usually reserved for those for whom reading and scholastic achievement is well below their mental age. Dyslexic students tend to read two to three grades behind their peers, but can score a little higher on arithmetic (Goldberg and Schiffman, 1972). Difficulties in learning to read have been called “litmus tests” for dyslexia (Fawcett and Nicolson, 1994). Reading difficulties are generally taken to be two standard deviations below their predicted reading age based on multiple regression studies (MacDonnell, 1998). Children with dyslexia, according to a Yale study in 1973, have a mean I.Q. of 102, whereas children who are backwards readers tend to have an I.Q. of 80. The Federation of Neurology (1968) defined dyslexic children as children with an I.Q. score of over 90 but who read at a level that is two years below their actual age. Therefore, dyslexia is a reading disability that is not to be expected when I.Q. is considered. A lack of response to teaching methods and persistence over time also distinguishes dyslexia from reading retardation (Frith, 1981, *ibid*). Also, the typical I.Q. profile of a developmental dyslexic shows a marked discrepancy between non-verbal (performance) and verbal I.Q. There is some evidence when using the WISC-R that suppressed scores on the so-called A.C.I.D. profile (subtests of arithmetic, coding, information and digit span) are associated with dyslexia and are thought to illustrate the interaction of phonological difficulties and reduced speed of processing (Fawcett and Nicolson, 1994). Nicolson and Fawcett (1992) found that dyslexic children showed impairment on at least one in four of

the A.C.I.D. sub-tests. However, recent research has shown that the A.C.I.D. profile as a pattern occurs in only about 7% of dyslexic children (Smith, 2001).

The correlation between I.Q. and reading is around 0.6, so learning disabled children do not necessarily read very badly (Thompson, 1984). This is why the entire repertoire of difficulties displayed by dyslexic children should be taken into account when diagnosing dyslexia and not just the discrepancies between IQ and reading level. Many symptoms of dyslexia, which will be discussed in the next section, need to be considered; difficulties in reading, writing and arithmetic, spatial and temporal problems, memory problems, problems of balance and co-ordination, neurological anomalies and socio-emotional problems. Intelligence testing is, however, the first “port of call” for a diagnosis of dyslexia and is *“a summary measure of performance on all tests of ability; an anchor for a regression matrix which includes co-normed measures of attainment in basic skills; reassurance for individuals and family that failure in literacy does not have to imply mental retardation and the broadest possible guide to setting expectations in teaching”* (Turner, 1997, pp. 39).

1.4 The Symptoms of Dyslexia

Several categories of problems exist in a unique pattern in each dyslexic person; as mentioned above, they may include difficulties in reading, writing and arithmetic, spatial and temporal problems, memory problems, problems of balance and co-ordination, neurological anomalies and socio-emotional problems. This huge variability makes dyslexia challenging to identify and diagnose. Each category of difficulty may be thought of as a dimensional trait with each individual dyslexic manifesting each category of difficulty to a greater or lesser extent, or not at all.

1.4a Difficulties in Reading and Writing and Arithmetic

Miles (1983) found that although many dyslexics learn to read all have a history of difficulties when learning to read, remain slow readers, have problems reading aloud and identify shapes rather than symbols when looking at letters. Using the Bangor Dyslexia Test, Miles listed some of the difficulties in reading and writing experienced by dyslexics. These included a limited ability to process symbolic language and bizarre spelling which

indicates a lack of phoneme-grapheme correspondence. Dyslexics also show subtle language impairments at age two manifested primarily by a slower vocabulary and syntactic development (Frith, 1997). There is quite often a tendency not to perceive similarities or differences between words, an inability to keep place when reading or a tendency to read without comprehension (Goldberg and Schiffman, 1972). Dyslexic students can also be insensitive to rhyme and alliteration and find it difficult to categorise words on the basis of common sounds, even in comparison with younger children who read no better than they do (Bradley and Bryant, 1983, cited in Snowling and Nation, 1997). Other deficits include pluralization of singular nouns or the omission of the plural suffix, usually 's', the abbreviation of a lengthy word (such as "talk" for "talking"), guessing words that are similar to the actual word to be read (e.g., "offer" for "official"), narremic substitution, which refers to the supplying of semantically similar words (e.g., "coat" for "jacket"), where the narreme is the unit of meaning, the inability to see misspellings in a text, unfamiliar words misread or not attempted and the inability to extract the theme of a text (Crichley, 1981). Crichley (1981) also listed the omission of short words such as articles, conjunctions and prepositions. Davis (1995) calls these "trigger words" which are words that cannot be pictured and so cause disorientation. Davis states that one compensatory strategy dyslexics use is picturing words as non-verbal representations, but words such as "the" or "and" have no pictorial representation and so cause a failure of the comprehension strategy that relies on the non-verbal route. Indeed, one of the compensatory strategies relied on by a dyslexic child is counting on fingers when doing mathematical tasks (Miles and Ellis, 1981).

Of course, confusion about one's mother tongue makes learning a second language almost impossible. Interestingly, dyslexic children and normal age-matched controls do equivalently well when asked to copy the script of a foreign language that is not familiar to either group (Vellutino, 1973). To a dyslexic, their own language appears to them as unfamiliar. Rack and Snowling (1985) suggest that dyslexics cannot access verbal or semantic information as easily as controls and so take a much longer time to name objects or to identify letters as semantic symbols. It has been estimated that dyslexics are seventy-eight percent slower at retrieving words and that speed of word retrieval is correlated with reading speed (Wolf, 1980, cited in Rudel, 1985).

Arithmetic is also affected by dyslexia with confusion over subtraction and addition. Numbers also tend to be transposed which, of course, has negative implications for subsequent calculations (Naidoo, 1988). Recall of digits, digit span, and reciting mathematical tables are also impaired (Miles, 1983).

1.4b Spatial and Temporal Problems

Dyslexic children may experience a range of spatial problems (Miles, 1983). Dyslexics confuse right and left, east and west, north and south. They also have problems distinguishing between 'b' and 'd' when reading and writing. Quite often, a tendency to read from right to left instead of from left to right leads to a perception of complete mirror images of letters so that letters seen in a mirror can be read more easily than the right way around (Goldberg and Schiffman, 1972). There is also a tendency to reverse whole words, for example, to read 'was' instead of 'saw'. Spatial thought is also very disorganised (Crichley, 1970).

Dyslexics also have temporal problems including problems over times and dates and days of the week, months of the year and telling the time (Seymour, 1986). They also have a lack of rhythm (Crichley, 1970). Benton and Pearl (1978) singled out rapid alternating finger movements as the sole factor distinguishing dyslexics and non-dyslexics with dyslexics very poor at keeping rhythm (cited in Denckla, 1985). Blank and Bridger (1966) also found that dyslexics were worse than non-dyslexics on tasks requiring temporal-spatial but not auditory-visual integration and that even tasks of merely perceiving and remembering temporal patterns was worse (cited in Bakker and Schroots 1981).

1.4c Memory Problems

Dyslexia can be associated with deficits in short-term, long-term and working memory. Impairment in verbal short-term memory has been correlated with dyslexia (Jorm, 1983, cited in MacDonnell, 1998) and verbal long-term memory tasks can also be impaired, for example there is a reduced primacy effect when learning lists and this has been linked to a failure to use phonological coding when storing information in long-term

memories (Rack, 1994). Auditory and visual short-term memory is also impaired (Snowling, 1985).

Dyslexia has also been associated with working memory limitations. Phonological decoding of words involves identifying the sound that the letters make, holding each sound in memory in a sequential manner until all the sounds have been identified and storing them in such a way that they can be blended together to form a word. Such a capability depends on the capacity of working memory (Adams, 1990; Baddeley, 1990; cited in Palmer, 2000). The structure of working memory is hypothesised to consist of three main components; a modality free central executive of limited capacity which represents attention, an articulatory or phonological loop which temporarily holds information in speech form and a visio-spatial sketch pad which is specialized for spatial or visual encoding. Through rehearsal, information can be kept in the temporary store and transferred to a more durable semantically based memory system (Baddeley and Hitch, 1974). If working memory capacity is insufficient, the ability to decode words phonetically becomes impaired. Palmer (2000) found that dyslexic groups have lower levels of central executive functioning than controls. The difficulty lay in the dual coding strategy used exclusively by the dyslexic group. The dyslexic group were delayed in the ability to inhibit the visual encoding of words in order to allocate resources to the phonological encoding of words and so were phonetically impaired. The inability to switch retrieval strategies is a central executive dysfunction. There is also an indication that phonetically similar letters cause more problems than dissimilar letters. A study by Baddeley (1966) compared performance on rhyming letters, e.g., BCGPT and non-rhyming letters, e.g., HKSLQ and found that performance was worse on the phonologically confusing letters, supporting the theory that information is held in phonological form, as working memory theory suggests. Furthermore, dyslexics show the same phonetic confusability effect when compared with younger normal readers matched on memory span (Johnston, Rigg and Scott, 1987) or reading level (Holligan and Johnston, 1988) and so dyslexics are less efficient at using phonological codes in short term memory (cited in Rack, 1994).

1.4d Difficulties of Balance and Co-ordination

Vellutino (1979) identified what he called neurological soft signs in dyslexics including abnormal reflexes and motor co-ordination patterns. Dyslexia has been positively correlated with a range of difficulties in balance and co-ordination including clumsiness, difficulties in clapping, swimming and bicycle riding, as well as walking backwards, balancing and catching balls (Augur, 1985; Haslum 1989, cited in MacDonnell, 1998). Furthermore, Nicolson and Fawcett (1994) found that dyslexic children have difficulty with fine motor movements such as threading beads, hand-eye co-ordination and balancing on one foot, especially if blindfolded, i.e., in the absence of continuous visual feedback. There are also early predictors of later problems with balance and co-ordination. Other symptoms include difficulties in fine motor co-ordination, for example, poor copying ability (Benton, 1978; Owen, 1971, cited in Rudel, 1985). In infancy, dyslexic children often display problems with crawling (Snowling, 1985). Denkla (1985) postulated a 'motor analyser,' that is dependent on the left hemisphere and is important for timed, sequential and detailed movement. It is deficient in the first decade of life in dyslexic children, indicating a maturational lag.

Crichley (1970) found that an infant destined to become dyslexic skip the crawling stage altogether and that lack of dominance in dexterity is an indicator of immaturity of cerebral development. Lack of cerebral dominance often manifests as ambidexterity in the individual and dyslexic children often display a lack of handedness (Crichley, 1970). Orton (1937) also noted a high frequency of mixed eye, hand and foot dominance in children with dyslexia. Newton and Thomson (1978) found a lack of cerebral dominance in eighty-eight percent of the dyslexics tested (cited in Newton, 1988).

1.4e Neurological Differences

Studies have shown anatomical differences between the brains of dyslexics and non-dyslexics. MRI studies show that the planum temporale cortical language area, normally larger in the left hemisphere, is symmetrical in children with specific reading disability (Stein and Walsh, 1997). The planum temporale is a triangular area situated on the superior temporal sulcus which coincides with part of Wernicke's area in the left hand side of the brain involved in phonological processing (Shapleske, Russell, Woodruff and

David, 1999). Autopsies have revealed that neurons of the medial geniculate nucleus are smaller in the left hemisphere of dyslexics. Post-mortem analyses of human dyslexic brains show the presence of focal neocortical malformations in cerebrocortical microgyria which have a role in discriminating between rapidly presented auditory stimuli (Felton, Naylor and Wood, 1990) and also the presence of focal cerebral dysplasias in the brain of developmental dyslexics which predominantly affect perisylvian regions in the left cerebral hemisphere of the cortex known to participate in the neurolinguistic system (Hynd and Semrud-Clikeman, 1989). Leonard and colleagues found that four anatomical abnormalities predicted short term and long term phonological processing memory ability; a marked rightward cerebral asymmetry, marked leftward asymmetry of the anterior lobe of the cerebellum, combined leftward asymmetry of the planum and posterior ascending ramus of the Sylvian fissure, and a large duplication of Heschl's gyrus on the left (Leonard, Eckert, Lombardino, Oakland, Kranzler, Mohr, King and Freeman, 2001).

fMRI and EEG studies have revealed differences of cerebral activations in dyslexic brains. A study by Georgiewa, Rzanny, Hopf, Knab, Glauche, Kaiser, and Blanz (1999) revealed significant differences in the patterns of activation in Broca's area and the left inferior temporal region for both non-word reading and in a phonological transformation task, indicating an alteration of the phonological system in dyslexic children. Pihko, Leppanen, Eklund, Cheour, Guttorm, and Lyytinen (1999) used ERP measurements to measure automatic change detection in the brain of Finnish infants with and without a genetic risk for dyslexia using syllables varying in vowel duration. This study found that infants at risk for dyslexia processed differences between syllables in the right hemisphere of the brain whereas controls showed the opposite pattern. Vellutino (1979) positively correlated deviant EEG patterns with dyslexia and concluded that dyslexia was a neurological disorder. Paulesin, Frith, Snowling, Gallagher, Morton, Frackowiak and Frith (1996) asked dyslexic adults to perform rhyme judgement and memory tasks and noted the brain activation. Dyslexics, in contrast to controls, did not display the usual concerted activation of four brain areas; Broca's area, Wernicke's area and the insula and inferior parietal lobule, all situated in the left hemisphere around the perisylvian fissure, suggesting that dyslexia results from a disconnection between language systems. Eden, Van Meter, Rumsey, Maisog, Woods and Zeffiro (1996) showed that the disconnection

between language systems was also correlated with dysfunctions in other parts of the dyslexic brain, for example areas of the brain involved in visual motion (cited in Hulme and Snowling, 1997).

1.4f Emotional problems

Crichley stated some socio-emotional repercussions of dyslexia, which may exacerbate the problem. A diagnosis of dyslexia has been correlated with low self-esteem. Boys, if they react to their dyslexia with aggression, can become involved in delinquency and crime. In a survey of remand homes in Inner London in 1968, Crichley found that sixty percent of inmates were more than two years behind in reading skills and some, he believed, were certainly cases of developmental dyslexia (cited in Crichley, 1970). It must also be stated that dyslexia may not be such a handicap if the appropriate school facilities are in place to help the child (Riddick, 1996). It is easier to teach a dyslexic child below the age of nine as this child may not yet have been defeated by failure and learning is still fun. This reinforces the importance of early diagnosis for intervention and for the self-concept of the child. It is also worth noting that rather than being seen as a detrimental label, a diagnosis of dyslexia is often received with relief by parents and children alike as a frame of common reference between clinicians, policy makers and parents (Riddell, Brown and Duffield, 1994). It is not the label of dyslexia per se that causes the self-esteem problems but the experience of failure that causes low self esteem and leads to an expectation to fail in a self-fulfilling process (Riddick, 1996).

Attention deficits have also been correlated with dyslexia. In a U.S. study, approximately 75% of attention deficit hyperactivity disorder (ADHD) boys were underachieving and about 33% were performing academically at least one grade below their expected grade level (Cantwell, 1986, cited in Kendall and Hammen, 1995). Learning disabled children may find it difficult to pay attention to important environmental stimuli and so miss what they are being taught in school, leading to this association between attention deficit disorder on the one hand and learning deficits on the other.

1.5 The Gender Difference in Dyslexia

There is a notable sex difference in the prevalence of dyslexia. Boys, more than girls, tend to develop dyslexia with a ratio of between 3:1 and 10:1, depending on the criteria of the sample (Goldberg and Schiffman, 1972). A Yale study in 1973 found that 76% of dyslexics and 54% of backwards readers were male. The result of a 1970 cohort study by Haslum (1989) postulated a link between motor difficulty and dyslexia but that the link was stronger for males than females. Other links that were stronger for males of this study included failure to walk backwards, low educational qualifications of the mother and the father's negative attitude toward the male child. In contrast, low educational qualifications of the father and the addition of siblings with reading problems affected girls of this cohort more. Boys and girls with dyslexia were equally impaired on a ball catching task.

There is a number of possible reasons why boys outnumber girls in the dyslexic population. Research suggests that the areas of the brain responsible for language are organised differently in males and females. Gender differences in the localisation of brain activation during phonological processing have been recorded. Females activate Broca's area bilaterally whereas males show greater left hemisphere dominance. The left hemisphere networks engaged by phonological and lexical semantic processing overlap in females but are spatially distinct in males. Brain activation in reading tasks tend to be more focal and anterior within the left hemisphere in females than males and brain activation during phonetic processing tends to be more bilateral in females than males (Pugh, Shaywitz, Shaywitz, Constable, Skudlarski, Fulbright, Brovien, Shankweiler, Katz, and Gore, 1995, cited in Lambe, 1999). fMRI studies have shown a difference in activation patterns with males displaying a lateralised activation pattern to the left inferior frontal gyrus regions and females a more diffuse neural system involving both left and right inferior gyri, suggesting sex differences at the level of phonological processing (Shaywitz, Shaywitz, Pugh, Constable, Skudlarski, Fulbright, Bronen, Fletcher, Shankweiler and Katz, 1995). Post-mortem studies have shown that language structures are more focally and anteriorly based within the left hemisphere of females (Kimmura, 1984). Also, morphological variations in the dyslexic brain have been found, most

notably a decrease in the grey matter in the left temporal lobe and in the temporo-parieto-occipital junction bilaterally and in the frontal lobe, caudate, thalamus and cerebellum. Such variations may contribute to deficits associated with dyslexia (Brown, Eliez, Rumsey, White and Reiss, 2001).

The Geschwind hypothesis, formulated in the 1980s, proposes a link between left-handedness, language disorders and autoimmune function. The link is abnormal testosterone secretion during development which is higher in males in any case but can also affect females in higher than normal quantities, leading to a difference in brain structure (cited in Kelley, 1993). The more slowly developing hemisphere, e.g., right hemisphere in left handers, is exposed to higher than normal levels of testosterone and inhibits the growth of the dominant hemisphere leading to a more symmetrical brain structure. This leads to a disruption of the normal left to right hemisphere balance during a learning task, yielding an inferior learning performance. There is also a shift from left to right hemisphere control of language and handedness, eventually leading to permanent learning disabilities in some cases, especially for boys (Hugdahl, 1993). Furthermore, allergies and migraine are more common in dyslexics and their families, linking left-handedness, language disorders and autoimmune function (Geschwind and Behan, 1982, cited in Rosen, Sherman and Galaburda, 1993). Geschwind also proposed that girls might be less affected by certain environmental factors such as quality of teaching, social class differences or outside pressures (Geschwind, 1981, cited in deFries, Gillis and Wodsworth, 1993).

The Geschwind hypothesis has been criticised for inconsistent results. Significant differences between groups on the occurrence of left handedness has not always been found and only fifty-three percent of children in one study reported one or more immune diseases compared to twenty-five percent of controls, which is not significant (Hugdahl, Synnevog and Sutz, 1990, cited in Hugdahl, 1993). Hormones other than testicular androgens, e.g., ovarian hormones, have been shown to be involved in cortical development (Tallal and Fitch, 1993). Witelson (1991) suggests that males and females are categorically different with respect to cerebral development and do not develop along the same growth continuum, which may explain the gender difference between them with regards to dyslexia. These cerebral differences between males and females may be due to

the fact that the individual areas of the brain in males do not mature as quickly as females (Goldberg and Schiffman, 1972). Females have greater maturity by the age of six and tend to be more motivated in a learning situation.

Males have a greater frequency of cerebral trauma accompanying cerebral haemorrhages and are at greater risk for emotional problems caused by these factors. The reason why males do not recover from cerebral traumas as well as females could be due to the fact that females rely more on bilateral representations for both verbal and visual-spatial abilities (Ray, Georgiou and Ravizza, 1979). Thus, females may have a compensatory capacity in the opposite hemisphere that males do not (cited in Tallal and Fitch, 1993). Also, there can be greater genetic difference between monozygotic and dizygotic female twins than male twins indicating that monozygotic females are less at risk than monozygotic males for developing dyslexia (DeFries, Gillis and Wadsworth, 1993). Thus, a difference in maturation rates, caused by genetic differences would appear to be the most likely cause of the gender difference in dyslexia.

However, the genetic make-up of a person cannot be considered in isolation. The environment in which a person is reared must also be considered. Another reason for the reported sex difference may be due, not to the difference in dyslexia between the sexes per se, but to different rates of identification of dyslexia in female and male children. A longitudinal study investigating the difference between research-identified and school-identified children with dyslexia by Shaywitz, Shaywitz, Fletcher and Escalor (1991) found no significant differences in the prevalence of reading disability in research-identified boys and girls in either the second or third grade. In Connecticut, school identification led to a classification of 13.6% of boys in second grade and 10% of boys in third grade as being at risk for dyslexia. In contrast, only 3.2% of girls in second grade and 4.2% of girls in third grade were identified as being at risk for dyslexia. They concluded that school-identified samples are subject to referral bias. In *The Psychology of Sex Differences* Jacklin and Maccoby (1974) reviewed the literature on the psychology of sex differences in order to establish valid sex differences. They pointed out that if a generalisation about a group of people is believed, whenever a member of a group behaves in the expected way it is noted and the belief is strengthened. However, observed but unexpected differences are dismissed as a myth. They called for studies to be grounded in

context. Ratings should not be stereotyped so that, for example, activity levels are recorded as 'appropriate' activity levels for a boy and 'appropriate' levels for a girl. These different measurements make it hard to compare results. For example, it has been hypothesised that sex-role training is more stringent for boys than for girls and parents may place more pressure on boys for academic achievement. It would therefore be more noticeable that boys were falling behind. Boys have the bulk of attention and instruction from teachers and girls are, consequently, neglected (Richardson, Koller and Katz, 1986). At elementary school, boys receive more formal instruction than girls despite being in the same classroom (Brophy and Good, 1973; cited in Beal, 1994). One reason why this might be the case is that boys tend not to be as successful in school as girls of the same age, finding it harder to adapt to the demands of the classroom and feeling a conflict between behaviours that lead to academic success, like being quiet and attentive, and those encouraged by the male peer group such as being loud and boisterous (Brophy and Good, *ibid.*). Thus, there is a two-way relationship, with boys demanding more attention and teachers giving it (Irvine, 1986; cited in Beal, 1994). Teachers give boys more attention also because of boys' lower average achievement. Girls get higher grades on average (A.A.U.W., 1992; cited in Beal, 1994) and in classrooms of 30 students teachers get better students to work quietly and with minimal instruction and give the bulk of their attention to those who lag behind. This creates a situation which may lead to a learning disability being noticed in boys more easily simply because they get noticed more but not necessarily because it presents in a higher proportion in the male population. These biased measurements often make it difficult to get a true picture of the gender difference in the occurrence of learning disabilities.

1.6 Causal Theories of Dyslexia.

There are several causal theories of the symptoms of dyslexia. These might be labelled socio-cultural theory, genetic theory, magnocellular deficit theory, phonological deficit theory, automaticity and cerebellar dysfunction theory and primitive reflex theory. Each of these theories will be discussed in turn.

1.6a Socio-cultural theory

Some authors, e.g. Beard (1993), state that the causal factor for dyslexia is an environmental one. Phonological deficit is caused by a lack of reading experience in early life. Beard argues that early reading and being read to gives the experience needed to develop phonological skills. Thus, a phonological deficit is caused by a lack of reading experience in early life. A longitudinal study provided support for the hypothesis that the awareness of rhyme and alliteration that children acquire before they go to school is correlated with the child's reading ability when they begin school. The home environment, therefore, may have a powerful influence on learning to read and spell in school (Bradley and Bryant, 1983). Dyslexic children who live in secure homes with a wide speaking vocabulary and are read to and allowed to read the books that interest them tend to persevere at reading more than other dyslexic children. But this is not necessarily a sufficient environment in which to overcome their learning difficulties (Cotterell, 1988).

The context in which a child is reared is seen as vital for learning. If a child is reared in a stressful or dangerous environment this may have a deleterious effect on his/her attention and motivation, thus undermining resources available for learning. For example, it has been reported that children whose parents had divorced were more likely to be behind their schoolmates at reading and mathematics (Robertson, 1999). Hebb (1958) stated that the mode of rearing has a permanent effect on behaviour, especially intellectual attainment, at maturity and significant correlations have been found between emotional disturbance and reading retardation, e.g., Tomkin, 1960 (cited in Myers and Hammill, 1969). Risk factors include poor occupational status of the father and a mother's education level, as mothers are often the ones to read to their children, a task they cannot do if they have never learned to read or if preoccupied by worry about the family's financial status (Snowling and Stackhouse, 1996). Interestingly, a higher incidence of persistent reflexes has been shown in infants raised in social isolation, institutionalised infants and children and adults who are mentally deficient or emotionally disturbed (Levy, 1944; Spitz, 1965; Berkson and Daveyport, 1962, cited in Thelen, 1979). This has been put forth as a causal theory of dyslexia (e.g., McPhillips, 2000). A number of reasons why these reflexes persist have been suggested; for example, in the case of rocking motions, for comfort to recreate rhythmical prenatal experience (Kris, 1954, cited

in Thelen, 1979). There is also a higher incidence of dyslexia among African-American dyslexics than Latino dyslexics in Texan prisons, which may reflect the greater degree of discrimination experienced and poorer education available to the former group (Crichley, 1968).

Culture can also have a huge impact on learning. A cultural study by Schieffelin and Cochran-Smith (1984) studied Kaluli children attending boarding school in Papua, New Guinea. These children remained illiterate because the village they returned to on the weekends did not value reading and writing (cited in Beard, 1993). Culture can also influence the way in which dyslexia manifests and how detrimental it is to the dyslexic person. Dyslexia has been shown to be less of a handicap in Italy or Spain than other European countries because of the consistency of orthography for pronunciation and German dyslexics have been shown to make fewer word or non-word reading errors because German orthography is also consistent with its phonology (Frith, 1997). . The crucial difference between dyslexics and non-dyslexics is a longer time taken and, often, defeat in the face of very long words (Wimmer and Frith, 1994, cited in Hulme and Snowling, 1997). In contrast, one sixth of English words are phonetically inconsistent making it more difficult to make connections between the symbols seen on the page and the sound of the words for pronunciation (Davis, 1995).

While the socio-cultural factor has been proposed to be the single most important factor in prevention and remediation of dyslexia socio-cultural factors are descriptive rather than explanatory, describing the manifestation of deficits but not actually explaining why they manifest, and cannot alone account for the pattern of deficits that manifest in the dyslexic population (Frith, 1997). Blythe (2000) warns against concentrating on psychosocial aspects of dyslexia, which although central to education, can dominate to the detriment of biological factors fundamental to learning. Behavioural problems caused by environmental problems are unlikely to be powerful enough to cause the disruption of basic psycho-neurological symptoms of dyslexia (Myers and Hammill, 1969). Some research groups have suggested that the recognition of dyslexia as a neurodevelopmental disorder has been hindered by the belief that due to the variable and culturally specific manifestations of dyslexia that it cannot be identified as a diagnosable disorder (e.g., Poulesu, Demonet, Fazio, McCronj, Chanoine, Brunswick, Cappa, Cassu, Habib and Frith,

2001). Poulesu et al. (2001) found a cross-cultural neurocognitive basis for dyslexia using PET scans to show the same reduced activity in a region of the left hemisphere of the brain in dyslexic people from Italy, England and France. Differences were found, however, in reading and phonological tasks and they concluded that differences in performance are aided or hampered by the orthography used from one country to another but that the underlying disorder is the same. Thus, Italian dyslexics performed better than the English or French groups because Italian orthography is more consistent. Dyslexia has been shown to be highly heritable and so the next section discusses genetic theories as a casual explanation for dyslexia.

1.6b Genetic Theories of Dyslexia

Vellutino (1979), based on his review of the literature, stated that dyslexia is highly heritable. Regions on chromosomes fifteen, one and six have been implicated in the occurrence of dyslexia (Cordon, Smith, Fulker, Kimberling, Pennington and deFries, 1994, cited in Hulme and Snowling, 1997). There is a fifty percent chance of a boy being born dyslexic if his father is dyslexic and forty percent if his mother is dyslexic with a lower probability for girls (Snowling, 1996). Phonological skills, in particular, have been found to be heritable (Olson, Rock and Forsberg, 1990, cited in MacDonnell, 1998) but other research suggests that it is not phonological ability that is inherited per se, but aspects of language processing (Snowling, 1996). Twin studies have reinforced the notion that phonological processing deficits are the causal factor in dyslexia (Stanovich and Siegel, 1994, cited in Snowling, 1996). Scarborough (1990) studied children in families where at least one parent was dyslexic and found that this population were more likely to develop dyslexia and showed problems from a very early age. From as early as two and a half years of age, these children made more speech errors and had limited syntax and by five years had phonological problems (cited in Snowling and Nation, 1997).

However, it has also been suggested that phonological processing is not inherited genetically but is passed on in the way parents teach their children to read, an environmental cause (DeFries, 1991). Ellis (1993) suggests that because reading is not naturally selected there can be no deficiencies in our genetic make-up or pattern of cognitive skills we are born with that are specific to reading and writing. Reading is an

skill that must be learned on a conscious level. The task of the reader is to recode graphemes into phonemes. To accomplish this, the reader first becomes aware of the phonological structure of spoken words and then recognises that orthography represents phonology. These skills are not instinctive and must be learned. Dyslexic children have a deficit at the phonological level of their language system that impairs their ability to segment language into phonemes thus disrupting their learning of reading.

Miles (1983) has suggested a causal chain from the genetic cause of dyslexia to the pattern of difficulties seen. Because of an underlying genetic deficit, there are deficiencies in the magnocellular pathways of the visual and auditory systems with the result that it is difficult for them to adequately synthesise sequences of sounds if they are presented rapidly, leading to a weakness at the phonological level resulting in problems with reading and maths. The magnocellular deficit will be discussed in the next section followed by the phonological deficit theory of dyslexia.

1.6c Theory of Magnocellular Deficit

It has been proposed that difficulties with reading may simply be the most obvious symptom in a highly literary society of more elementary differences in perception (Grant, Zangaladze, Thiagarajah, and Sathian, 1999). Dyslexic children can display a wide range of sensory deficits including visual, auditory and tactile and these can be identified at two to four weeks of age (Rawon, 1986). Magnocellular dysfunction is thought to play a part in these deficits. The magnocellular system is specialised for processing fast temporal information (Stein and Walsh, 1997). Van Essen, Anderson and Felleman (1992) provide an account of the strategies used for efficient information processing within the hierarchy of sub-divisions in the primate visual system. The two major processing streams that divide from the optic nerve into two fibre bundles are further subdivided into ninety percent parvocellular (P) pathways projecting to the P layers of the lateral geniculate nucleus (LGN) and ten percent magnocellular (M) ganglion cells projecting to the M layers of the LGN (Perry, Ochler and Cowey, 1984). The primary role of the P cells is to encode information about luminance contrast for low and moderate temporal frequencies (1-20 HZ) and the transmission of spectral information, or colour. M cells encode information about luminance contrast for higher temporal frequencies (5 - 20 HZ) and the

presence of fine-grained details without reproducing precise spatial relations. The thickly myelinated axons of the M stream makes it well suited for various tasks requiring motion information (Talcott, Hansen Assoku and Stein, 2000) but this is not the only type of information it conveys. Higher levels of the visual system, therefore, need to access information from both the P and M systems during the performance of many tasks and, correspondingly, there is anatomical crossover between them (Bartels and Zeki, 1998).

Autopsies of the brains of dyslexics have shown diminution of neuronal size in the lateral geniculate nucleus in the magnocellular lAyes of the brain (Livingstone and Risen, 1991, cited in Grant et al, 1999). Galaburda (1985) found that magnocells are twenty percent smaller in dyslexics at post mortem (cited in Stein and Walsh, 1997).

Magnocellular abnormalities can cause problems of visual perception. Lovegrove (1980) found that dyslexics have slightly reduced contrast sensitivity at low spatial frequencies and low luminance levels favoured by the magnocellular system (cited in Stein and Walsh, 1997). Abnormalities in the visual magnocellular lAyes of the LGN has been linked to reduced motion sensitivity, unsteady binocular fixation, and consequential poor visual localization, particularly on the left side (left neglect), which lead to an inability to fixate properly on letters (Stein, 2001). Other evidence for an M pathway deficit in dyslexic brains include reduced or delayed electrophysiological responses to stimuli processed mainly by the M pathway, reduced fMRI responses to stimuli processed mainly by the M pathway and poor motion discrimination (Demb, Boynton, Best and Heeger, 1998). Lesion studies with primates have shown that damage to the magnocellular lAyes of the LGN produce losses in sensitivity, or even complete blindness, to stimuli having both high temporal and low spatial frequencies (e.g., Merigan and Maunsell, cited in Stuart, McAnally and Castles, 2001).

The transient system of the magnocellular system handles the rapid processing of information and the sustained system handles slow-moving information. Both are both mutually inhibitory (Lovegrove, 1994). During reading, the sustained system extracts detailed information during fixations whilst the transient system extracts more general information from the periphery of vision and integrates the information from successive fixations. It is the transient system that is damaged in the dyslexic magnocellular system leading to a persistence of information across fixations that may result in a

superimposition of images and unstable visuomotor control (Stein, 1994). Dyslexics are poorer at identifying letters close to fixation but superior at peripheral identification of letters (Geiger and Lettvin, 1987, cited in Biscaldi, Gezeck and Stuhr, 1998). Eden, Stein, Wood and Wood (1994) found a correlation between poor fixation control and reading problems due to the instability in eye convergence and poor pursuit fixation, which is related to m-pathways. Furthermore, a relationship was found between poor convergence control and a lack of phonological awareness (Biscaldi, Gezeck and Stuhr, 1998). The magnocellular system does not deal exclusively with visual modalities but also has a role in auditory perception. M neurons of the thalamic auditory relay nuclei are smaller in dyslexic than non-dyslexic individuals and dyslexics often have deficits in their central auditory pathways (Galaburda, Menard and Rosen, 1994, cited in Saarelma, Renvall, Jousmaki, Kovala and Hari, 2002).

Leppaenen, Pihko, Eklund, and Lyytinen (1999) indicated an early biological marker related to the auditory modality that may contribute to the development of speech and phonological processing that is deficient in the dyslexic child at birth. The research found that infants born with a high familial risk of dyslexia process auditory stimuli durations differently from control infants at birth and concluded that this early deficit was an early marker related to the development of language problems later in life. Galaburda, Menard and Rosen (1994) found that, like visual magnocells, auditory magnocells in the medial geniculate nucleus are abnormal in dyslexic brains (cited in Stein and Walsh, 1997). Processing non-verbal auditory stimuli presented in rapid succession is impaired in dyslexic people and this deficit correlates positively with phonological impairment (Tallal, 1980, cited in Pihko Leppaenen, Eklund, Cheour, Guttorm, and Lyytinen, 1999). Dyslexics also have problems perceiving brief auditory cues (Tallal and Piercy, 1973, cited in Snowling, 1996).

Dyslexics also differ from normal controls in their tactile perception. In a grating orientation discrimination threshold study, dyslexics had a mean discrimination threshold of twice the normal rate (Grant et al, 1999). The dyslexic group were also significantly worse when using the dominant hand and attempting to discriminate grating ridge width. The authors conclude that, because several ridges and grooves of the grating were in

contact with the participant's fingertips at any one time, defective lateral meshing could underlie these results, suggesting abnormal spatio-temporal masking in dyslexics.

Stein and Walsh (1997) also state that the cerebellum is a major target to type M efferents and so a magnocellular deficit could affect balance and co-ordination. The M stream is also evident in the posterior parietal cortex that is sensitive to direction of movement, gaze, eye movement control, visuo-spatial attention and peripheral vision. The reading process and saccade systems are both controlled by visuo-spatial attention and fixation systems, and these are impaired or develop slowly in dyslexics (Biscaldi, Gezeck and Stuhr, 1998). Dyslexics display very poor saccadic control, make a higher number of saccades and have longer fixation durations when they read text above their ability levels (Hyana and Olson, 1995 cited in Biscaldi, Gezeck and Stuhr, 1998). This would account for many of the reading deficits displayed by dyslexics as reading without eye movement control results in blurring and masking making reading extremely difficult. Thus, Stein and Walsh conclude that in dyslexia magnocellular neuronal cells, that play a major role in all sensory, sensorimotor and motor systems throughout the brain may be selectively damaged during early development causing the wide-ranging symptoms of dyslexia.

Magnocellular theory research has been criticised for various reasons. However, although the magnocellular deficit appears to correlate with difficulties in phonological processing, e.g., Hogben, 1996, there is no clear causal link between them (cited in Hogben, 1997). A magnocellular deficit may be part of a more general deficit in the nervous system and pathological factors impeding development of the magnocellular deficit may also affect the development of language areas of the brain, in which case a magnocellular deficit would have no direct role in reading disability but would merely serve as a marker to another deficit that does have a causal role (Livingstone, Risen, Drislane and Galaburda, 1991, cited in Hulme and Snowling, 1997). For example, the visual problems displayed by dyslexics such as seeing words the wrong way around (e.g., "was" for "saw") is a common error in young children's reading at an early developmental stage, suggesting a developmental lag rather than perceptual problems as the cause of dyslexia (Stuart and Colheart, 1988, cited in Rack, 1994). Furthermore, binocular stability also arises out of a developmental process with 54% of six year olds, 70% of seven year

olds and 85% of nine year olds displaying binocular stability (Stein, Riddell and Fowler, 1986, cited in Stein, 1994).

A second criticism of magnocellular theory is that many M-deficits in the visual system are relatively mild and so the effect on reading would be milder than is seen in dyslexic individuals. Skottun (2000) pointed out that magnocellular neurons respond to coarse low spatial frequency patterns under rapid temporal change. However, reading involves dealing with stationary characters with fine details and so the magnocellular system could not be responsible for a reading deficit. Recently, Saarelma et al. (2002) demonstrated that although dyslexic adults display a normal auditory startle reaction, the facilitation lasts longer leading to a general sluggishness of sensorimotor processing and attention shifting. Thus, even though the M-deficit is mild, it could still cause a significant impairment of reading-related skills due to sluggishness during attention shifting. Stuart, McAnally and Castles (2001) stated that complete inattention or ineffectively focused attention could lead to elevated thresholds in spatial contrast sensitivity which undermines a lot of the magnocellular research data which has relied solely on methods such. This is a valid criticism because, as the authors point out, attention deficit disorder is often comorbid with dyslexia and so dyslexic participants in would have faired badly in these tasks, not because of elevated thresholds in spatial contrast sensitivity but their inability to stay attentive to the task that they were required to do.

A third and final problem with the magnocellular theory as a causal theory of dyslexia is that because of the exclusionary criteria of the World Federation of Neurology definition (Crichley, 1970), a great majority of studies on dyslexia have excluded dyslexics with the very traits magnocellular theories propose as symptoms of dyslexia, resulting in little research evidence to support the theory. Other theories have primarily focused on the linguistic characteristics of dyslexia. The most widely held causal theory of dyslexia, the phonological deficit theory, will be discussed in the following section.

1.6d Theory of Phonological Deficit

The phonological theory of dyslexia states that the causal factor in dyslexia is a cognitive deficit related to the language system, specifically, a deficiency in the processing of linguistic units, or phonemes, that make up all spoken or written words (Shaywitz, 1996). According to the dual route theory of reading, a word may be read as a whole via visual features (lexical or logographic route) or assembled from the series of word segments such as the individual graphemes (the sub-lexical route). The logographic route is typically found in the early stages of reading, whereas the sub-lexical route is dependent on word segmentation and blending using phonological rules (Beech, 1994). Dyslexic children do not have the phonological skills required to learn to read at the critical time (Snowling, 1987). The term 'phonological awareness' is used to refer to awareness of words as sequences of discrete phonemes (Rack, 1994, cited in Lambe, 1999). Children who do well on tests of phonological awareness are quick to understand how phonemes and graphemes relate in orthography and use this knowledge of letter-sound rules as a self-teaching device (Share, 1995, cited in Palmer, 2000). Bruce (1964) used subtraction tasks in order to examine children's phonological awareness, for example, asking children to delete the 's' sound at the start of struck to make a new word. The abilities of children varied according to age. Five-year-old children could not do the task at all, six year olds could manage to get six percent of words, seven and eight year olds could correctly transform twenty-nine and fifty-five percent of words respectively, and at nine years children could decipher eighty nine percent of words (cited in Beard, 1993). However, dyslexic children traditionally do much worse at these tasks and even those who have achieved the same level of reading as younger age groups do worse than them when reading nonsense words, because of their lack of ability to perceive a grapheme-phoneme correspondence (Beard, 1993). Learning to read involves strengthening a system of connections between orthographical and phonographical brain centres (Seidenberg and McClelland, 1989, cited in Snowling and Nation, 1997). Many dyslexic children who have been given extra phonological training intervention, therefore, have benefited in reading (Beard, 1993). Poor performance on short-term memory, long-term memory, picture naming and verbal repetition would all be consistent with a

deficiency in the use of phonologically based information suggesting a causal link from phonological skills to reading development (Rack, 1994).

Despite the improvements made by phonological intervention studies the phonological theory by itself cannot account for the wide-ranging symptoms of dyslexia. The Jyväskylä Longitudinal Study of Dyslexia (JLD) found that the most likely precursor of a diagnosis of dyslexia is measures of phonological development but that the causal link between phonological skills and later phonemic awareness skills is weak (Lyytinen, 1997). However, most of the literature on dyslexia focuses on a phonological deficit in dyslexia because of the manifestation of dyslexia primarily as a reading disorder. If dyslexia is a reading disorder, the problem must be in the stages of processing that are needed only for reading, i.e., a module that performs orthographic to phonological conversion (Klein and McMullen, 1999). Reading involves a lot more than one “linguistic centre” of the brain as phonological theory suggests. Reading involves the cross-modal linking of disparate abilities required for the skills of reading and writing and producing functional units in the brain that behave as if they were modular, i.e., composed of dissociable cognitive subsystems (Ellis and Young, 1998, cited in Turner, 1997). Shaywitz and his colleagues studied disruption in the functional organisation of the dyslexic brain. A functional disruption and overall under-activation was found in the posterior cortex of dyslexics. This region included visual and language areas and the association cortex in the angular gyrus that is involved in carrying out cross-modal integration used in reading (for example between the visual print and the phonological equivalent). This disruption was associated with phonological impairment (Shaywitz, Shaywitz, Fulbright, Constable, Mencl, Shankweiler, Lieberman, Skudlarski, Fletcher, Katz, Marchione, Lacadie, Gatenby and Grove, 1998). The angular gyrus of the left hemisphere has been shown to be dysfunctional in the brains of dyslexics with the right hemisphere posterior regions compensating in the mediation of phonological processing (Pugh, Mencl, Shaywitz, Shaywitz, Fulbright, Constable, Skudlarski, Marchione, Jennes, Fletcher, Lieberman, Shankweiler, Katz, Lacadie and Gore, 2000). fMRI studies have also revealed disrupted neural responses to phonological and orthographic processing in dyslexic children, with only dyslexic children displaying reduced activity in the left temporo-parietal cortex during letter rhyming and letter matching tasks (Temple,

Poldrack, Salidis, Deutsch, Tallal, Merzenich and Gabrieli, 2001). Phonological deficits appear to have a corresponding functional disruption at neural level.

Learning to read and write is one of the most difficult skills a person will ever master and taxes visual, auditory and manual skills (Stein and Walsh, 1997). Many lower level psychological processes contribute to the single act of reading including phonemic awareness, phonological decoding, the ability to process stimuli rapidly and to automatise the process, memory and the ability to recognise words (Grigorenko, 2001). It is no wonder then that a general automatisisation deficit, caused by an underlying neurological problem, should manifest when the child attempts to learn such a complex task. Phonological deficits have been shown to be even greater where there is strong reliance on automatic processing (Yap and Van Der Leij, 1994) supporting the theory that it is a general automatisisation deficit rather than a phonological deficit that is the primary causal factor in developmental dyslexia.

1.6e Theory of Automatisisation Deficit and Cerebellar Dysfunction

The inadequacies of the magnocellular theory and phonological theory of dyslexia led to the development of a new school of thought which sought to integrate all of the theories together to make sense of the disparate evidence from each field of research. The theory of Automaticity, as devised by Nicholson and Fawcett in 1990 has two components, Conscious Compensation (CC) and the Dyslexic Automatisisation Deficit (DAD). The theory of Conscious Compensation states that dyslexic children need to invest a large amount of their attention in order facilitate a task such as to maintain balance and, thus, if a second task interferes with this concentration the child will not be able to perform the primary task, i.e. maintain balance. The theory of Dyslexic Automatisisation Deficit states that the skill of motor balance and other skills such as reading are deficient in dyslexic children because of a general failure to automatise skills. An automatic process is defined as “well learned in long term memory, is demanding of attention only when a target is presented, is parallel in nature, is difficult to alter, to ignore or suppress once it is learned and is virtually unaffected by load” (Schiffrin and Schneider, 1977, cited in Nicolson and Fawcett, 1990, p. 127). Dyslexics display difficulties in Rapid Automate Naming (R.A.N.) tasks when naming both verbal and visual materials

(Denckla and Rudel, 1976). Thus, their inability to access verbal and semantic material may have to do with a failure to automatise this skill. Nicholson and Fawcett suggest that instead of looking at specific skills such as reading, research should concentrate on general skills that manifest in dyslexic children. Phonological deficit, then, instead of being a causal factor in dyslexia was proposed to be part of an underlying deficit that produces problems when trying to learn any skill, including phonological encoding. Thus, theories should focus on an underlying deficit that could produce the varied patterns of difficulties that manifest in dyslexia. However, Miles (1994) has suggested a taxonomy for a theory of learning difficulties suggesting that such a theory should be able to account for anatomical, genetic, magnocellular and auditory deficits as well as the range of difficulties experienced by the learning disabled.

Attempting to do just that Nicholson, Fawcett and Dean (1994) proposed that a dysfunction in the cerebellum might underlie the major deficits displayed by dyslexics and recent research suggests that the role of the cerebellum extends beyond motor control into higher cognitive functions. Dyslexia has long been associated with abnormal balance, motor control and muscle tone, all of which are under the control of the cerebellum. Recent evidence of biochemical and functional abnormalities in the right cerebellum of individuals with dyslexia, suggests cerebellar involvement in the disorder (Lyddy, 2002). Flourens was the first to discover the motor function of the cerebellum. He found that when the cerebellum was ablated to a greater and greater degree, the animal gradually lost the faculty of regular movement and co-ordination (Flourens, 1824, cited in Francher, 1990). However, the cerebellum has been targeted as the most likely site to be implicated in learning for a diverse range of skills, which are non-motoric.

Schmahmann and Sherman (1998) performed neurological examinations and anatomical neuro-imaging studies on twenty patients with diseases confined to the cerebellum and noted the nature and severity of changes in function. Behavioural changes were observed in patients with lesions involving the posterior lobe of the cerebellum and vermis including impairment of executive functions such as planning, verbal fluency, abstract reasoning and working memory and personality changes such as blunting of affect or disinhibited behaviour and language deficits such as aggramatism, dysprosodia and mild anomia. Furthermore, lesions of the anterior lobe of the cerebellum produced only

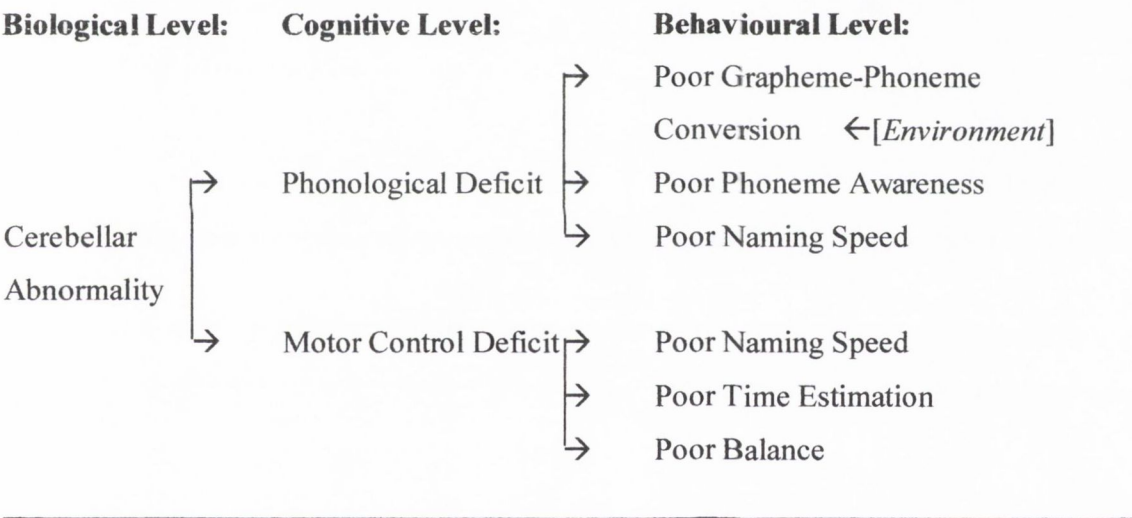
minor changes in executive and visual-spatial functions. The constellation of deficits caused by the disruption of cerebellar modulation of neural circuits that link prefrontal, posterior parietal, superior temporal and limbic cortices with the cerebellum was named Cerebellar Cognitive Affective Syndrome. Other consequences of damage to the cerebellum, cited in their paper include difficulties of concept formation, learning of paired associates, intellectual slowing (Kish, el-Awar, Schut, Leach, Oscar-Berman and Freedman, 1988), poor performance on tests of fluency and perseveration (Appollonio, Grafman, Schwartz, Massoquoi and Hallett, 1993) and impaired error detection (Fiez and Raichle, 1997). Also, children with damage to the cerebellum can display mutism and dysarthria, personality changes, poor initiation of voluntary movements (Pollack, Polinko, Allingent, Towbin, Fitz, 1995) and expressive language and visual spatial dysfunction (Levinsohn, Cronin-Golomb and Schmahmann, 1997).

Thus, these studies show that when the reciprocal connections between the cerebellum and the frontal, temporal and limbic areas are disrupted a myriad of dysfunction in executive function, spatial cognition, personality and linguistic difficulties are observed which lead to an overall lowering of intellectual function (Schmahmann and Sherman, 1998). There are two-way connections linking the cerebellum and Broca's area, which make it possible for the cerebellum to affect language dexterity (Nicolson and Fawcett, 1994). Nicolson and Fawcett (1994) also reported recently that behavioural and neuro-imaging tests had indicated that dyslexia is associated with cerebellar impairment on all the classic clinical tests of cerebellar function including postural stability, muscle tone, coordination and dexterity in about 80% of cases and concluded that cerebellar dysfunction is present from birth (Nicolson, Fawcett and Dean, 2001). Behavioural tests of cerebellar function found that dyslexics, like patients with cerebellar lesions, had a dissociation of time estimation (impaired) and loudness estimation (unimpaired) (Nicolson, Fawcett and Dean, 1996). Mild cerebellar impairment, therefore, provides a possible explanation for the phonological deficits in dyslexia which may arise initially from inefficient articulatory control attributable to cerebellar impairment and also the range of problems cerebellar function, including tests of muscle tone and coordination, with which dyslexic children present (Fawcett, Nicolson, Dean, 1996). Indeed, previous studies with children with cerebral palsy showed that direct stimulation of the cerebellum

affected muscle tonus (Glanda and Zoltan, 1987) and that physical therapy that emphasizes the practice of activities that challenge stability can be used to improve posture in patients with cerebellar dysfunction (Gill-Body, Popat, Parker and Krebs, 1997). Neuro-imaging studies reported by the research team found that a PET study showed that dyslexic participants showed significantly less cerebellar activation in the right hemisphere and vermis (Jenkins et al., 1994, cited in Nicolson, Fawcett and Dean, 2001). Furthermore, the right cerebellum has been shown to display a functional deficit, exhibiting decreased blood flow in response to both learned and novel motor task, indicating that the behavioural signs of cerebellar abnormality reflect underlying abnormalities in cerebellar activation (Nicolson, Fawcett, Berry, Jenkins, Dean and Brooks, 1999). Earlier this year, a study using MRI found that the cerebellar structures in dyslexic brains are more symmetric than in non-dyslexics suggesting alterations in the neurological organisation of the cerebellum and that the degree of cerebellar symmetry was correlated with the severity of dyslexics' phonological decoding deficit (Rae, Harasty, Dzendrowskyj, Talcott, Simpson, Blamire, Dixon, Lee, Thompson, Styles, Richardson and Stein, 2002). Other previous studies have similarly shown symmetry in the language area of the temporal lobe (Stein and Walsh, 1997) and Rae and her colleagues (2002) speculate that increased cerebellar hemispheric symmetry might be expected to reflect an increased cerebral hemispheric symmetry in dyslexics, which would support studies linking dyslexia with mixed dominance (e.g., Crichley, 1970). Rae et al. (2002) state that the right cerebellum is involved in word generation and verbal working memory, specifically in phonological rehearsal, and cerebellar symmetry would lead to deficits in phonological decoding and working memory. Previously, biochemical differences had also been found in the left-temporo-parietal lobe and right cerebellum of dyslexic brains that were not found in controls (Rae, Lee, Dixon, Blamire, Thompson, Styles, Talcott, Richardson and Stein, 1998). The research team found that the ratio of choline-containing compounds to N-acetylaspartate is reduced in the right cerebellar hemisphere of dyslexics, which was linked to magnocellular and cerebellar deficits, which were, in turn, linked to the wide symptoms of developmental dyslexia including visual, auditory, phonological, linguistic and motor deficits.

Not only is the cerebellum a major target for type M efferents, it is the head ganglion of the magnocellular system (Stein, 2001) and so the cerebellar deficit theory integrates well with the magnocellular theory (Stein and Walsh, 1997) and Nicolson et al., 2001, suggest that cerebellar deficit is an alternative, or perhaps parallel mechanism to magnocellular abnormality with dyslexic children displaying one or other or both of these abnormalities at the biological level. Nicolson and Fawcett’s theory of cerebellar deficit is shown in Figure 1 below.

Figure 1: Nicolson and Fawcett’s Model of Cerebellar Abnormality (1995)
(Adapted from Frith, U., 1997)



As can be seen from Figure 1, Nicolson and Fawcett’s Theory of dyslexia attempts to integrate phonological, genetic and magnocellular theory (through the interconnections between the cerebellum and the magnocellular pathway). It also provides room for the influence of culture and the environment on the expression of dyslexia. Furthermore, a genetic basis for the impaired development of magnocells has been found on the short arm of chromosome six which is involved in the production of antibodies which can affect the developing brain, particularly the magnocellular system (Stein, 2001). The cerebellar

hypothesis provides an explanation of many of the major deficits in dyslexia, particularly related to phonological impairment and motor control, and brings together genetic, socio-cultural and magnocellular theories of dyslexia. Nicolson and Fawcett (1994) suggest that dyslexics suffer from problems with a wide range of primitive skills and that the underlying cause must lie deep in the central nervous system as the research surrounding magnocellular theory along with diffuse cortical abnormalities would suggest that there are more anatomical anomalies in the dyslexic brain than the cerebellum alone. Also, if cerebellar dysfunction causes the behavioural manifestations of developmental dyslexia, it would be reasonable to conclude that dyslexics would resemble patients with acquired cerebellar damage and this has not been supported by research (Zeffiro and Eden, 2001). Thus, the theory of automatization deficit and the cerebellar hypothesis are descriptive rather than explanatory theories and the mechanism underlying these deficits must be sought.

Zeffiro and Eden suggest instead that damage to cerebellar circuits such areas in the perisylvian neocortical regions, which receive projections from the cerebellum via the thalamus and project to the cerebellum via corticopontine projections to the pontine tegmentum, could mimic cerebellar disease but yet lead to a wider myriad of disruptions than cerebellar abnormalities alone. Moretti, Bava, Torre, Antonello and Cazzato (2002) suggested that reading mistakes of patients with cerebellar lesions was a result of possible alteration of the diffuse connection system from the cerebellum to different cerebrocortical and subcortical structures concerned with attentive, alerting and linguistic processes. To account for the wider structural anomalies, a theory of cerebellar-vestibular dysfunction in dyslexia was developed (Levinson, 1988). The vestibular cortex, located in the inner ear, carries information regarding the current position of the different body parts in relation to each other and in relation to inertial and gravitational forces and is involved in spatial awareness and temporal information (Goddard-Blythe, 2001b). Levinson found that, using a set of diagnostic methods and criteria for cerebellar-vestibular dysfunction, over 94% of both learning disabled and dyslexic samples showed two or more abnormal neurological or ENG parameters, whereas only 1% had neurological signs of a cerebellar disorder. It was proposed that learning disabilities and dyslexia might reflect a single cerebellar-vestibular-based disorder, shaped by a diverse group of cerebellar-vestibular

determining mechanisms rather than a distinct neurophysiological disorder, and which may also trigger altered and compensatory cerebral processing and dominance mechanisms leading to a unique manifestation of the disorder in each individual. Indeed, it has been shown that anti-motion-sickness medications, which may be considered cerebellar-vestibular stabilizers, can improve reading performance in dyslexia (Wilsher et al., 1987, cited in Rae et al., 1998). Furthermore, both the vestibular nuclei and the cerebellum are regulators of muscle tonus and are interconnected (Krkljes, 1990). Furthermore, eye movement aberrations in cerebellar patients show evidence of an intact hyper-responsive vestibular system (Cooper and Pivik, 1991) indicating that the vestibular system is dependent on the inhibitive influence of the cerebellum for proper function. These studies collectively show that it may not be cerebellar dysfunction per se that causes dyslexia but a disruption of the cerebellar projections which link areas of diffuse functions, such as linguistic and motor, which would lead to a unique pattern of difficulties in each dyslexic person, which would depend on which areas had been disconnected from the cerebellar areas.

Recently, this view has been challenged by a view of dyslexia as delayed or abnormal development. A maturational lag could account for many of the symptoms of dyslexia. For example, poor saccadic eye movement control at a particular age could be a marker for developmental delay (Biscaldi, Gezeck and Stuhr, 1998). Saccadic eye movement control rapidly develops from childhood to adulthood in a predictable pattern and similar development is seen in dyslexic children but at a much slower rate and early visual and visuomotor problems strongly correlate with sub-types of dyslexia (Fisher, Biscaldi and Otto, 1993; Fisher et al, 1993, cited in Biscaldi, Gezeck and Stuhr, 1998).

The next section discusses a theory of maturational lag as a causal factor in dyslexia and attempts to do this by discussing foetal movement patterns and their role in development.

1.7 Theory of Persistent Reflexes

A neural basis for a maturational lag that could produce a range of deficits observed in dyslexia is represented in the theory of persistent primitive and postural reflexes.

1.7a Definition of Postural and Primitive Reflexes

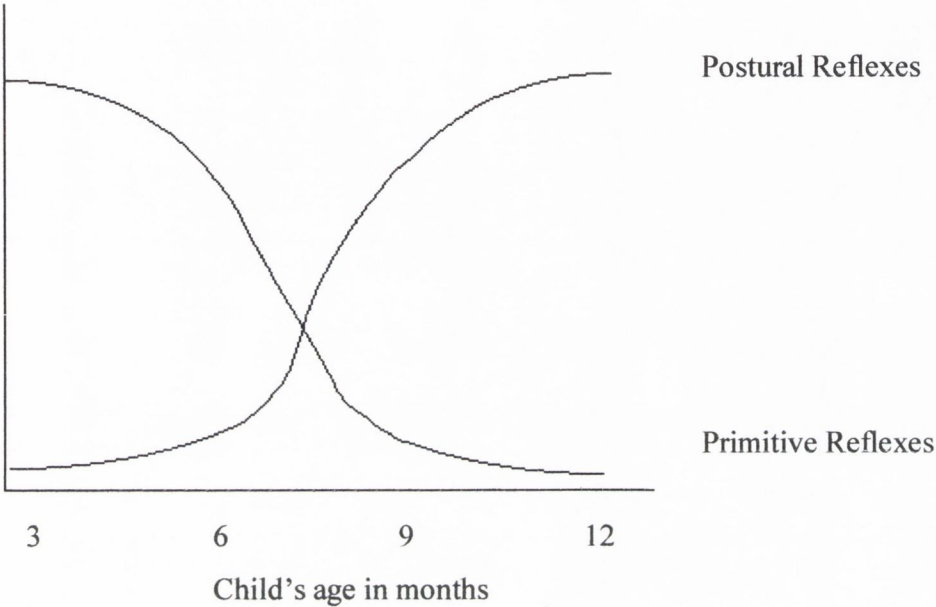
A primitive reflex is one of the names given to the responses and activities that may be observed in the newborn and young infant (Touwen, 1984). A reflex action involves a prompt, consistent, stereotyped response to a specific stimulus that shows that the underlying neurological pathways are already established and defined. There is no opportunity for variation or choice of action (Holt, 1977). They are called primitive reflexes because they are transient in nature (Prechtl, 1984), beginning in utero, and then tend to disappear at around eighteen weeks (Okado and Kajima, 1984). The observation of the appearance and evolution of reflex activity from foetal life onwards provides insight into the elaboration of the nervous system (Holt, 1977).

Normal infants are born with numerous primitive reflexes under the control of centres such as the brain stem, cerebellum, midbrain and basal ganglia. The cerebral cortex, as it develops, eventually inhibits these centres leading to a decrease in primitive reflexes and they eventually become integrated into more functional postural and voluntary motor movements. Purposeful motor movements and postural changes depend on the appearance and subsequent integration of primitive reflexes. Piaget named these movements secondary circular reactions and considered them as a necessary stage in cognitive development (Piaget, 1952, cited in Santrock, 1997). Primitive postural reflexes follow an orderly sequence of onset and disappearance starting at the foetal period and extending through the first year of life (Gesell, 1954; Andre-Thomas, 1959, cited in Capute, Accardo, Vining, Rubenstein and Harryman, 1978). Postural reflexes also appear in the foetal period and do not disappear but actually persist throughout life. The temporal history of each can be seen in Figure 2 below.

Figure 2: The Persistence of Primitive and Postural Reflexes

(taken from Capute, Accardo, Vining, Rubenstein and Harryman, 1978)

Performance



There are three types of postural reflex, attitudinal reflexes, righting reflexes and statokinetic reflexes (Gesell, 1988). The attitudinal reflexes determine orientation of the body with respect to gravity. The foetus distributes tonal charges to his limbs that become more controlled with age. The head and neck exert influence on the attitudes in the womb producing a tonic neck reflex and reaction to gravity is mediated by proprioceptive impulses arising from the neck in torsion. The main groups of attitudinal reflexes are the tonic neck reflexes and the tonic labyrinthine reflexes and the changes in degree and distribution of tone produced by these reflexes are sustained and last as long as the position of the head is maintained (Bobath, 1971). The asymmetric tonic neck reflex (ATNR) consists essentially in an increased tone in the extensors of the chin extremities and in a decreased tone in homologous muscles of the occiput extremities when the head is turned to the side (Vasella and Karlsson, 1962). The tonic labyrinthine reflex is initiated by the stimulation of the labyrinths of the semi-circular canals and leads to changes in muscle tone of the arms, shoulders, head and trunk. The righting reflexes are

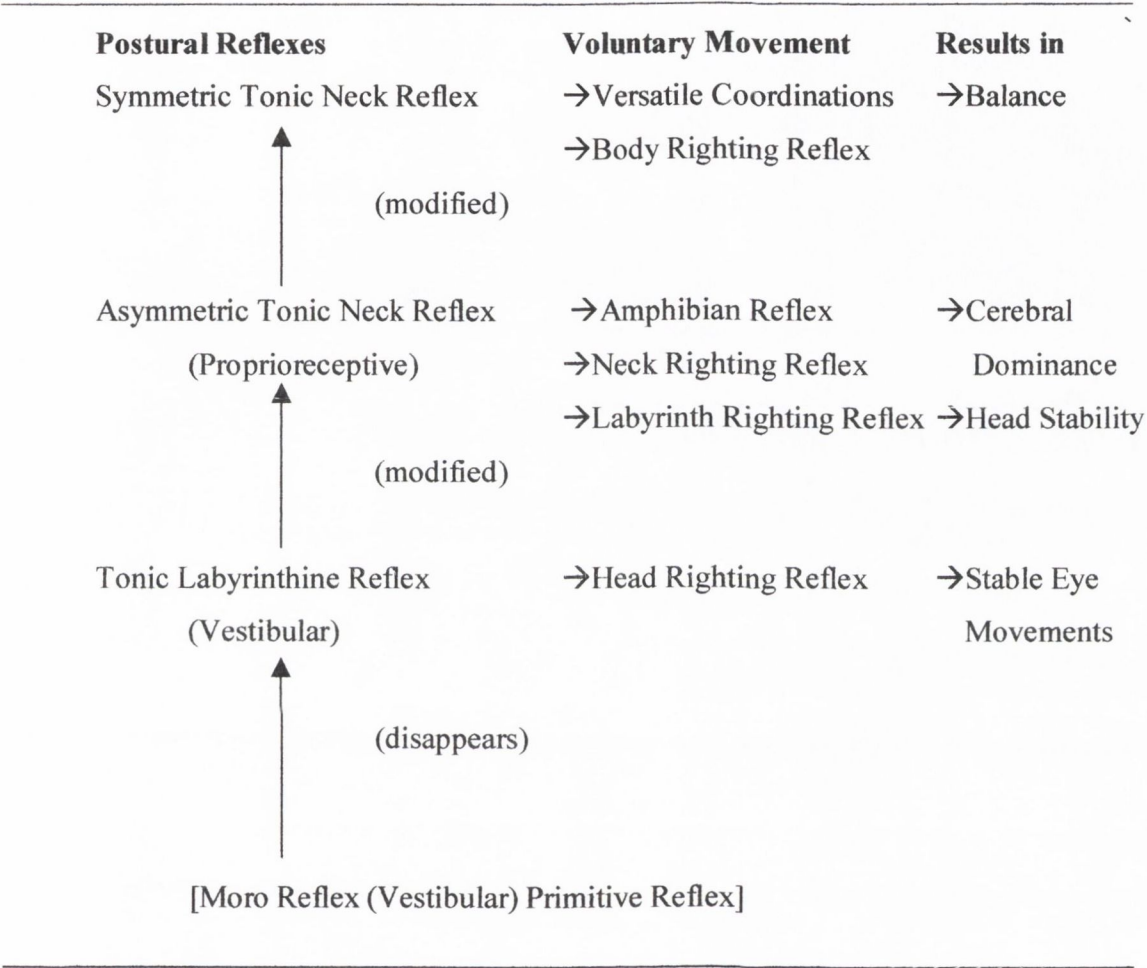
triggered by stimulation of the otoliths and semi-circular canals that react to change of position and to acceleration of movement respectively. The statokinetic reflexes are produced by movements and are designed to keep the organism moving in the right direction.

1.7b The Evolution of Postural Reflexes

The simplest purposeful motor movements and postural changes depend on the appearance and subsequent integration of primitive reflexes (Capute et al. 1978). There is a fixed pattern of integration of postural reflexes in order to produce voluntary movements and these are discussed in *A Teacher's Window into the Child's Mind* by Sally Goddard (Goddard, 1996) and will be summarised here. The earliest reflex, the Moro reflex appears at nine weeks in utero and is present at birth. It is an involuntary reaction to threat that activates the sympathetic nervous system. The Moro reflex is a primitive reflex and so it is necessary that this reflex disappear for postural reflexes to appear and so needs to be mentioned here. At between two and four months, this reflex is inhibited and replaced by the adult startle response. If this inhibition fails to occur, the child may be highly sensitive to stimuli and in a permanent state of arousal and hyper-vigilance. The Tonic Labyrinthine Reflex or TLR, a postural reflex, emerges in utero and is present at birth. The Moro and TLR reflex are closely linked in early months of life as both are vestibular in origin and are activated by an alteration of position in space but they differ in their classification. The TLR is a primitive method of response to gravity and a postural reflex and so does not disappear like the Moro reflex. Between the ages of four and six months, the response is merely modified so that head control can develop and, at this time, the oculo- and labyrinthine head righting reflexes emerge. Head control is an essential prerequisite for the development of all later functions and aids movement, tonus and balance in early childhood. The integration of the TLR by the increasing strength the head-righting reflex leads to stable eye movements. The tonic neck reflex pattern, a postural reflex, is a fundamental feature of the development of human behaviour. There is also an interaction between the posturing of the eyes and the posturing of the limbs and trunk. Posturing involves the combination of both labyrinthine and proprioceptive impulses. The asymmetric tonic neck reflex (ATNR) is present in the first twelve weeks

and by sixteen weeks the infant displays freedom of head movements with a variety of arm and leg movements and a decrease and modification in asymmetric attitudes. By twenty weeks the mid-position of the head prevails and symmetric arm and leg posturings predominate (Hill and Volpe, 1999). These changes between twelve and twenty weeks coincide with maturing oculomotor control and cerebral dominance. Thus, as the ATNR diminishes and is modified, the symmetrical tonic neck reflex (STNR) predominates and oculomotor control develops. The STNR is eventually integrated into versatile coordinations that display varied patterns of coordination. By seven to twelve months, the neck-righting reflex is modified by the body-righting reflex, which has an important role in the child's early attempt to sit and stand (Illingworth, 1987). By the time the individual has acquired anti-gravity control and the resultant movements, further development occurs as the child tries to put them to a variety of uses, for example, the development of sensorimotor integration (Holt, 1975). Thus, with the evolution of one postural reflex to another, each reflex appears to disappear, but in actual fact, the reflexes are modified to produce more sophisticated movements eventually leading to versatile coordinations and balance (See Figure 3 below).

Figure 3: The Evolution of Postural Reflexes to Voluntary Movement
 (Adapted from Franklin, 2000, Personal Communication, and Illingworth, 1987)



Both primitive and postural reflexes combine a substrate for the development of voluntary motor control for which they are eventually integrated (Easton, 1972, cited in Capute et al., 1978). The integration is often grossly interpreted as disappearance during early life and so both primitive and postural reflexes are often both classed as primitive reflexes (Capute et al., 1978). However, the postural reflexes do not disappear and frequently re-emerge with cerebral insults (Bobath, 1971). Thus, it is more appropriate to class the TLR, ATNR and STNR as postural reflexes which are modified but do not disappear.

1.7c Function of Primitive and Postural Reflexes

Primitive reflex movements are adaptive movements for that stage of life in which they appear and are not immature adult reflexes as the name suggests (Oppenheim, 1984). Prechtl (1984) has listed three main functions of primitive reflexes; they affect the survival or development of the foetus and are specifically adapted to the intra-uterine conditions, they anticipate post-natal functions such as breathing movements that begin in utero and possibly have a role in the formation of the skeletal system as movements become more complex. After birth and up until about eighteen weeks, the neonate displays amazing continuity of movement patterns, for example, the stepping movement observed in the womb lasts until week eight to ten after birth (Touwen, 1976, cited in Prechtl, 1984). To account for the continuity of these reflex movements, some theories state that human infants are born at least a year too early, as the nervous system is not fully developed at birth (Prechtl, 1984), and that the movements are not uncoordinated and random but specific and patterned and seem to be useful (de Vries, Visser and Prechtl, 1984). The first appearances of primitive reflexes occur in mid-gestation and peak at about six to seven months after birth before declining (Thelen, 1979). Thelen in 1979 noted the quantity and variety of rhythmical and highly stereotyped behaviour of infants, which were interpreted as manifestations of incomplete cortical control of endogenous patterning in maturing neuromuscular pathways. Prigogine and Stengers (1981) speak of a “pre-biological adaptational mechanism” in order to explain these stereotypical behaviours stating that this system shows an adaptive organisation in a predictable way although the environment is variable (cited in Touwen, 1984).

Postural reflexes, a subset of primitive reflexes, contribute to maintaining the characteristic orientation of the body in space with respect to gravity (labyrinthine reflex) and with respect to the interrelationship of body parts (tonic neck and righting reflexes) and the automatic control of balance and co-ordination, and persist into adulthood. Thelen (1979) noted that by the end of the first year, the overall frequency of stereotyped behaviour is declining but the number of different stereotypies rises, with new stereotypies being added to the repertoire without the loss of the older behaviour. Because postural reflexes are smoothly integrated into adult motor behaviour they can be grossly interpreted as disappearing in the early period of life and, therefore, both are described as primitive

reflexes but, in reality, increase in strength as components of voluntary movements (Capute, et al., 1978). For example, hands-on-knees rocking has been interpreted as one of the steps an infant goes through after a creeping posture is assumed but before forward progress can be made. Thus, the primitive reflex appears, or reappears, in a transition stage of motor behaviour (McGraw, 1941; Louie, 1949, cited in Thelen, 1979). Such responses are interpreted to appear at precise stages in neuromuscular maturation because of immature voluntary control over newly functional pathways and may be seen with greater frequency in human infants because of their comparatively slow maturation rates and involve the neuromuscular pathways which will be needed for the next stage of maturational development. As maturation enlarges processing capacity, reflexes are replaced with goal-directed behaviour (Thelen, 1979). Specific combinations of primitive and postural reflexes differ for each motor skill.

Each of the postural reflexes has different functions in early life (Goddard, 1996). The Moro reflex is an early startle reflex and survival mechanism to alert, arouse and summon assistance. In utero, the Moro reflex plays a major part in the baby's breathing mechanism; its appearance coincides with the earliest breathing-like movements in the womb and the first breath after birth. The TLR is a primitive method of response to gravity after birth and ensures that any movement of the head in a vertical direction beyond the midline will cause extremes of flexion or extension throughout the body to occur, training balance, muscle tone and proprioception in the early months. It ensures a smooth transition from the reduced-gravitational environment in the womb to the gravitational pull of the outside world. The ATNR facilitates movements, develops muscle tone and provide vestibular stimulation in the womb. Thus, continuous motion, provided by the ATNR, stimulates the balance mechanisms and increases neural connections in the womb. The ATNR also participates in the birth process. The reflex is activated by the contractions of the birth canal and, once the head presents, the midwife turns the head from side to side so that the infant can ease down the birth canal by twisting and turning from one side to the other. During the first months after birth, the ATNR ensures that the windpipe is open when the baby is in a prone position and increases extensor muscle tone on each side of the body, providing a basis for later reaching movements and hand-eye coordination. It later enables the child to creep and crawl on the

stomach. This is replaced by the STNR that allows the baby to defy gravity by rising up on hands and knees from the prone position to allow the baby to creep and crawl on hands and knees. This allows the eyes to cross the midline, integrating the vestibular, proprioceptive and visual systems to give a sense of balance, space and depth. Each reflex has a purpose and will not be fully suppressed unless it has successfully completed its developmental task (Goddard, 1990). Normal motor development represents the gradual unfolding of a child's latent abilities, modifying and adapting simple movements into finer and more selective movement patterns and skills, with the greatest and fastest changes taking place in the first eighteen months (Bobath and Bobath, 1975).

1.7d The Embryology of Behaviour

The motor system of the infant shows adaptive organisation in a predictable way despite the change of environment from the womb to the extra-uterine world. The nervous system has the specific property of reacting to very small changes in the environment by a reorganisation and reordering of the whole structure, resulting in an efficient and highly reliable functional system. Thus, during the reorganisation of structure, functional activities will be switched off in favour of new, more adaptive repertoires and so reflexes give way to more complex motor movements. The development of the nervous system has as much to do with atrophy as with growth. Cajal observed that the dendrites of spinal motor neurons and cerebellar Purkinje and granule cells undergo great changes during development, some regressive (cited in Jacobson, 1970). In the initial outgrowth phase, an excessive number of dendritic branches, apparently directed at random, are followed by a phase of regulation and re-absorption of the dendrites. Selection among the dendritic outgrowths takes place on a functional basis. Als' (1984) synactive theory of development states that at each stage of development there is a drive for integration of subsystem functioning fueled from within and affected by the environment. However, in the womb, there is little exogenous stimulation and so the movements the foetus makes actively shapes its neurological development. There is much evidence to suggest that deliberate movements by an organism actively shape the development of the brain. Connections in the brain can be altered via the synchronization of inputs that forms the basis of associative learning (Hebb, 1949, cited in Gazzaniga, Ivry

and Mangun, 1998). The more stimulation a particular brain area gets, the more cortical representation it develops and the greater connectivity of a brain circuit. For example, studies using fMRI signals show that the left hand digits of string players have more cortical representation than the right hand (Hund-Georgiadis and von Cramon, 1999).

The cognitive growth of the foetus, or for that matter the child and adult, depends on the capacity of neurons to undergo embryonic growth that is the basis of plasticity (Gesell, 1988). The onset of reflex movements in the monkey foetus has been correlated with the first appearance of synapses in the spinal cord and it has also been shown that motor neurons develop before sensory neurons in the same region of the nervous system (Bodian 1966; Morest, 1969 cited in Jacobson, 1970). In the human foetus, the ATNR provides continuous motion, which stimulates the balance mechanism and increases neural connections. The reflex facilitates the kicking movements of the foetus, develops muscle tone and provides vestibular stimulation (Goddard, 1996). Myelination, the process which insulates the neurons to allow electrical impulses to be transmitted more quickly along the nerve fibres, is not completed in the womb but continues after birth, and the course of myelination is accelerated by increasing mobility of infants during their first year of life (Carlson, 1999). Indeed, it has been shown that myelination of the central nervous system is enhanced during the creeping and crawling stages and crawling on the stomach is facilitated by the presence of the ATNR in the first few months after birth (Goddard, 1996). These observations suggest that the initial reflex movements that a foetus makes are produced endogenously and not as a reaction to stimuli. Movements, however slight, have a directive influence on growing nerve fibres. The primary tonus of the foetus is also endogenous and more sustained tonal reactions occur as the connections with spinal neurons matures. Therefore, the foetus is active in its development and growth. In the absence of such movements, therefore, it would be expected that an infant might display a developmental lag. The child's innate drives and neural capacities lead to an effortful mastery of demands such as gravity, sounds, sights, tactile stimuli and those arising from muscles, which all influence the growth of the nervous system. Therefore, a child whose central nervous system has not developed in a normal manner is unable to organise a response to these demands and this child is likely to become learning disabled (Ayres, 1972).

1.7e The role of persistent reflexes in maturational lag

The persistence of primitive reflexes can lead to a maturational lag. However, the interweaving of primitive reflexes with voluntary neuromotor function is most clearly visible in the reappearance of these infantile reflexes in older children and adults who have suffered brain damage (Bobath, 1971) or when they are not sufficiently subsumed into voluntary movement and disrupt its function (McPhillips, 2000). With cerebral insults, however, primitive reflexes reappear and are considered neurodevelopmental markers for brain dysfunction (Capute et al., 1978) or an indication of demyelination in progressive diseases such as multiple sclerosis (Goddard-Blythe and Blythe, 1995). For the maintenance of posture and equilibrium, the central nervous system utilizes lower centres of integration with their phylogenetically and ontologically older patterns of co-ordination, namely, the cerebellum, mid-brain and basal ganglia. The release of motor responses integrated at these lower levels from the restraining influence of higher centres, especially the cortex, leads to abnormal postural reflex activity (Bobath, 1971). The degree of the release of tonic reflexes, resulting in an inhibition of higher postural reflex activity, appears to be in direct proportion to the severity of the individual case and treatment consists of the inhibition of pathological postural reflexes combined with the facilitation of righting and equilibrium reactions (Bobath, 1985). Righting reflexes modify and inhibit the tonic reflexes and help to maintain muscle tone sufficient to counteract gravity and to allow movement to take place (Bobath, 1971).

Blythe (2000) suggests that some children run the risk of having a specific learning disability, not because of a lack of intelligence, but because the basic systems fundamental to learning are not fully in place by the time they start school and so they may not be neurologically ready for new learning. Motor skills provide outward signs of central nervous system maturity in the relationship between brain and body. It is between the ages of six and a half and eight years that connections are strengthened between the balance mechanism, the cerebellum and corpus callosum, which work together to give egocentric information, which is needed for sense of direction and a functional relationship between visual perception and balance. Thus, a child's biological stage is more important than chronological age in deciding when they should go to school. If not

at an appropriate stage of developmental readiness, the brain struggles to maintain conscious control over attention, balance and co-ordination, which takes away from learning resources. Therefore, children with persistent primitive reflexes will not be ready to start school at the same time as other children. This is exemplified by the later stage in which dyslexic children learn to crawl and talk compared with other children. Rider (1972) found that learning disabled children had significantly more abnormal reflex responses and lower scores on achievement tests. Thus, pre-school diagnosis is vital.

1.7f The Asymmetric Tonic Neck Reflex and Learning Disabilities

If primitive and postural reflexes persist they will disrupt the normal development of neural and sensory functions (Prechtl, 1984). Assessment of primitive reflexes is important for the recognition of possible neurological damage in the prenatal and perinatal period. Gesell states that the embryology of behaviour can be conceptualised in terms of posture which involves both tonal discharge and the expenditure of energy and that the embryogenesis of the mind must be sought in the beginnings of postural behaviour (Gesell, 1988). Muscle tonus is an involuntary condition of tension and is a continuous type of reflex that allows the maintenance of posture and is necessary for movement. Muscle tone is dependant on an intact proprioceptive arc, its source lying in the muscle itself. Movements of the body stimulate the proprioceptive organs (Bobath, 1971). For Gesell, tonus is behaviour mediated through the motor neurons of the spinal and cranial nerves. The asymmetric tonic neck reflex (ATNR) consists essentially of an increased tone in the extensors of the chin extremities and in a decreased tone in homologous muscles of the occiput extremities when the head is turned to the side. The reflex is tonic meaning that the tone of the extremities is influenced as long as the head is kept rotated or tilted and it is referred to as asymmetric to distinguish it from the tonic reflexes in which flexion or extension of the neck leads to symmetrical modifications of tone in all four extremities (Vasella and Karlsson, 1962). The ATNR has also been identified in ninety percent of six-month-old patients with spasticity, which lasted into their second year of life and was retained into later childhood if the children were severely handicapped (Paine, 1964). The ATNR is also the most frequently observed persistent primitive reflex in infants with neurological lesions (Paine, 1964) especially of the pyramidal tracts (Vasella and

Karlsson, 1962). Babies with persistent ATNR sit up at a much later stage than other babies because the reflex usually disappears after this stage has been reached, subsumed into the voluntary movement of sitting up without support when placed (Paine, 1964). In a study of 129 infants with abnormal motor development in association with chronic brain syndromes, the ATNR was retained as the only abnormal motor sign, other than delayed milestones in seven of the seventeen cases of general psychomotor retardation (Paine, 1964).

Chronic persistence of the ATNR is associated with neurological problems such as cerebral palsy in which children may have difficulty with movement and will not only develop at a slower rate but also follow an abnormal developmental course (Bobath and Bobath, 1975). When the infant is supine, the infant may seem to lie with head turned to the side with extension of the extremities on that side (chin side) and the flexion of the contralateral extremities (occiput side) and this may also be observed in the sitting position and is then called the “fencer” position. If strong, this reflex will dominate motor behaviour. For example, a patient will only be able to flex an arm by turning the head and can only sit unsupported if the head is in the midline. The slightest movement of the head will produce a tonic neck reflex with a resultant stiffening of one side and loss of balance (Bobath and Bobath, 1971). To test for the reflex the child is placed supine and is first observed for active head turning and subsequent extremity movement. Next, the reflex is elicited by a passive sideways turning of the head (180°) from one side to the other and holding for five seconds when the baby is supine. Response indicating the presence of the reflex involves the extension of the arm and on the side to which the head turns and the flexion of the opposing limbs (Capute et al., 1978). The ATNR reflex is ipsilateral and the receptive field lays in the region of the upper points of the neck especially the atlanto-axial and atlanto-occipital joints (McCouch, Deering and Ling, 1951). The ATNR also influences the grasp reflex, with a reinforcement of the grasp reflex on the face side of the ATNR. The interaction between these two reflexes explains the occasional inclusion of finger extension and flexion in the ATNR pattern (Pacella and Barrera, 1940; cited in Capute et al., 1978).

A milder persistence of the ATNR is associated with less severe disorders such as specific reading difficulties (McPhillips et al., 2000). McPhillips et al. suggest the

persistence of primitive reflexes, which they call primary reflexes, as a neurological basis for dyslexia. The McPhillip's study concentrated on the asymmetrical tonic neck reflex (ATNR) that has an important role in visuomotor development. The ATNR is normally evident at two to three months when it appears to prepare the way for the integration of head turning, visual fixation and reaching and so is probably fundamental to the establishment of visually directed reaching and hand-eye co-ordination (Holt, 1977). Continued persistence of the ATNR interferes with numerous functions, for example, in the young child it is impossible to crawl on the stomach with fluent cross-pattern movements as such movement involves crossing the midline, i.e., crossing into the contralateral space, and a consequent stiffening of one side or the other. Furthermore, the eyes have great difficulty crossing the midline in order to read a sentence fluidly, which can affect both reading and writing (Goddard, 1995). The McPhillips study found that the rehearsal and repetition of the ATNR might inhibit the persistence of the ATNR in later childhood. The repetition of primitive reflex movements normally plays a major part in their ultimate inhibition. Delayed or postponed inhibition can, therefore, be brought about at a much later stage in development than infancy (McPhillips, 2000).

McPhillips et al. (2000) divided a sample of sixty dyslexic children, aged eight to eleven, into three conditions, an experimental condition, a placebo-control and a control group. The experimental group was given a specific movement programme aimed at inhibiting the ATNR based on the replication of primary movements, the placebo-control group was given non-specific movements to rehearse, i.e., not based on the replication of primary movements, and the control group was not given any intervention. The researchers also stated their intention to have a second control group, consisting of dyslexic children who did not have a retained ATNR but reported that a 100% prevalence of the ATNR was found in a sample of dyslexic children using the Schilder test (see section 2.3c for a description) and so the inclusion of this control group was not possible. The researchers reported that the entire experimental group benefited significantly from the repetition and practice of the primary reflex movements whereas changes in the placebo-control and control groups were non-significant. These movements were proposed to have eliminated the persistent primary reflex, specifically the ATNR, which was supposedly causing the maturational lag leading to an improvement of abilities in

many domains including reading. If the prevalence rate of 100% for the ATNR in the dyslexic sample as postulated by the McPhillips study is reliable then dyslexia may have a direct neurodevelopmental cause and a definite treatment.

1.7g The ATNR in the normal population

However, because the McPhilip's study did not use a non-dyslexic control group when testing for the prevalence of the ATNR it could not be reliably established by this study whether the ATNR is present in the non-dyslexic population and not just in a sub-population of dyslexic children. In any case, the ATNR was misclassified by McPhilip's study as a primitive or, as he called it, primary reflex. However, the ATNR normally classified as a postural reflex. Therefore, the assertion that the experimental group improved because this reflex had persisted and had not been abolished through the repetition of movements is at odds with motor developmental theory. The ATNR is not normally abolished but is integrated into voluntary movements. This integration may be related to why the exercises worked. Midbrain mechanisms are responsible for integrating the tonic neck and postural reflexes and are the neurological substrate for all the more highly developed behavioural mechanisms of righting and equilibrium (Denny-Brown, 1962; Rider, 1972, cited in Parmenter, 1975). The neck-righting reflex inhibits the ATNR by enabling the child to resist the automatic reflex to turn the neck and allowing the child to have more control over the movement of the head (Paine, 1964). Also, at twenty weeks of age, tonic neck reflexes tend to disappear and symmetric head and arm postures prevail. The disappearance of these reflexes seems to coincide with increasing voluntary movement abilities and is a developmental rather than a learned suppression (Byers, 1953, cited in Vasella and Karlsson, 1962). Persistent tonic neck reflexes can lead to a failure to develop motor skills (Vasella and Karlsson, 1962). Therefore, the presence of the ATNR per se is, of course, not a pathological neurological sign. However, easy elicitation of the reflex during reflex testing in older children may well be.

Because postural reflexes are smoothly integrated into adult motor behaviour as the higher centres of the brain matures (Ayres, 1972), they are rarely seen except under stressful circumstances such as oxygen deficiency, lack of sleep, heavy exercise or sports (cited in Parmenter, 1975). When stress is eliminated, inhibition returns to normal

(Hellebrandt, Hautz and Krikorian, 1956, cited in Parmenter, 1975). The ATNR can be reliably elicited in the foetus, the normal child and normal adult. Hellebrandt, Schade and Cairns (1962) demonstrated that active rotation of the head of normal adults evoked the asymmetric tonic neck reflex (ATNR) in the sitting, standing and, most reliably, the quadrupedal positions (cited in Parmenter, 1975). Wells in 1944 demonstrated that if extensor muscles of the limbs are rendered hypertonic, tonic neck and labyrinthine reflexes are elicited in a clearly demonstrable fashion. Parmenter (1975) found that varying degrees of elbow flexion or muscle tone changes were elicited in all eighty children tested from first and third grade. Ayres (1972) stated that the extent to which the ATNR is inhibited reflects the degree of maturation of postural mechanisms and stated that the ATNR is elicited more easily under the age of eight because the postural mechanisms are less mature. Ikai (1950) also found that the intensity of the reflex, which was strongest in children aged seven to eight, had diminished by the age of nine to ten and between 11 and 25 years was quite difficult to elicit. Carr and his colleagues (1974) postulated that this occurs not because the reflex disappears but because older children (post puberty) are able to maintain more elbow extension than the younger children (cited in Parmenter, 1975). The presence of the ATNR per se is not indicative of neurological immaturity but diminished inhibition of the ATNR is (Parmenter, 1975). Therefore, although all children may have an ATNR reflex, only the inability to inhibit it is indicative of neurological immaturity as older children should have the muscle tone to resist the reflex. Although the influence of the ATNR is present throughout life, the age of the child and the degree to which the ATNR is integrated into the central nervous system may determine the ease with which the response is elicited (Ayres, 1972). Thus, the Parmenter (1975) finding that children in the 3rd grade inhibited the ATNR more than 1st grade children may indicate the degree of maturation and integration of the nervous system. Marinelli (1983) concluded that the ATNR should not be considered pathologic unless it is obligatory, i.e., the child cannot break the reflex while the head is being rotated and does not display resistance in order to break down the change in muscle tone. Also, the ability to elicit must always be compared with what is normal for the child's age (Dodge, 1975). Thus, if a child can inhibit the ATNR, this is indicative of a more mature nervous system

and should be possible in post-pubescent children. From the prenatal period to adulthood, however, this postural reflex is evident.

1.7h Other Reflexes as Predictors for Developmental Lag

However, in the neurological examination of the infant, practitioners rarely look for a specific reflex but a cluster of independent symptoms, which interact to form the pattern of neurological disorder in the infant. In order to examine possible damage to the nervous system, practitioners look at poor sucking, poor tone and abnormal posture, abnormal spontaneous movements and marked asymmetries and neonatal seizures (Prechtl, 1977). The primitive reflexes that are most important for developmental diagnosis are the asymmetric tonic neck reflexes, the grasp reflex, the Moro reflex, the biceps, knee jerk and plantar response, the absence of sucking or swallowing (except in premature babies) and the tonic neck and labyrinthine reflexes (Illingworth, 1987). Illingworth gives the example of the grasp reflex as a possible indicator for neurological damage. The strength of the grasp reflex in a full term baby may be an index of maturation as an advanced newborn may show virtually no grasp reflex. Alternatively, a persistent grasp reflex long after the newborn period is a pointer to neurological damage. Similarly, a persistent tonic neck and labyrinthine reflex can be an indicator of possible cerebral palsy. Bobath and Bobath found that the cause of motor disability for patients with various lesions of the central nervous system – the majority of whom had cerebral palsy – was largely due to the release of abnormal and widespread reflex patterns of posture and movement from the inhibition which was normally under the control of the higher centres of the central nervous system. The central nervous system generally acts as a restraining influence for motor responses integrated at lower levels, namely, the cerebellum, mid-brain and basal ganglia, and without this restraining influence, postural reflex activity is exaggerated (Bobath and Bobath, 1971). The ATNR and the Tonic Labyrinthine Reflex are commonly tested in order to predict neurological damage. It is not the integration of the ATNR but the Tonic Labyrinthine Reflex (TLR) that leads to a head righting reflex stabilisation of eye movements. Both the ATNR and the labyrinthine reflexes are considered to be the most important diagnostic tests for cerebral palsy (Illingworth, 1987). The TLR affects both vestibular and proprioceptive functioning

because any movement of the head forwards or backwards beyond the midplane induces flexion or extension of the muscle groups throughout the body. A retained TLR affects the development of full ocular and labyrinthine head-righting reflexes, essential to body posture, muscle tone and eye movements (Goddard-Blythe and Hyland, 1998). In severe cases of cerebral palsy the child cannot turn to one side as the neck righting reflex is inhibited by the labyrinthine reflex (Illingworth, 1987). Rider (1972) found that children in a perceptual-motor training program showed much higher incidence of abnormal responses at the midbrain level than at any other central nervous system level and that integration of the TLR was directly related to academic achievement. If the TLR is not integrated this will lead to difficulties with eye movements and head righting and, without the head righting reflex, the ATNR cannot be integrated either and children with reading difficulties will still display an easily elicited ATNR but not because it is causal to their problem but merely correlated (Prechtl, 1977).

However, it is not just the ease of elicitation of these reflexes that must be considered, but also the cluster and particular pattern of reflexes with which a child presents. The McPhillips' study has simplified representation of the process to one reflex without considering the interactions between the different reflexes that are present (McPhillips et al, 2000). The movement programme, implemented by McPhillips et al., is based on the cluster analysis theory of the role of motor reflexes and not on one solitary reflex. Individual reflex abnormalities affect functioning of specific skills, whereas the combined effect of a cluster of persistent reflexes has an impact on learning in general and, in particular, those areas of learning that require cooperation of the motor system, such as reading which requires occulo-motor functioning (Goddard-Blythe, 2001a).

As for the link between the ATNR and reading, Johnson (1973) found that the ATNR, tonic labyrinthine, neck righting reflex and bilateral postural integration were all related to reading achievement. In some of the children, mostly male, the ATNR was related to reading comprehension. When inhibitory control of higher centres is disrupted or delayed, primitive patterns dominate to the exclusion of higher, integrated activities and so there is a relationship between postural reflex level and academic performance (Rider, 1972).

However, the theory of persistent reflexes needs empirical data in order to support or refute it and the studies that have been done thus far have not been methodologically sound. Neither McPhillips nor the studies undertaken by the Institute for Neuro-Physiological Psychology in Chester have used control groups of non-dyslexic participants in their studies (e.g., McPhillips, 2002, Goddard-Blythe, 2001a). Furthermore, publications from Chester profess that eighty-five percent of those children of average to above average intelligence who have specific learning difficulties and who do not respond to normal remedial intervention have a cluster of aberrant reflexes (Blythe, 1992). No indication is given, however, of what percentage of children do not respond to remedial intervention. Nor is there any discussion of how to classify the other fifteen percent of children who do not respond to remedial teaching and who do not have reflexes. Furthermore, there is no discussion as to why there should be a difference of this nature between the children. Further research is needed to understand why the movement programme based on these reflex movements improved reading difficulties in the McPhillips study and why the non-specific movement programme did not. A study by Byl, Byl and Rosenthal (1989) assigned dyslexic boys randomly to an exercise programme aimed at stimulating the vestibular system or an aerobic exercise programme (n=22). Only the boys who took part in a vestibular exercise programme made gains in accuracy and speed of spatial perceptual processing. Thus, it may not be the case that the exercise programme used by McPhillips improves the symptoms of dyslexia by rehearsing and inhibiting persistent reflexes, but instead that the vestibular system is stimulated. This would lend support to Nicolson and Fawcett's theory of cerebellar abnormality and refute the theory of persistent primitive reflexes.

This study attempts to investigate the theory of persistent reflexes in a controlled randomised study. The aims of the study will be discussed in the next section.

1.8 The aim of the present study

There are a number of aims of this study. First, the prevalence of a persistent asymmetric tonic neck reflex will be assessed in dyslexic children and an age-matched control group who are not dyslexic. This will throw further light on the validity of the theory of persistent primitive reflexes as an explanatory model for dyslexia, as well as whether persistent reflexes are only present in a dyslexic population, which McPhillips et al. (2000) propose or in all children, which researchers such as Parmenter suggest. Second, one further neuro-developmental reflex, the TLR, will also be assessed in order to evaluate its role because of its link to academic achievement (Rider, 1972) and because its integration is essential to the integration of the ATNR (Prechtl, 1977). Third, an attempt will be made to replicate a relevant finding by Parmenter (1975) who found that children scored significantly higher for persistent reflexes when the head was turned to the right rather than the left when investigating the ATNR. Fourth, the method used by McPhillips to measure the ATNR in his study, the Schilder Test will be contrasted with another clinical method of evaluating the ATNR, the Ayres II Test, in order to assess the reliability of the Schilder Test for the ATNR (the different results found by different investigators may be accounted for by the methodology used). Finally, the relationship between the presence of the TLR and ATNR will be investigated as the TLR is theoretically presumed to predate the ATNR (Capute et al., 1978) and so should not be present if the ATNR has been integrated.

2. Method

2.1 Overall Design of the Study

The type of research undertaken in this study is a field study, a non-experimental scientific enquiry of the interactions between (relations among) psychological and educational variables in real social structures (Kerlinger, 1986). The study is an exploratory field study aimed at uncovering relations between dyslexic status and the persistence of primitive reflex movements. In order to investigate this, the study has a 2 X 2 factorial design. The participants in the study ($n = 49$) were assigned to either a dyslexic or non-dyslexic group based on IQ and reading scores. Thus, the independent variable is dyslexic status. Both groups were compared to each other in terms of a dependent variable, persistence of primitive reflex movements. The study was a double-blind trial, i.e. the examiner did not know which children were dyslexic when conducting the tests to eliminate testing bias and the children were not aware of which of the groups they were in. The participants were age-matched and sex-matched.

2.2 Participants

Sixty Irish schoolboys from working to lower middle class communities in Dublin West were originally selected for the study, but by the time testing had commenced, eleven had changed schools and could not be contacted and so forty-nine children remained to participate in the study. These children were aged between nine years and two months to ten years and five months (mean: 9.75 years, SD: 4.23 months) and so were similar in age range to participants in the original McPhillips study. The children had previously participated in a study testing the validity of the Dyslexia Early Screening Test. Thirty of the sample in this previous study had been deemed to be at risk of dyslexia (MacDonnell, 1995; cited in MacDonnell, 1998). It is through their involvement in this study that the children were recruited.

2.3 Materials

Five tests were administered to the participants:

- The Wechsler Intelligence Scale for Children - Third Edition (WISC-III)
- The Wechsler Objective Reading Dimensions (WORD)
- The Schilder Test for Asymmetric Tonic Neck Reflex
- The Ayres Test II for Asymmetric Tonic Neck Reflex
- A Test for the Tonic Labyrinthine Reflex (TLR)

2.3a The Wechsler Intelligence Scale for Children (WISC-III)

The Wechsler Intelligence Scale for Children - Third Edition (WISC-III) is designed for the assessment of children between six and sixteen years and consists of twelve tests, six rating verbal ability and six rating performance ability, two dichotomous scales identifying two principal modes by which human abilities express themselves. The IQs are calculated on the basis of five verbal and five performance tests. The Verbal Scale consists of information, similarities, arithmetic, vocabulary, comprehension and an additional optional test, digit span. The Performance Scale consists of picture completion, picture arrangement, block design, object assembly, coding and an additional optional maze test. The two optional tests are not included in calculating the child's IQ if all of the other tests can be administered. The Verbal Score is the sum of the child's scaled scores on the five regularly administered verbal tests and the performance score is obtained by adding the child's scaled scores on the five regularly administered performance tests. The Full Scale Score is obtained by summing the Verbal Score and the Performance Score. This score can then be used to determine a child's IQ by reference to norms for his age group at the time of testing.

Confidence in the reliability of the WISC-III is based on the US standardisation study, which showed the stability of scores through a test-retest method of thirty-five students. This concluded that practice effects in the WISC scores were smaller over longer test-re-test intervals (e.g. Juliano, Haddad and Carroll, 1988, cited in Weschler, 1974). Thus, the WISC could be used to test these children, as it had been three years since the previous MacDonnell study. The WISC sub-tests have also been shown to be objective, with an inter-rater reliability on most subtests in the high 0.90s (Wechsler,

1974). A subsequent UK validation study led to the construction of norm tables which correlated closely with the US study (Wechsler, 1974). Thus, the WISC has been shown to be reliable across time and culture. The validity of the WISC has been demonstrated across many studies, for example, the WISC has been shown to measure the factors it claims to measure, verbal ability and performance ability, which combine to form a global intelligence construct, by the work of numerous authors (e.g., Kaufman, 1975, *ibid*). The US and UK standardisation studies also demonstrated strong evidence for the internal validity of the WISC, i.e., that there is empirical evidence for the structure of its scores (Wechsler, 1974). Thus, the WISC has been demonstrated empirically to be both a reliable and valid measure of a child's cognitive ability.

2.3b The Wechsler Objective Reading Dimensions (WORD)

The WORD consists of individually administered tests designed for the assessment of children between six and sixteen years. The three components of the test are Basic Reading, Spelling and Reading Comprehension. The tests take approximately twenty minutes to administer and yield three scores which are combined to calculate a Composite Literacy Score and a sense of quality of a child's attainment in each area results from contrasting performance in each of them. The Basic Reading sub-test consists of a series of pictures and printed words for assessing decoding and word-reading ability. For easier items, the child responds by pointing and for more advanced items the child responds verbally. The Spelling sub-test consists of a series of dictated letters, sounds and words for measuring encoding and spelling ability, which is assessed through written responses. The Reading Comprehension sub-test consists of a series of printed passages and orally presented questions designed to tap skills such as recognition of stated detail and making inferences, some of which are accompanied by a picture-oral response (Rust et al., 1993). A composite of the test scores can also be used to get an age equivalent at which a given raw score is average or typical and a standardised score and percentile rank representing where the score falls on the normal curve.

The WORD has been shown to have reliable results across time and age groups in a study of 367 children from a US standardisation sample who were tested twice (Rust et al., 1993). Accumulated data from studies of the WORD, including content-, construct-

and criterion related evidence of validity has led to the conclusion that the WORD subtests measure the constructs they were designed to measure and norms have been established (e.g., construct-validity assessed by Roid, Twing, O'Brien and Williams, 1992, *ibid*). The test is suitable for investigating discrepancies between a child's reading ability and achievement because it was developed with the WISC - III and normative samples from the two test batteries are linked (Rust, Golombok and Trickey, 1993). Many authors have supported the view that it is important to use normed and linked data for achievement and ability tests in diagnosing and assessing learning difficulties (e.g. Berk, 1984, *ibid*). Thus, this current study used two tests with well established norms and solid evidence for reliability and validity.

2.3c Schilder test for ATNR

To carry out the Schilder test for the ATNR, the child is requested to stand upright with feet together and arms held straight out in front at shoulder level with wrists relaxed. The child is instructed that his/her head will be passively moved from one side to the other by the examiner but to keep the arms in exactly the same position; only the head should move. Standing behind the child, the examiner asks the child to close his eyes while the child's head is turned slowly to one side and then to the other. The child is asked to try to keep his arms in exactly the same position while the head is passively moved. The examiner then turns the child's head 70 - 80° (or until the chin is over the shoulder) and holds that position for five seconds and then repeats this movement over the other shoulder. Again, the position is held and repeated on one side and then the other. If the extended arms move in the same direction as the head turn or the arms are dropped or swaying or loss of balance results, this is an indicator of the persistence of the ATNR. The scoring method used was taken from the original McPhillips study: a negative response, i.e., the child does not move his arms, receives a score of zero. A slight movement of the arms (up to 20°) to the same side as the head is turned or a slight dropping of the arms receives a score of one. An arm movement greater than 45°, either to the side or down or swaying and loss of balance receives a score of three. The score for each side of the body was taken separately and then a total obtained for both sides.

Currently no norms have been established for the Schilder test and its reliability and validity have not been investigated. However, this research will assess the reliability of this test as part of the results and will be presented in the results section.

2.3d Ayres Test II for ATNR

Ayres II involves placing the child in a quadrupedal position with the elbows slightly abducted to prevent the arms from locking into a straightened position. The examiner kneels in front of the child facing him. The child's body weight is evenly distributed between the upper and lower extremities, knees five to six inches apart, arms slightly abducted, elbows extended, wrists hyper-extended in the neutral position, hands flat with fingers extended, head in neutral position and neck relaxed. The elbows are flexed slightly to prevent voluntary locking of the elbows that may account for the failure to elicit this reflex (taken from Parmenter, 1975). The examiner, with two hands on either side of the face, passively turns the head until the chin is over one shoulder joint and holds for five seconds and then over the other and holds for five seconds and any change in muscle tone is noted. The scoring is along a five-point scale and each side is scored separately and then all the scores are combined to give a composite score. A score of zero means no elbow flexion, that is, full elbow extension is maintained and no visible muscle tone changes are noticeable. A score of one indicates a slight muscle tone change, which are minimally visible, but no muscle change. A score of two indicates a slight visible elbow flexion of less than 30° . A score of three indicates an elbow flexion of between 30° and 60° with little shoulder depression and no noticeable trunk movement. A score of four indicates that the elbow flexes more than 60° causing depression of the shoulder with trunk movement but balance is maintained and the contralateral leg does not leave the surface. A score of five is recorded when an obvious loss of balance occurs when the elbow flexes more than 60° and, in addition, the forearm may touch the surface, the contralateral leg may leave the surface and the quadrupedal position may be lost causing collapse of the body position.

Currently no norms have been established for the Ayres II test and its reliability and validity have not been investigated. However, this research will assess the reliability of this test as part of the results and will be presented in the results section.

2.3e A Test for the Tonic Labyrinthine Reflex (TLR)

The test for the Tonic Labyrinthine Reflex must be administered to the child without socks and shoes and with trousers raised slightly to the knee. The child stands with hands by his side and his eyes closed. The examiner moves the head up and back so that the back of the head approaches the back of the neck. The head is then moved down so that the chin rests on the chest. A child with a very persistent TLR will lose balance, or in extreme cases, fall over and these are scored as a three and a four respectively. If a child has a mild TLR he may move the toes as if trying to hold on to the floor (scored as a two) or the ligaments in the lower leg and feet will be seen to move as the child struggles to keep balance without moving the feet (scored as a one). And, if none of these reactions are noted, the test is scored as zero (Franklin, 2000, personal communication).

Currently no norms have been established for the TLR test and its reliability and validity have not been investigated. However, this research will assess the reliability of this test as part of the results and will be presented in the results section.

2.4 Procedure

2.4a Before the testing session: Preparation

Fifty boys from the original MacDonnell study were located, twenty-five who had been deemed at risk for dyslexia and twenty-five controls. In the weeks preceding the testing session, letters of permission were sent to all the parents of the participants (See Appendix B) and signatures for each participant were returned. The six schools were also sent letters and signed agreement from the principal obtained (See Appendix A). A separate information sheet and letter were also sent requesting permission for the child to take part in the reflex tests, so that the parents were aware of this new addition to the testing procedure and to get permission to videotape the session (See Appendix C). The researcher also arranged to meet some of the parents to discuss these tests and the theory behind them so that informed consent could be obtained from the parents. One parent did not give consent, reducing the sample to forty-nine.

2.4b During the testing session: Administering the tests

The tests were given to the participants in a quiet, designated room in the child's own school. Each participant was first given the WISC-R that took, on average, seventy minutes and given a break. The participants were then brought back and the WORD was administered which took, on average, twenty minutes and then the three reflex tests which took, on average, five minutes. The three reflex tests were videotaped because of the practical difficulties of conducting the tests and scoring them simultaneously and also to enable inter-rater reliability to be scored on the video at a later stage due to the subjective nature of the scoring involved.

2.4c After the testing session: Collating and analyzing the data

Scoring the Reflex Tests

The reflex tests were scored before the intelligence and reading tests to avoid observer bias when watching and rating the video data. It was, therefore, not known which children had performed better on intelligence and reading tests before viewing and scoring their reflex movements. A copy of the scoring instructions and scoring sheet is given in Appendix D. The three reflex tests were scored first by the examiner and then by a colleague separately. Six of the children were used as practice (three dyslexic and three non-dyslexic by chance; this was not known to either examiner) and then ratings for the other forty-three participants were correlated to obtain a measure of inter-rater reliability. For each of the reflex measures, the right and left side were scored separately and correlated with the second rater's scores using the Spearman Rank Correlation Coefficient.

Next the WISC-III tests were scored to obtain a full scale IQ (F.S.I.Q.). The standard scores for each of the WORD sub-tests were calculated in order to decide which of the children had a specific difficulty in any or a combination of the different sub-tests. A specific difficulty is defined as present if a score is one standard deviation below the norm on the sub-test in question, i.e. a score of 85 or less on the reading or spelling sub-tests (MacDonnell, 1998). The age equivalent scores were then obtained in order to

calculate a discrepancy between the predicted age of the child and the reading age as scored in the Basic Reading Sub-test. The scores were used to calculate which children were dyslexic and which were not, based on the discrepancy between predicted reading age and IQ scores using the predicted-achievement method. Any child found to have a discrepancy of two years or more between predicted age and reading age on one or more WORD sub-tests was diagnosed as being dyslexic (Dyslexia Association of Ireland, personal communication, 2001).

When it was decided which children were dyslexic and which were non-dyslexics based on this criterion, the reflex scores on the two ATNR tests and the TLR test were compared in order to determine prevalence in both the dyslexic and non-dyslexic groups using the biserial correlation method. A factorial analysis of variance was then run on the data in order to ascertain whether there was any association between dyslexic and non-dyslexic status and any of the reflex test scores or whether the side to which the head was turned differed significantly as Parmenter had reported (Parmenter, 1975).

The Schilder test was also correlated with the Ayres II test as a measure of early reflexes. Next, the raw data were analysed to see whether any of the children scored zero on the reflex tests, which would indicate that these reflexes are not present. Finally, the relationship between the presence of TLR and ATNR was analysed as the TLR is theoretically presumed to predate the ATNR and so should not be present if the ATNR has been integrated. All of these results are presented in the next section.

3. Results

3.1 Inter-rater Reliability for Scoring Reflexes

The video of the children participating in the reflex movements was scored twice independently, once by the researcher and once by a colleague. Six of the children were used for training and in order to agree a baseline for scoring procedures, and were not used in the final analysis for inter-rater reliability ratings. A copy of the criteria and scales used to rate each reflex is included in Appendix D. Forty-three children’s reflex movements were scored separately and the level of agreement analyzed using Spearman Rank Correlations. Table 1 below summarizes the inter-rater reliability for the different reflex measures.

Table 1: Inter-rater Reliability between Observers for Reflex Scores

Reflex Test	Spearman Correlation
Schilder Right	0.806*
Schilder Left	0.840*
Ayer’s Right	0.526*
Ayer’s Left	0.580*
TLR Up	0.631*
TLR Down	0.807*

* Indicates that result is statistically significant at the .01 level (2 tailed).

All of the correlations between raters were significant at the .01 level. The most highly correlated observations between raters were on the Schilder left scoring highest with rho = 0.840, followed by the TLR down test with rho = 0.807 followed by the Schilder right with rho = 0.806 The next most reliable inter-rater score was the TLR up test with rho = 0.631 followed by the Ayres tests with rho = 0.580 on Ayer’s left and rho = 0.526 on Ayer’s right. Thus, the Schilder right and left and TLR down were the most highly correlated reflex measures between raters. This is represented graphically below in scatter plots Figure 4 to Figure 9. (In all scatter plots n=49 but repeated scores occlude each other and appear as only one dot on the graph).

Figure 4:

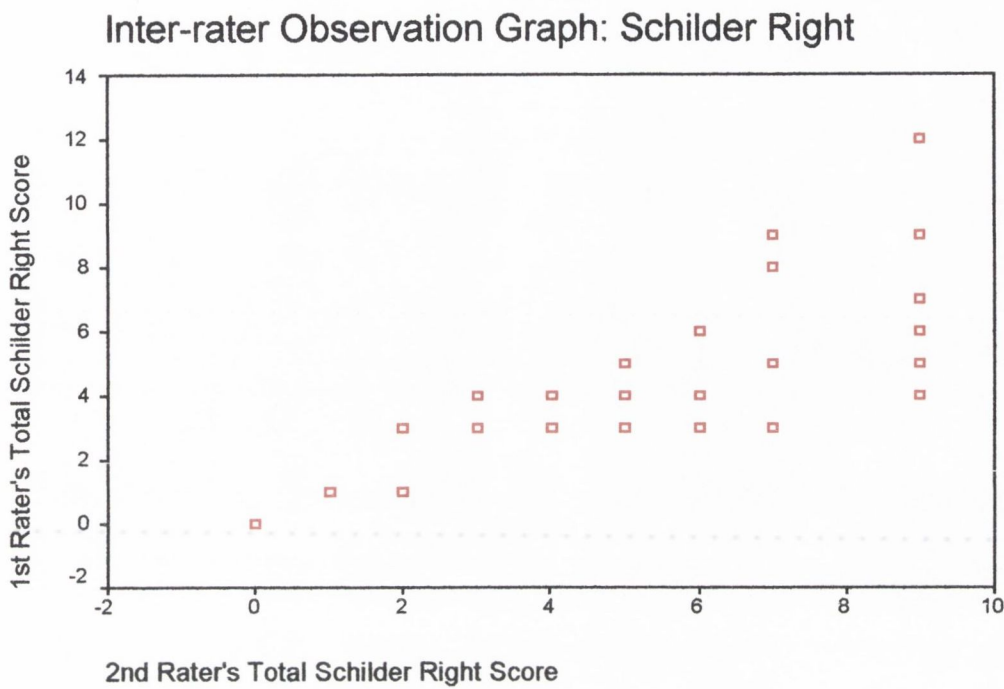


Figure 5:

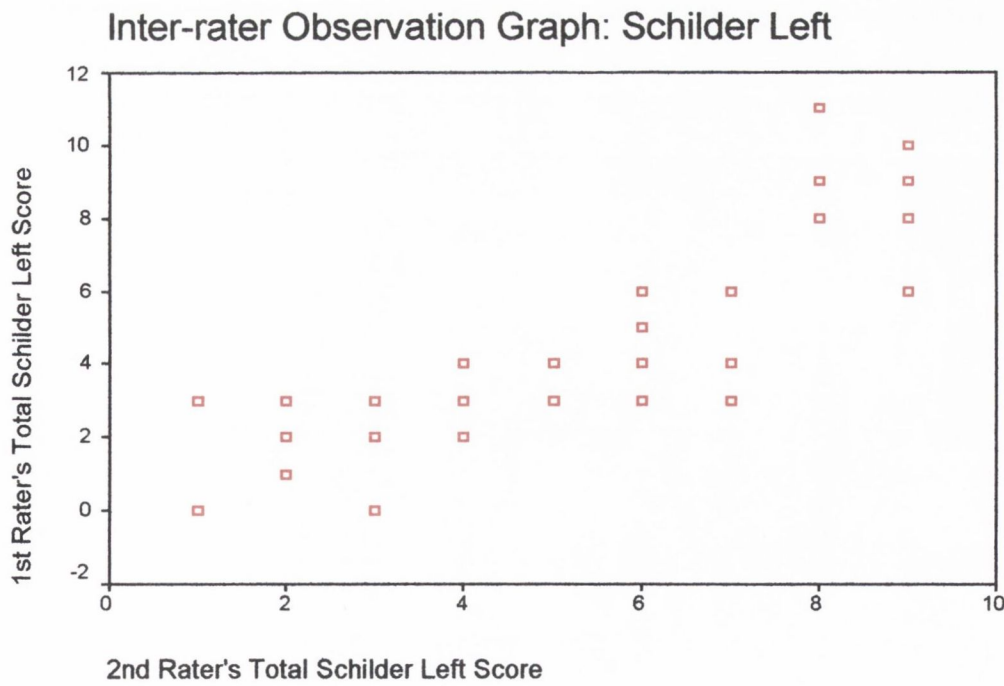


Figure 6:

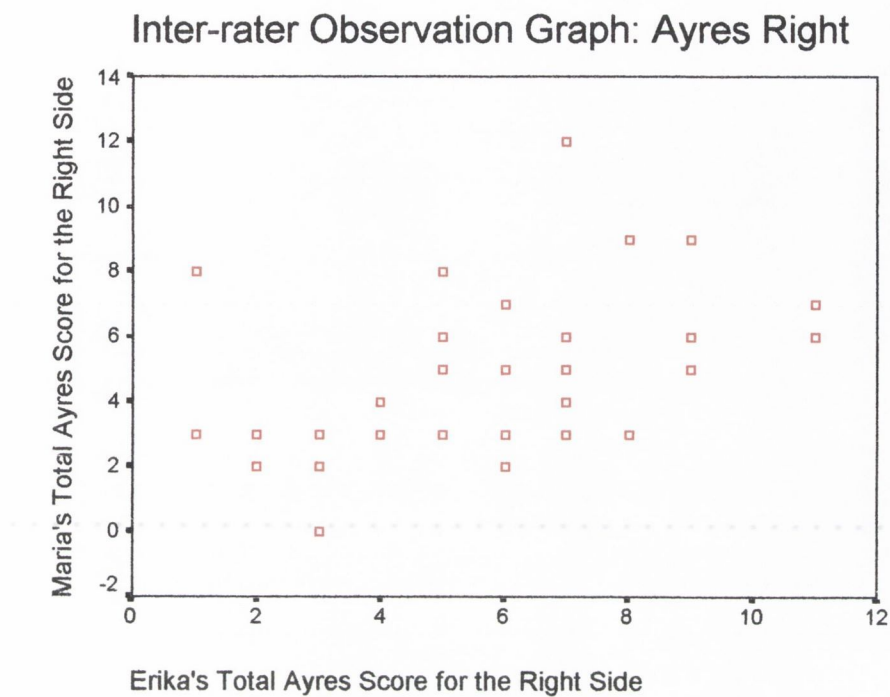


Figure 7:

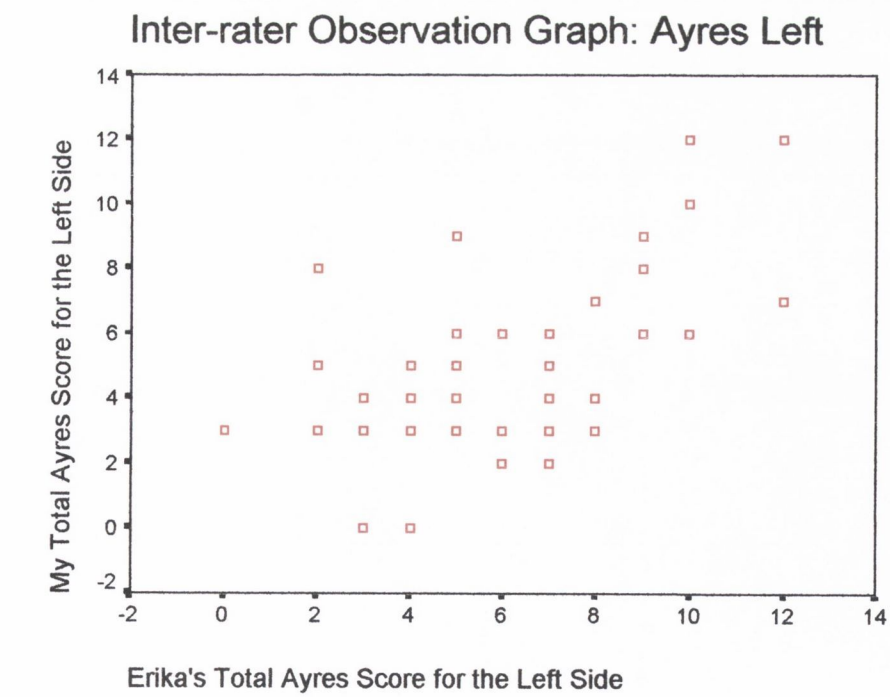


Figure 8:

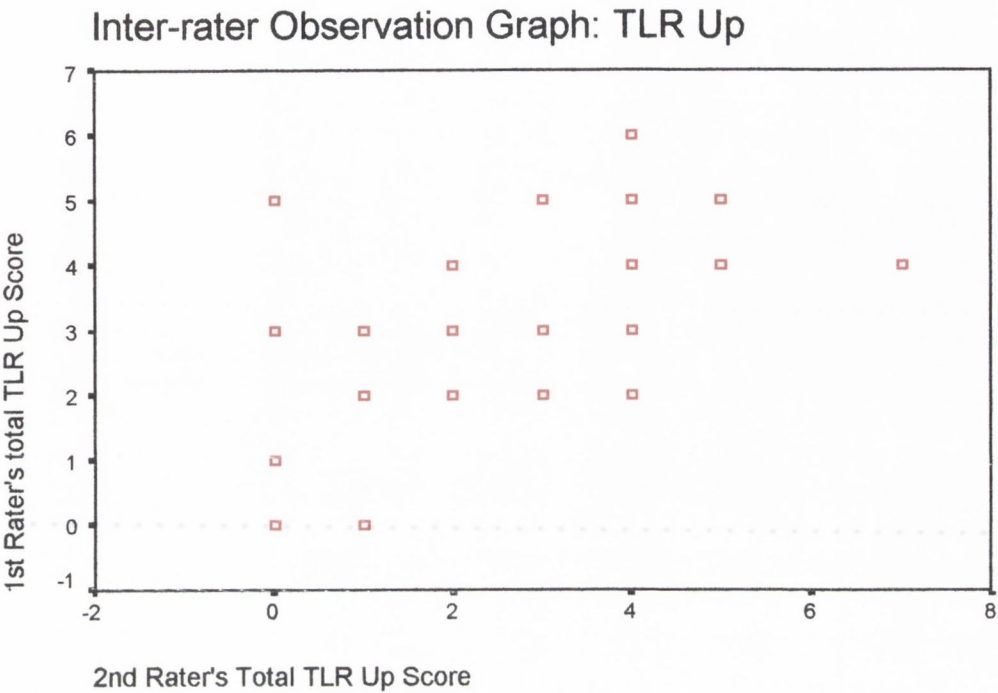
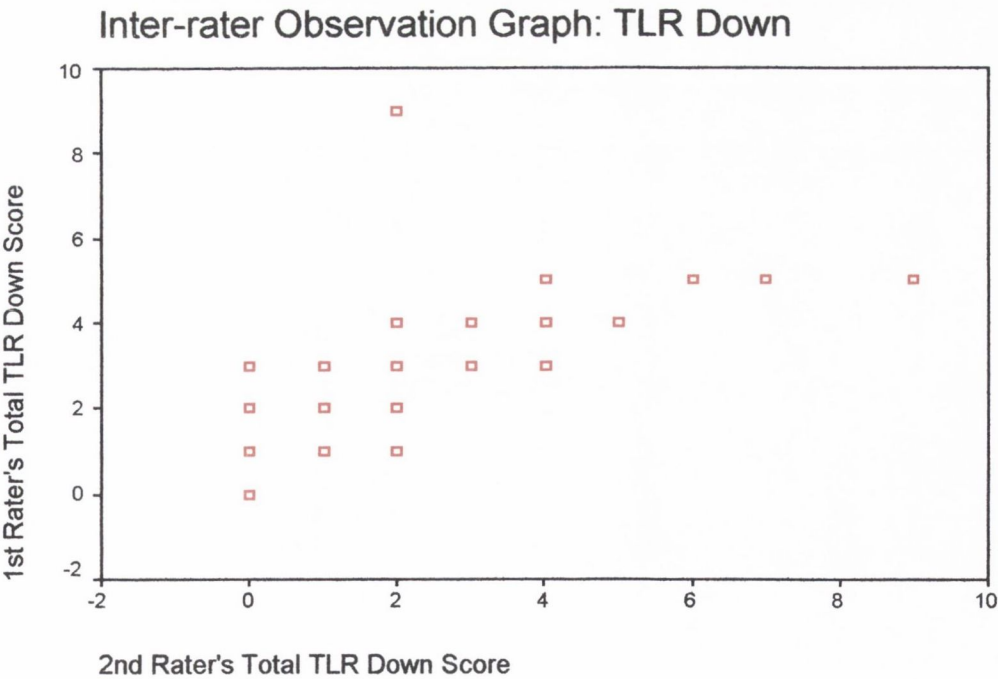


Figure 9:



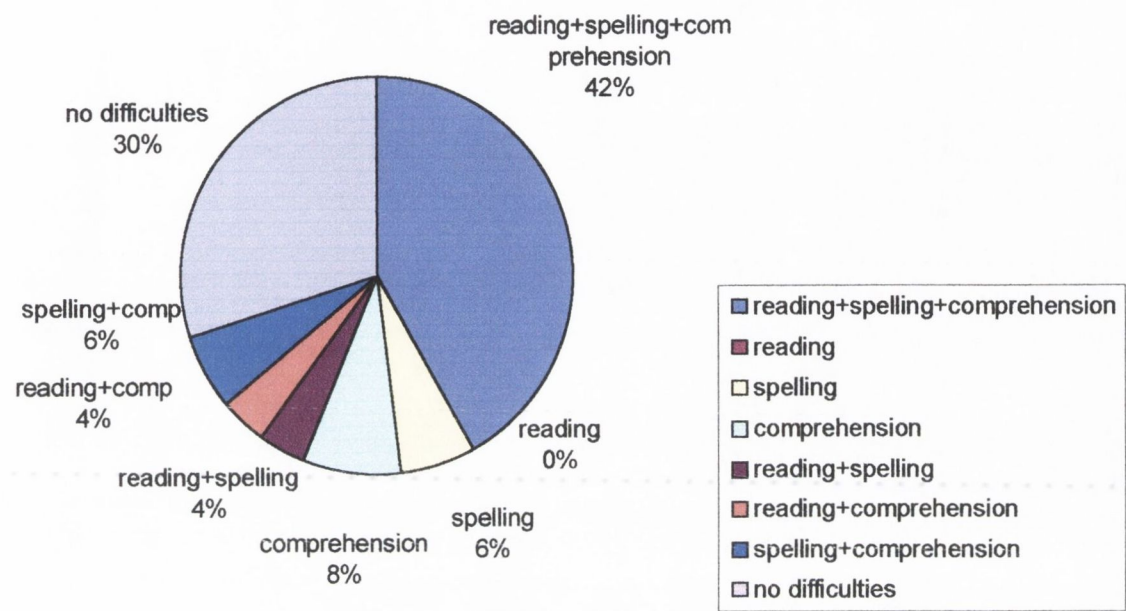
3.2: Identification of Specific Reading, Spelling or Comprehension Difficulties

Children defined as poor readers when their reading ability is one or more standard deviations below the norm, i.e., a W.O.R.D. Basic Reading Standard Score of 85 or less. Similarly children are identified as poor spellers when their spelling ability is one or more standard deviations below the norm, i.e., a W.O.R.D. Standard spelling score of 85 or less and the same for reading comprehension. Figure 3 below shows the different categories of difficulties experienced by the children.

Table 2: Categories of Difficulties Experienced by Children	Number	Percentage
Reading and spelling and comprehension	21	42
Reading only	0	0
Spelling only	3	6
Comprehension only	4	8
Reading and spelling	2	4
Reading and comprehension	2	4
Spelling and comprehension	3	6
No difficulties	15	30

Based on this evidence, twenty-one boys (42%) of the sample were identified as having difficulties in all domains, reading, spelling and comprehension. None of the sample was identified as having reading difficulties alone, three were identified as having spelling difficulties alone (6%) and four boys were identified as having comprehension difficulties alone (8%). Two boys were found to be poor readers and spellers and two boys were also found to be poor at reading and comprehension (4% each). Three boys had difficulty with spelling and reading comprehension (6%) and fifteen boys showed no significant difficulties in any domain (30%). Fig 10 below shows a pie chart with a breakdown of the difficulties experienced by the children in percentages.

Figure 10: The Percentage of Specific Difficulties identified by the Word Sub-tests



3.3: Diagnosis of Dyslexics Based on Specific Sub-test Difficulties

A two-year discrepancy was used between the child’s predicted age and actual subtest age equivalent in order to diagnose specific literacy difficulties. Of the children who had difficulties, twenty-eight did not meet the criteria for diagnosis of dyslexia and twenty-one did. Of the twenty-one who met the criteria, two had specific reading and spelling difficulties, two had specific reading and comprehension difficulties and eighteen had specific reading, spelling and comprehension difficulties. All of these had a performance score at least two years below their chronological age on these sub-tests.

Of the twenty-eight who did not meet the criteria but still had some evidence of specific learning problems, three had spelling difficulties, four had comprehension difficulties, one had a reading and comprehension difficulty, three had spelling and comprehension difficulties and two had reading, spelling and comprehension difficulties. These results are represented on Table 3 below.

Table 3: The Specific Sub-Test Difficulties of the Dyslexic and Non-Dyslexic Children

Specific Difficulty	Dyslexic	Non-Dyslexic
Reading difficulties	0	0
Spelling difficulties	0	3
Comprehension difficulties	0	4
Reading and spelling difficulties	2	0
Reading and comprehension difficulties	1	1
Spelling and comprehension difficulties	0	3
Reading and spelling and comprehension difficulties	18	2
No difficulties	0	15
Total in Each Group	21	28

3.4: Do higher reflex scores significantly correlate with a diagnosis of dyslexia?

To determine the relationship between dyslexia status and presence of reflexes, the researcher’s raw scores only were correlated using the point biserial coefficient, which is used where one variable is dichotomous, in this case, dyslexic or non-dyslexic, and the other measure is continuous. Because the categories dyslexic and non-dyslexic are not discrete categories as such but categories constructed such that people are assigned to if they are above or below a certain point, this is called an "artificial" dichotomous variable as opposed to a "true" dichotomous variable (such as male/female) (Howell, 1997). The artificial dichotomous variable has an underlying continuum. The Pearson Correlation Coefficient (which is really a point biserial correlation) is thus calculated initially and then adjusted to make it a biserial correlation (rb) using the formula below (See Howell, 1997). First, the results of the Pearson Correlation Coefficient are shown in Table 4 below.

Table 4: Statistical Relationship Between Diagnosis and Scoring on Reflex Tests.

Variables	Pearson Correlation Coefficient	Significance Level
Dyslexia vs. Right Schilder Score	-0.050	0.733
Dyslexia vs. Left Schilder Score	-0.054	0.712
Dyslexia vs. Right Ayres Score	0.390*	0.006
Dyslexia vs. Left Ayres Score	0.347**	0.015
Dyslexia vs. Up TLR Score	0.209	0.149
Dyslexia vs. Down TLR Score	0.072	0.625

*Indicates Correlation is significant at the .01 level (2 tailed).

**Indicates Correlation is significant at the .05 level (2 tailed).

The Adjusted Biserial Correlation (Rb) is calculated by the formula:

$$R_b = \frac{R_{pb} / \sqrt{p_1 p_2}}{y}$$

where Rpb = Pearson r,
P1 = the proportion who fall in the dyslexic category
P2 = the proportion who fall in the non-dyslexic category
y = the ordinate of the normal distribution at the point of the
distribution cutting off P1 percent of the area on one side and
P2 percent at the other side.

The adjusted correlation coefficient is represented in Table 5 below.

Table 5: Biserial Correlation between Diagnosis and Reflex Scores.

Test	Pearson	Biserial Correlation
Dyslexia vs. Right Schilder Score	-0.050	-0.063
Dyslexia vs. Left Schilder Score	-0.054	-0.068
Dyslexia vs. Right Ayres Score	0.390	0.492**
Dyslexia vs. Left Ayres Score	0.347	0.438*
Dyslexia vs. TLR Up Score	0.209	0.264
Dyslexia vs. TLR Down Score	0.072	0.091

- ** Indicates significant at the 0.001 level
- * Indicates significant at the 0.01 level

Thus, as can be seen from Table 5 above, only the Ayer’s test shows a relationship between reflex scores and dyslexia status. The Ayer’s right test correlates significantly with dyslexia status (Rb = 0.492, p = 0.001) as does the Ayer’s left test (Rb = 0.438, p = 0.01). Neither the Schilder nor TLR tests significantly correlate with dyslexia status.

3.5: Do any of the reflex tests show significant differences between the groups and is there a difference between the sides to which the head is turned?

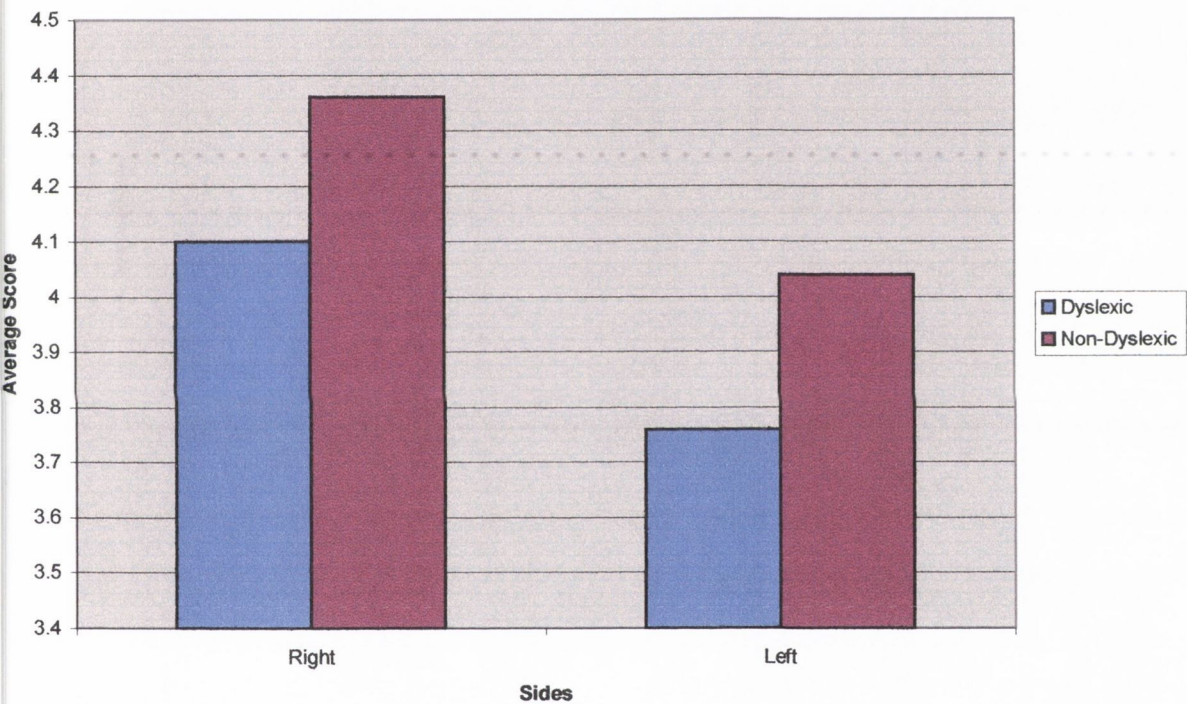
A between subjects 2 x 2 factorial analysis of variance with repeated measures on the second factor was run to determine whether there were any significant differences between the groups on any of the reflex tests and whether the side to which the head is turned makes any difference to the scores. Preliminary plots were also drawn in order to see if there was a difference or interaction between diagnosis and the side to which the head is turned. These plots are represented below in Figures 10 – 12 and means for each group by condition are presented in Table 6.

Table 6: A Comparison of Dyslexic and Non-Dyslexic Reflex Scores.

	N	Minimum	Maximum	Mean	Std. Dev.
Non-Dyslexic Schilder Right	28	1	12	4.36	2.71
Dyslexic Schilder Right	21	0	9	4.10	2.55
Non-Dyslexic Schilder Left	28	0	11	4.04	2.67
Dyslexic Schilder Left	21	0	9	3.76	2.39
Non-Dyslexic Ayres Right	28	0	12	3.43	2.63
Dyslexic Ayres Right	21	3	11	5.48	2.16
Non-Dyslexic Ayres Left	28	0	12	4.36	2.93
Dyslexic Ayres Left	21	3	12	6.43	2.68
Non-Dyslexic TLR Up	28	0	5	2.29	1.56
Dyslexic TLR Up	21	0	6	3.00	1.84
Non-Dyslexic TLR Down	28	0	9	2.68	2.02
Dyslexic TLR Down	21	0	6	2.95	1.80

As can be seen from Table 6, the average Schilder right score of 4.36 (SD = 2.71, range = 1 – 12) is higher for non-dyslexics than dyslexics, who scored 4.10 (SD = 2.55, range = 0 – 9). The average Schilder Left score is also higher for non-dyslexics at 4.04 (SD = 2.67, range 0 – 11) than for dyslexics, who score 3.76 (SD = 2.39, range 0 – 9). This is represented graphically in Figure 11 below.

Figure 11: Average Scores of Dyslexics and Non-Dyslexics on the Schilder tests with a Comparison of Sides

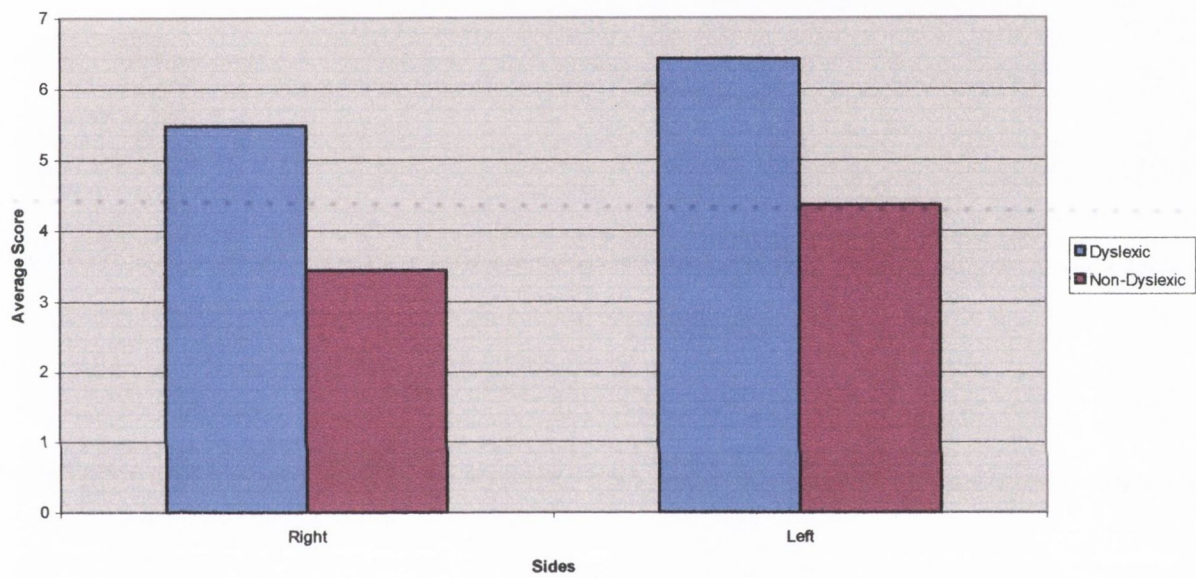


Anova results for the dyslexic and non-dyslexic groups on the Schilder test showed that the Schilder test did not discriminate significantly between dyslexics and non-dyslexics ($F(1, 47) = 0.133, p = 0.717$). Factorial analysis of variance of the Schilder test did reveal a significant main effect for the side to which the head is turned ($F(1, 47) = 4.414, p = 0.041$), with the effect for right turning greater than for left, but no interaction effect ($F(1, 47) = 0.001, p = 0.970$).

The average Ayres right score, in contrast, is higher for dyslexics at 5.48 (SD = 2.16, range = 3 – 11) than for non-dyslexics, who scored 4.36 (SD = 2.63, range 0 – 12). Similarly, the average Ayres left score is higher for dyslexics at 6.43 as opposed to the non-dyslexic average of 4.36.

This is represented in Figure 12 below.

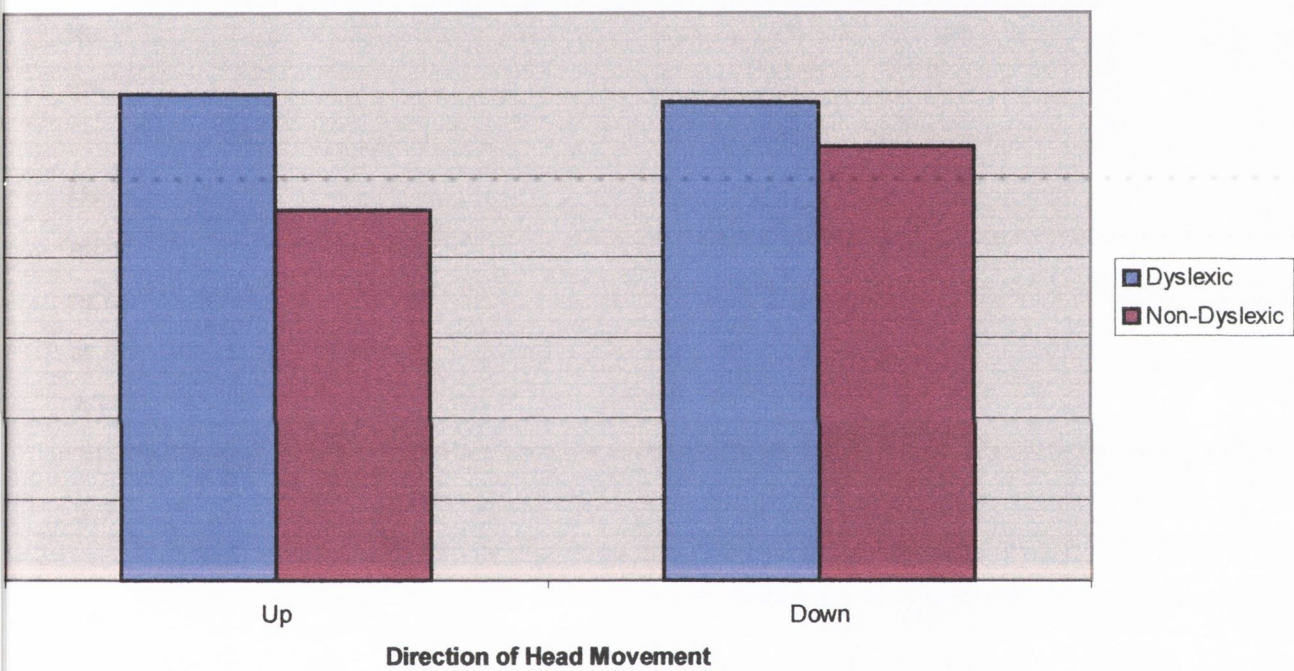
Figure 12: Average Scores of Dyslexics and Non-Dyslexics on the Ayres Tests with a Comparison of Sides



Anova results for the dyslexic and non-dyslexic groups on the Ayres test revealed a significant difference between groups ($F(1,47) = 8.332, p = 0.006$). Factorial analysis of variance of the Ayres test also showed a significant main effect for the side to which the head is turned ($F(1, 47) = 12.256, p = 0.001$), with the effect for left turning greater than for right, but no interaction effect ($F(1, 47) = 0.002, p = 0.965$).

The average TLR up score is higher for dyslexics at 3.00 (SD = 1.84, range 0 – 6) than non-dyslexics, who scored 2.29 (SD = 1.56, range = 0 – 5). And, finally, the average TLR down score is higher for dyslexics at 2.95 (SD 1.80, range 0 – 6) than for non-dyslexics, who score 2.68. This is represented in Figure 13 below.

Figure 13: Average Scores of Dyslexics and Non-Dyslexics on the TLR tests with a Comparison of Head Movement Direction



Anova results for the dyslexic and non-dyslexic groups on the TLR test revealed that the TLR test does not significantly discriminate between groups ($F(1, 47) = 1.063, p = 0.308$). Factorial analysis of variance of the TLR test also showed no significant main effect for the side to which the head is turned ($F(1, 47) = 0.675, p = 0.416$) and no interaction effect ($F(1, 47) = 1.098, p = 0.300$).

3.6: Do the Schilder and Ayres tests of the ATNR correlate?

Given that factorial analysis of variance revealed that the Schilder test did not significantly discriminate between dyslexics and non-dyslexics ($F(1, 47) = 0.133, p = 0.717$) and the Ayer's test did significantly discriminate between them ($F(1, 47) = 8.332, p = 0.006$), it is questionable whether both tests are measuring what they profess to measure, i.e., the ATNR. A correlation was run between the total Schilder score (right plus left scores) and the total Ayer's score (right plus left scores). The results were not significant (Spearman's $Rho = -0.098, p = 0.503$). The reflex measures that supposedly measure the same reflex measure, the ATNR, do not correlate significantly with one another and so questions arise as to their reliability and validity.

The Ayer's test discriminated between the groups and the Schilder did not. However, these results must be considered in light of the previous results of inter-rater reliability that showed that the Schilder is a more reliable measure than the Ayer's test for rating degree of persistence of the ATNR. Thus, whereas the Ayer's test is more effective at for discriminating between groups, the Schilder test is a more reliable measure of the ATNR reflex.

3.7: Do any of the children not score at all on measures of persistent reflexes?

The data were analyzed in order to ascertain if any of the children had scored zero on any of the reflex measures. The number of children in the dyslexic and non-dyslexic groups who scored zero on the various reflex tests is presented in Table 7 below.

Table 7: A Comparison of Zero Scores on Reflex Tests According to Diagnosis

Test	Number of Dyslexics who Scored Zero	Number of Non-Dyslexics who Scored Zero
Schilder right	1	0
Schilder left	2	1
Ayres right	0	3
Ayres left	0	3
TLR up	2	5
TLR down	3	4

Four of the children out of the twenty-one dyslexic children (19.05%) scored zero on one or more of the reflex tests. One child scored zero on three of the tests, two children scored zero on two of the tests, and one child scored zero on one of the tests. Looking separately at right/left and up/down scores, as appropriate, one child scored zero on the Schilder right test, two children scored zero on the Schilder left test, two children scored zero on the TLR up test and three children scored zero on the TLR down test. However, none of the dyslexic children scored zero on either of the Ayer's Tests.

In comparison ten of the non-dyslexic children out of twenty-eight (35.71 %) in this study scored zero on one or more reflex measures. Two children scored zero on three of the tests. Six children scored zero on two of the reflex tests and two children scored zero on one of the reflex tests. One child scored zero on the Schilder left, three children scored zero on the Ayres right, three children scored zero on the Ayres left, five children scored zero on the TLR up and four scored zero on the TLR down. However, none scored zero on the Schilder right test. Interestingly, two thirds of the non-dyslexic group scored on one or more of the reflex tests, which should not be possible according to the McPhillips theory of persistent reflexes.

There are two very interesting observations to be made here. The first is that the entire non-dyslexic group scored on one or more of the reflex tests. The second is that some of the dyslexic children failed to score on the Schilder and TLR tests. It may be recalled that McPhillips, who used the Schilder test, was unable to find dyslexic children who did not show retention of the ATNR. On the other hand, none of the dyslexic children scored zero on the Ayer's test, the only test found to reliably discriminate between dyslexic and non-dyslexic groups.

To analyze these results further, and to compensate to an extent for the demonstrated poor inter-rater reliability, ratings were categorised again using either zero or one as evidence of an absent reflex (to allow for a margin of error). The results are shown in Table 8 below.

Table 8: A Comparison of Low Scores (0,1) on Reflex Tests According to Diagnosis

Test	Number of Dyslexics who Scored Zero or One	Number of Non-Dyslexics who Scored Zero or One
Schilder right	3	3
Schilder left	3	3
Ayres right	0	4
Ayres left	0	4
TLR up	5	9
TLR down	5	9

This less stringent but perhaps more reliable analysis of scoring revealed that nine of the children out of twenty-one dyslexic children (42.86%) scored zero or one on one or more of the reflex tests. Four children scored zero or one on one test, three scored zero or one on two of the reflex tests and two scored zero or one on three of the reflex tests. Three dyslexic children scored one or zero on the Schilder right and three dyslexic children scored one or zero on the Schilder left test. Five dyslexic children scored one or zero on the TLR up and five scored one or zero on the TLR down test. None of the dyslexic children scored zero or one on either of the Ayer's tests.

This method of scoring revealed that ten of the children out of the twenty-eight non-dyslexic children (35.71%) scored zero or one on one or more of the reflex tests. Seven non-dyslexic children scored zero or one on one reflex test and seven non-dyslexic children scored zero or one on two of the reflex tests. One non-dyslexic child scored zero or one on one of the reflex tests and one scored zero or one on all six reflex tests. Three non-dyslexic children scored zero or one on the Schilder right and three non-dyslexic children scored zero or one on the Schilder left. Four of the non-dyslexic children scored zero or one on the Ayres right and three of the non-dyslexic children scored zero or one on the Ayres left test. Nine of the non-dyslexic children scored zero or one on the TLR up test and nine the non-dyslexic children scored zero or one on the TLR down test.

Thus, using less stringent criteria for the absence of a persistent reflex, some dyslexic children still failed to score on the Schilder and TLR test, but none of the dyslexic children scored zero on the Ayer's test, the only test found to reliably

discriminate between the groups. Furthermore, based on this criterion, there still remains convincing evidence for the persistence of reflexes in the non-dyslexic group.

3.8 Is there a correlation between the presence of the TLR and the ATNR?

The presence of the TLR and the presence of the ATNR, as measured by the more reliable discriminatory test, the Ayres test, were also correlated to investigate whether it is the case, as the theory of the evolution of postural reflexes suggests, that a retained TLR cannot exist without a retained ATNR as the TLR must be integrated before the ATNR is integrated (Capute et al., 1978). The data was analyzed in order to see in which patterns children displayed retained or absent ATNR and TLR reflex movements. The results of this analysis are shown below in Table 9.

Table 9: The Relationship Between the Presence of the ATNR and TLR

	No TLR	TLR
No ATNR	0	1
ATNR	6	43

As can be seen from Table 9, none of the children showed an absence of both the TLR and ATNR, and forty-three children had an ATNR and TLR both of which is possible according to the theory of emergence of postural reflexes. Six children had an ATNR with no TLR, indicating a fully integrated TLR but a retained ATNR, supporting the theory of emergence of postural reflexes. However, one child in the sample scored zero on the ATNR and yet scored on the TLR test, indicating a fully integrated ATNR but a retained TLR. This should not be possible according to the theory of the developmental emergence of these reflexes. All of these results are discussed in the following section.

4. Discussion

4.1 An analysis of inter-rater reliability for scoring reflexes

When analysing correlations, both the strength and significance of the correlation must be taken into account (Coolican, 1999). A correlation of 0.8 is considered to be strongly correlated and so scores on the Schilder right and left and TLR down are strongly correlated. A score of 0.6 or 0.5 are said to be moderately significant which would refer to the Ayer's right and left and TLR up tests. A look at the scatter plots reveals a strong correlation indicated by the linear graph for the Schilder right and left and the TLR down. The other graphs representing the Ayres right and left and the TLR up reveal only a random pattern and moderate to weak correlation between rater's observations.

The statistical significance of these results must also be considered. All of the inter-rater observations were significant at the 0.01 level and so there can be little confidence in the reliability of raters' scores (Coolican, 1999). An analysis of the individual ratings reveal poor coefficients of determination which calculates the amount of variance explained by the rater's scores by squaring r and multiplying by 100. A correlation of 0.806 on the Schilder right test explains 65% of the variance and 0.84 on the Schilder left test explains 70% of the variance which is quite a high proportion of the variance. In contrast, the Ayer's right correlation of 0.526 explains only 28% of the variance and the Ayer's left score of 0.58 explains only 34% of the variance, which is quite low. Finally, the correlation of 0.631 on the TLR up test explains 40% of the variance, which is moderate, and a correlation of 0.807 on the TLR down test explains 65% of the variance, which is quite high. Thus, the Schilder test appears to be the most reliable test with which to measure the ATNR and the Ayres is the least reliable. The TLR test is moderately reliable and moving the head down appears to be a more reliable method of detecting a TLR response than moving the head up.

However, in observational studies, data can only be said to be objective when two observers agree on results at a level of ninety-percent agreement according to Fassnacht (1982) and these correlations must be statistically significant. The observations were statistically significant but with the highest agreement between raters at 70% (Schilder right) so the tests cannot be considered highly reliable.

There are a number of possible reasons for a lack of complete agreement between raters. Firstly, the Schilder Test for the ATNR specifies that a score of 1 represents a slight movement of the arms, up to 20° , to the same side as the head is turned and a score of 2 represents a movement of the arms between 21° and 45° as the head is turned and a score of 3 represents a head movement of more than 45° (taken from McPhillips, 2000). The visual difference between 20° and 21° is impossible to discern subjectively. It is also possible to score a 1 if there is a slight dropping of the arms, a 2 if there is a marked dropping of the arms and a 3 if there is swaying and loss of balance. Thus, both the horizontal and vertical planes of movement must be taken into account when scoring which makes scoring all the more complex. In addition, there is no definition of “slight” and “marked” dropping of the arms. This evaluation is left up to the observer’s subjective judgement. In order to score this movement accurately, degrees might perhaps be marked on a nearby wall and the floor, and a reading taken from both in order to make this reading more accurate, although there might be difficulty with parallax error. Also, it would be quite impossible for one rater to score this alone and a second rater would be needed at the time, and the degree of movement agreed objectively on the wall and floor by one while the other observer turns the head. Despite these difficulties, there was still quite good agreement between raters using the video recordings.

Secondly, the Ayer’s II test for the ATNR specifies a score of 1 for slight muscle tone changes, a 2 for slight visible elbow flexion of less than 30° , a 3 for elbow flexion between 30° and 60° with little shoulder depression and a 4 for flexion more than 60° (taken from Parmenter, 1975). Thus, if an elbow flexes 30° , a score of either 2 or 3 could be scored. Also, it is impossible to score while doing the movement so a video camera must be used. However, with this complex reflex where the back, elbows, shoulders and legs must be observed, the camera can only see one side. The camera cannot be positioned head on, as the examiner, kneeling in front, would block the view of the child, so two cameras would be preferable. Also, some way of objectively measuring the degree of flexion of the elbow would be useful. The fact that only one perspective of the child’s position was filmed possibly accounts for the lower inter-rater reliability between scorers.

Thirdly, for the TLR test it is difficult to ascertain a ligament movement from the resolution of a video camera at the time of administration. Consequently, discriminating

between scores of 0 and 1 (i.e. between no movement and movement of the ligaments of the feet and lower leg in the absence of foot movement) is highly negligible and subjective. It would be preferable to score the TLR at the time of administration and to use two raters because of the difficulty in perceiving these slight movements.

According to the literature on scoring methods, there is a huge difference between different authors in the procedures used. To take the Ayres test for ATNR, for example, two publications from the INPP differ in their scoring methods. Goddard (1996) specifies scoring 1 if there is a slight bending of the opposite arm but Field and Blythe (1989) specify scoring 1 if there is visible muscle movement but no movement in the elbows. A score of 2 is given by Goddard for a definite bending of the opposite arm but Field and Blythe specify only a slight bending of the occipital elbow. There is a need for a standardization of these procedures and a more objective measure of measurement.

4.2 Do higher reflex scores significantly correlate with a diagnosis of dyslexia?

Children were diagnosed as dyslexic or not based on a two-year discrepancy method between the child's predicted age and age equivalent based on literacy scores. Specific reading, spelling and comprehension difficulties were identified (see Table 2 and Figure 12). Twenty-one were diagnosed as dyslexic, the remaining twenty-eight as non-dyslexic (See Table 3). Having made this distinction between groups, the diagnosis was correlated with the first rater's reflex measurements using a biserial correlation.

The Schilder right and Schilder left biserial correlations were just -0.063 and -0.068 respectively and were obviously not statistically significant. Furthermore, this inverse relationship means that a high score on the Schilder test is more likely if the child is not dyslexic rather than dyslexic. This finding contradicts the findings of the McPhillips study, which stated that the ATNR, as measured by the Schilder test, was an invariant characteristic of his dyslexic sample. Furthermore, McPhillips assumed no evidence of the ATNR in non-dyslexics. Their conclusion that the ATNR is only present in the dyslexia population is refuted by this study, as is their observation that no dyslexics without persistent ATNR could be found.

On the other hand, the correlation between scores for the Ayer's right and dyslexic status was found to be significant at 0.492 and for the Ayer's left was found to be

significant at 0.438. This supports the results found by Parmenter (1975) that the ATNR, as measured by the Ayres test correlates with a diagnosis of dyslexia. The TLR up and TLR down correlations were 0.264 and 0.091 respectively and were not statistically significant, contradicting the work of Johnson (1973) who linked a persistent TLR with poor reading performance. The biserial correlations reveal that only the Ayer's test scores are related to dyslexia status.

4.3 Do any of the tests show significant differences between the groups on reflex scores and is there a difference between the sides to which the head is turned?

Figure 11 shows the average scores for each of the groups on the Schilder test and shows that the non-dyslexic group score higher on Schilder right (mean = 4.36) than dyslexics (mean = 4.10) and also on Schilder left (non-dyslexic mean = 4.04; dyslexic mean 3.76). These means indicate that non-dyslexics are more likely to score higher on the Schilder test for the ATNR than dyslexics, which contradicts the theory based on the notion that persistent reflexes are higher in dyslexics. Factorial analysis of variance of the Schilder test showed that the Schilder test did not reliably discriminate between groups ($F(1, 47) = 0.133, p = 0.717$). Therefore, the most reliable test used in this study, according to the inter-rater correlated scores, fails to discriminate between dyslexic and non-dyslexic groups, which refutes conclusions of the McPhilip's study.

Figure 12 shows the average scores for each of the groups on the Ayer's test and shows that the dyslexic group score higher on Ayer's right (mean = 5.48) than non-dyslexics (mean = 3.43) and also on Ayer's left (dyslexic mean = 6.43; non-dyslexic mean = 4.36). Thus, the dyslexics are more likely than non-dyslexics to score higher on retained reflex scores as measured by the Ayres test, supporting the theory of persistent ATNR being higher in dyslexic populations. The Ayres II test was found to significantly discriminate between dyslexic and non-dyslexic groups ($F(1,47) = 8.332, p = 0.006$). Paradoxically then, the more unreliable measure of the ATNR as assessed by inter-rater reliability, was found to be better at discriminating between dyslexics and non-dyslexics and so is consistent with Parmenter's finding that the ATNR, as measured by the Ayres II test, discriminates between dyslexic and non-dyslexic groups (Parmenter, 1975).

Figure 13 shows the average scores for each of the groups on the TLR test and shows that the dyslexic group score higher on the TLR up (mean = 3.00) than non-dyslexics (mean = 2.29) and also on TLR down (dyslexic mean = 2.95; non-dyslexic mean = 2.68). However, these differences were not statistically reliable ($F(1, 47) = 1.063, p = 0.308$). Thus, Johnson's (1973) finding that the TLR was related to reading achievement is not confirmed here. However, given the unreliability of the testing method used, this result must be treated with considerable caution.

The Ayer's test found an effect for the side to which the head is turned, supporting the work of Parmenter (1975) who found, using the Ayer's test, that the right side showed a larger response than the left ($F = 20.53, p = 0.001$) but does not support her findings that this side difference significantly distinguished dyslexics from non-dyslexics. None of the tests, including the Ayres test, found an interaction effect. The Schilder test also found an effect for the side to which the head was turned ($F = 4.414, p = 0.041$), but no interaction effect ($F = 0.001, p = 0.970$) and the TLR test found neither an effect for the side to which the head was turned ($F = 0.675, p = 0.416$) nor an interaction effect ($F = 1.098, p = 0.300$).

In summary, only the Ayres test was found to significantly discriminate between dyslexics and non-dyslexics and to correlate moderately with dyslexia. However, the ability of the Ayres test to discriminate between groups is called into question, as the Ayres test was also the most unreliable of the measures used. Of the ATNR measures used, only the Schilder is reliable. However, this test demonstrably fails to discriminate between dyslexics and non-dyslexics, undermining McPhillips theoretical position. The TLR down is reliable but fails to show any relationship with dyslexia status. These results do not conclusively support the ability of these tests to discriminate between groups.

In addition, preliminary studies from the Trinity Dyslexia Research Group have not been supportive of the theory of persistent reflexes as a causal theory of dyslexia. In independent studies this year, no correlation has been found between persistent reflexes, including the ATNR, STNR and TLR, and a diagnosis of dyslexia (Allman, 2002; Orr, 2002). Orr found that although primitive reflexes were present in all of the eight to ten year old children she assessed, they could not discriminate between dyslexic and non-dyslexic groups (Orr, 2002). This research project highlights the need for more objective

and reliable procedures before any conclusions can be made about the causal role for reflexes in dyslexia.

4.4 A comparison of tests for the ATNR

The Ayer's test is the only test that seems to discriminate dyslexic children from non-dyslexic children. The Ayres and Schilder test that both profess to measure the same reflex, the ATNR, do not correlate well (Spearman's $Rho = -0.098$, $p = 0.503$). It may be the case that the Schilder test is not as good at eliciting the ATNR because it is easier to resist the reflex movement (Franklin, 2000, personal communication). In the Ayer's II quadruped position with slightly abducted elbows, the child cannot lock his or her arms, making it more difficult to resist the influence of the ATNR. With the Schilder test, however, there is no way to prevent locking of the arms or stiffening of the body to prevent the movement. Thus, although the Schilder test generated a more reliable measure, it is perhaps less valid than the Ayres II as a procedure for testing the presence of the ATNR. However, without standardized and accurate measurement of these reflex movements it cannot be established for certain which test is the more reliable and valid.

4.5 Do any of the dyslexic children not score at all on measures of persistent reflexes and do non-dyslexic children show evidence of persistent reflexes?

Given that the analysis of variance revealed that only the Ayres II test satisfactorily discriminated between dyslexics and non-dyslexics, the next question that might be asked is whether any dyslexic children scored zero on the reflex tests and indeed whether non-dyslexic children showed evidence of retained reflexes. Four of the dyslexic children out of twenty-one (19.05 %) in this study scored zero on one or more of the reflex measures. These results do not confirm the evidence of the McPhilip's study, which reported that no dyslexics could be found who did not score on the Schilder measure of the ATNR (McPhillips, 2000). Interestingly, however, none of the dyslexic children scored zero on the Ayer's Test, the only test found to reliably discriminate between dyslexic and non-dyslexic groups, supporting the Parmenter study which used the Ayres test to discriminate between groups. However, as this test was also the most unreliable of the measures used, its use as a diagnostic tool cannot be considered conclusive.

Eighteen of the non-dyslexic children out of twenty-eight (64.29%) also had retained reflexes and so it is not the case that only dyslexic children have these reflexes. These results are consistent with studies that show that persistent reflexes are present in the non-dyslexic population from babies in the womb (Minkowski, 1921), to schoolchildren (Parmenter, 1975), to normal adults (Hellebrandt, Schade and Cairns, 1962).

4.6 Is there a correlation between the presence of the TLR and the ATNR?

Analysis of the data revealed that one child did indeed score zero on the ATNR and yet scored on the TLR test, indicating a fully integrated ATNR but a retained TLR. According to the theory of the evolution of postural reflexes (e.g., Capute et al., 1978, See Figure 2) it should not be possible to have a retained TLR without a retained ATNR because the TLR has to be integrated before the ATNR can be integrated. Thus, if the TLR is present and the ATNR is absent, this calls into question the order of emergence of the different reflexes and the effect they have on one another. These results suggest that the TLR and ATNR are independent and do not affect one another in the way that the theory of persistent primitive reflexes suggest.

4.7 Implications of the findings of this study for the theory of persistent reflexes as a primary cause of dyslexia

To summarize, this research study found that the Schilder test of the ATNR, despite being the most reliable measure according to inter-rater reliability, failed to discriminate between dyslexics and non-dyslexics and, in fact, showed a very weak but inverse relationship between dyslexia and presence of a retained ATNR. In contrast, the Ayer's test, despite being the most unreliable measure according to inter-rater reliability, showed a positive relationship between a diagnosis of dyslexia and higher scores of a persistent ATNR and this relationship was found to significantly discriminate between the two groups. Given these differences it cannot be assumed that these tests, as used in this study, are even measuring the same phenomenon. Thus, there can be less confidence in the McPhillips study, which found that the ATNR is linked to dyslexic status on the basis of Schilder results. On the other hand, there can be more confidence in the Parmenter

study, which used the Ayres test to discriminate between the groups. These results are consistent with Parmenter's suggestion that the ATNR may be present in all children, but to a greater degree in dyslexic children. The TLR test also failed to discriminate between groups. Clearly, the relationship between dyslexic status and persistence of retained reflexes is not as clear-cut as some have argued.

In addition, the presence of a retained TLR in the absence of the ATNR in one child undermines the conclusion that the TLR must be integrated before the ATNR (e.g., Illingworth, 1987). Nevertheless, it could be that the methodology used here to measure the ATNR may not have been sensitive enough to detect the reflex, if it was present, and so this suggestion remains tentative.

4.8 Suggestions for future research

4.8a The standardization of methodology

Empirical research in this field is needed in part because of the rapidly expanding availability of neurodevelopmental therapy based on the theory of retained reflexes as an intervention for learning difficulties. However, before progress can be made in diagnosis and research, standard and reliable procedures for the measurement of retained reflexes need to be developed. These procedures need to consider scoring criteria, methods for determining rater reliability and the prevention of reflex inhibition by the person observed. It might be possible to make judgement of the presence of persistent reflexes more objective by using computerised analysis of video recordings made under well-controlled conditions. The Trinity Dyslexia Group in conjunction with the department of electronic engineering is currently attempting to design such a programme. As an example of where basic procedures might be improved in determining the presence of the TLR, Orr (2002) stated that there is no standardized width the feet must be apart in the standard TLR test. The child is simply asked to stand comfortably. The child whose feet are closer together, therefore, has more chance of falling over (as weight is not distributed as widely) than a child who stands legs apart. She suggests having a block of wood to place between the child's feet in order to keep the same distance apart for each child. Methods of standardization such as that suggested by Orr would strengthen the research around persistent reflexes.

4.8b The use of modern technology

Fetal studies such as Gesell (1989) have found that when the foetus rehearses reflex movements in the womb, there is an increase in the growth of the neurons of the brain and this supports overall central nervous system development. Measures would clearly be useful to assess what changes are taking place in the older child or adult brain during the rehearsal of these movements. Paulesin, Frith, Snowling, Gallagher, Morton, Frackowiak and Frith (1996) showed that dyslexics, in contrast to controls, did not display the usual concerted activation of four brain areas in language functioning; Broca's area, Wernicke's area and the insula and inferior parietal lobule, suggesting that dyslexia results from a disconnection between language systems. Eden, Van Meter, Rumsey, Maisog, Woods and Zeffiro (1996) showed that the disconnection between language systems was also correlated with dysfunctions in other parts of the dyslexic brain, for example areas of the brain involved in visual motion (cited in Hulme and Snowling, 1997). Thus, any improvement in dyslexics' performance should show some increase in concerted activation of previously disconnected language systems in the left hemisphere and a resultant improvement in function of other areas of the brain. Use of fMRI equipment would be very useful to determine any change in brain function in children who have undertaken the movement programmes suggested by the INPP or McPhillips to see if any neurological changes do take place. Future research should attempt to account for why this movement integration therapy works by using imaging technology to establish which areas of the brain are activated by the movement programme and any changes the rehearsal and repetition of these movements make to the activation of the brain and the connection of different brain areas.

fMRI could also be useful in investigating structural differences in dyslexic and non-dyslexic brains in order to explain the results of research into the effects of persistent reflexes in dyslexic children. fMRI could also be used to determine whether they are a feature of a wider cerebellar abnormality. Recently, an MRI study found that cerebellar structures in dyslexic brains are more symmetric than non-dyslexics and that the degree of cerebellar symmetry was correlated with the severity of dyslexics' phonological difficulties (Rae et al., 2002). Furthermore, the right cerebellar hemisphere of dyslexics

has been shown to display a functional deficit, in response to both learned and novel motor tasks (Nicolson et al., 1999). fMRI studies can be complimented by studies of biochemical differences in the cerebellum also conducted by the team who found a reduced ratio of choline-containing compounds to N-acetylaspartate in the right cerebellar hemisphere of dyslexics. These differences in functionality, biochemistry and symmetry between the hemispheres may account for the proposed difference in the persistence of reflexes in some dyslexic children. Nicolson et al. (2001) propose that dyslexia is associated with cerebellar impairment on all the classic clinical tests of cerebellar function, including postural stability and muscle tone in about 80% of cases, but not in every dyslexic person tested. Therefore, it appears likely that persistent reflexes are another symptom of a cerebellar abnormality that underlies dyslexia and is a descriptive, rather than explanatory theory of dyslexia. Further evidence is needed to establish the nature and prevalence of cerebellar dysfunction in dyslexic and non-dyslexic participants.

Furthermore, due to the wider range of dysfunction displayed by dyslexic participants which are not directly under the control of the cerebellum, including phonological, neuro-imaging studies should look at not only damage to the cerebellum, but damage to the projections from the cerebellum to other regions, such as different cerebrocortical and subcortical structures concerned with linguistic processes (Moretti et al., 2002). In particular, fMRI could be used to look at the relationship between the cerebellar and vestibular dysfunction, which Levinson (1998) proposed accounted for over 94% of the abnormal neurological or ENG parameters in dyslexic and learning disabled participants. Both vestibular nuclei and the cerebellum regulate muscle tonus through an interconnected system (Krklijes, 1990) and so any investigation of the role of persistent reflex movements in dyslexics or non-dyslexics should take these structures into account.

4.8c Scientific investigation of the evolution of reflexes

This research has shown, albeit tentatively, that it may not necessarily be the case that the TLR has to be inhibited before the ATNR can be inhibited and perhaps it is more probable that a parallel, rather than serial development characterizes the evolution of postural reflexes. One child, in fact, has a retained ATNR but no TLR, undermining the theory that the TLR must be inhibited before the ATNR can be inhibited. Thus, although

part of an interconnected system, perhaps each reflex emerges and diminishes along its own developmental path rather than being under the influence of other reflexes. This theory, however, is highly speculative and needs scientific research.

4.8d Scientific comparisons with other causal theories of dyslexia

It may be useful to consider retained reflexes from a different perspective, such as that provided by the theory of automatisisation deficit and cerebellar dysfunction (Nicholson and Fawcett, 1990; Nicholson, Fawcett and Dean, 1994). The retained reflexes studied here are involved in postural adjustment in the maintenance of balance and their retention may be associated with underdeveloped balance mechanisms. As Nicholson and Fawcett (1994) found, dyslexic children have difficulty with fine motor movements such as threading beads, hand-eye co-ordination and balancing on one foot, especially if blindfolded, i.e., in the absence of continuous visual feedback (and hence, would have implicated the vestibular system). All of the primitive reflex tests are carried out with the eyes closed and so are similar to Nicholson and Fawcett's balance tests and Levinson's tests cerebellar-vestibular dysfunction. Levinson found that over 94% of a dyslexic sample showed cerebellar-vestibular dysfunction and Byl et al., 1989 found that the boys who took part in a vestibular exercise programme (as opposed to an aerobic exercise programme) made gains in accuracy and speed of spatial perceptual processing. These results suggest that it is stimulation of the vestibular system that may improve performance. Thus, it may not be the case that the exercise programme used by McPhillips improves the symptoms of dyslexia by rehearsing and inhibiting persistent reflexes, but instead that the vestibular system is stimulated, improving balance. Indeed, all of the tests require movement in the absence of visual input, stimulating the vestibular system. It may be the case that the non-specific movements used by the control group in the McPhillips study did not show improvement because these exercises did not stimulate the vestibular system in the way in which the movements in the treatment group did. The findings of McPhillips et al., therefore, might better be interpreted within the framework of the theory of cerebellar-vestibular abnormality rather than the theory of persistent primitive reflexes. Whether the cerebellum is the direct or indirect cause of dyslexia, it appears to be the most likely candidate for a causal theory of dyslexia due to its

interconnections with many different functional areas of the brain, including areas for the processing of temporal, spatial, memory, balance and phonological information.

Dysfunction of the cerebellum or its connections to these areas of the brain could lead to the individual pattern of dysfunction which is seen in dyslexia.

4.8e Could there be a role for the theory of persistent reflexes within a framework of cerebellar dysfunction theory?

This study refutes the theoretical position proposed by McPhillips et al. (2000) by demonstrating that the Schilder test for the ATNR fails to discriminate between dyslexic and non-dyslexic children, some children with little evidence of retained reflexes develop dyslexia and many children with retained reflexes do not develop dyslexia. This study has also shown that the Ayres test for the ATNR and the test for the TLR are unreliable which undermines the work of the INPP who use these measures as diagnostic tools to discriminate between dyslexic and non-dyslexic children, despite the lack of non-dyslexic controls in their research. It is, therefore, entirely possible that, as Parmenter suggests, these reflexes are present in all children and are not exclusively a characteristic of dyslexic children. However, this question cannot be answered without more reliable, standardised measures of reflex movements and the inclusion of non-dyslexic control groups.

When compared with Nicolson and Fawcett's (1995) theory of cerebellar abnormality and Levinson's theory of cerebellar-vestibular dysfunction (Levinson, 1998), the theory of persistent primitive reflexes would appear to be an inferior candidate for a causal theory of dyslexia. The cerebellar model brings together, genetic, socio-cultural, magnocellular, phonological and automatisisation deficit theories together to provide an explanation for the wide range of dyslexic symptoms including literary difficulties, spatial and temporal difficulties, balance and co-ordination difficulties and neurological anomalies. A wide range of scientific research, which uses modern technology, such as fMRI (e.g. Georgiewa, 1999), neuroscientific investigations (Nicolson et al., 1996, cited in Nicolson, Fawcett and Dean, 2001) and behavioural trials (Nicolson et al., 1999) have been incorporated to support this theory. In contrast, the theory of persistent reflexes is, at present, a theoretical position with little standardisation of procedures or use of controlled scientific research to support it. The results of this study show that the theory of persistent

reflexes cannot be used as a diagnostic tool for dyslexia at present due to the unreliability of the procedures. However, the research into the role of persistent reflexes in dyslexia cannot be dismissed out of hand without further research and so its role within this framework must be considered.

Because of the unsound methodology of the persistent reflex literature, it is very difficult to speculate whether there is room for that theory within this framework. It may be the case that persistent reflexes are present in everyone to varying degrees (Parmenter, 1975) and that persistent reflexes have nothing whatsoever to do with dyslexic status. However, it may also be the case that a cerebellar deficit could lead to the re-emergence of aberrant reflexes, in which case, dyslexics would display these reflexes to a greater degree than the normal population, which McPhillips and the INPP argue. In this case, persistent reflexes may fit into the framework of the cerebellar theory as another secondary consequence of cerebellar abnormality as it has been shown that cerebellar abnormality can affect muscle tone, coordination (Fawcett, Nicolson and Dean, 1994) and posture (Gill-Body et al, 1997). Nicolson and his colleagues, having previously found that dyslexics had a dissociation of time estimation (impaired) and loudness estimation (unimpaired) similar to patients with cerebellar lesions (Nicolson, Fawcett and Dean, 1994) reported recently that dyslexia is associated with cerebellar impairment on all the classic clinical tests of cerebellar function, including postural stability and muscle tone in about 80% of cases (Nicolson et al., 2001). Thus, it may be the case that aberrant reflexes are present in some dyslexics but not in others. This research found that only 57.14% of the dyslexic children showed evidence of persistent reflexes on one or more of the reflex tests and that the remaining 42.86% of children showed no evidence of persistent reflex movements on one or more of the reflex tests. Thus, although this rate is lower than the 80% prevalence rate found by Nicolson and his colleagues, it supports their theory that not all dyslexics show behavioural manifestations of cerebellar dysfunction. However, 64.29% of the non-dyslexic group displayed persistent reflex movements on one or more reflex tests, supporting the work of Parmenter that these persistent reflexes are present in all children to some degree. However, Parmenter also stated that reflex movements manifested more strongly in the dyslexic population, using the Ayres method, and the

Ayres method was found to be the only test that discriminated between the groups and the only test on which dyslexics were always found to have evidence of a retained ATNR.

However, without applying rigorous scientific methodology to the tests of persistent reflexes this question cannot be answered for the present. It is most probable, however, that the theory of persistent reflexes could not provide an explanation of the symptoms of dyslexia as satisfactorily as the theory of cerebellar abnormality or a theory of cerebellar system abnormality.

5. Conclusion

This piece of research attempted to compare dyslexics with age-matched controls in order to ascertain whether retained reflexes could significantly distinguish one group from the other. The research refutes McPhilip's argument that persistent reflexes are present only in populations with specific learning difficulties. The results of this research suggest that these reflexes appear to be present in both the normal population and the dyslexic population and cannot be used to discriminate between the groups. The only test that did discriminate between the groups, the Ayres II test was unreliable. The ambiguous and unstandardized nature of the measurements, as illustrated by poor inter-rater reliability and the wide variations in the scoring method used, makes it difficult to compare these results with the other studies in the literature. If research in this area is to progress, standardization of reliable measurement procedures must be developed. The theory of the evolution of postural reflexes is called in to question and the theory of persistent reflexes, as an explanation for the multi-faceted symptoms of dyslexia, is not supported. It could be that persistent reflexes are just a symptom of a wider cerebellar system dysfunction and not a definitive explanation for the root cause of dyslexia.

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Appendices

Appendix A: Letter of Permission to Schools
Appendix B: Letter of Permission to Parents Regarding Educational Assessment
Appendix C: Letter of Permission to Parents Regarding Reflex Tests
Appendix D: Criteria and Scoring Sheets for Recording Reflexes

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Appendix A: Letter of Permission to Schools

February 18th, 2001.

Dear Principal,

You may recall that, some three years ago (i.e. March to May, 1998), your school took part in an educational research project which was conducted by myself and supervised by Dr. Ray Fuller, of Trinity College, Dublin. The purpose of the study was to determine the usefulness of the then newly developed Dyslexia Early Screening Test (Nicolson and Fawcett, 1996), on a population of Irish schoolchildren. The findings of that study, which were based on a sample of Irish schoolboys, suggested that the Dyslexia Early Screening Test (D.E.S.T.) is not a valid predictor of children who will later develop dyslexia.

Three years on, we wish to follow-up on the sample of schoolboys who were assessed in the Spring of 1998, for we are keen to discover how these schoolboys are now performing academically. In order to conduct this follow-up research project, I ask for your agreement to take part in the study and your help in locating the children concerned. Once identified, these schoolboys will be offered an educational assessment, which I would conduct in the Spring of 2001, having first secured parental consent.

Enclosed with this letter, you will find a complete list of the schoolboys which I wish to reassess, for the purpose of the follow-up study. If you agree to take part in the study, I would respectfully ask you to consult the list, enter the information required on each boy, and then mail the list to me, in the stamped, addressed envelope provided, by Monday, March 5th, 2001. I have also enclosed a sample of the parental consent form, for your consideration.

I look forward to your response.

Yours faithfully,

Dr. Gina MacDonnell, B.Sc., D. Clin.Psych,
Senior Clinical Psychologist.

Appendix B: Letter of Permission to Parents Regarding Educational Assessment

Dear Parent (s),

You may recall that our school took part in an educational research project, in the Spring of 1998, in which your son received an educational assessment. Three years on, our school has agreed to take part in a follow-up study, which offers an educational assessment to all the schoolboys who were assessed three years ago.

Each assessment will take approximately 1 hour 15 minutes and will be conducted during school hours, by a clinical psychologist. The assessment will provide an estimate of your child's intellectual ability (using the Wechsler Intelligence Scale for Children) and reading ability (using the Wechsler Objective Reading Dimensions). Following the assessments, the psychologist plans to make recommendations on each child and these will be share with the school. If the results from a particular child's assessment indicate that he needs extra help, the psychologist is prepared to meet with parents to discuss this.

If you are interested in your son receiving an educational assessment, please complete the permission form below and return it to the school.

Yours sincerely,

Principal.

------(Please Detach)-----
Permission Form

Name of the Child _____

Date of Birth _____ Teacher _____

I give permission for my son to receive an educational assessment _____

I would prefer if my son did not receive an educational assessment _____

(Please tick one)

I understand that the assessment results will be made available to the school.

Signature of parent _____

**Appendix C: Letter of Permission and Consent Form to Parents
Regarding Reflex Tests**

Title of the Study: The Role of Persistent Early Reflexes in Learning Disabilities

Researchers: Maria McEvoy, B.A. (Psychology) and
 Dr. Gina MacDonnell, Senior Clinical Psychologist

Information about the study:

Dear parent,

As part of an educational assessment of your child, we wish to conduct a three minute test of your child’s balance and reflexes. A new theory of learning proposes that children with learning problems have retained childhood reflexes that should have disappeared when the child became older to allow for further motor developments to take place. We wish to test this theory by assessing your child using these reflex tests and comparing these with how your child actually does on measures of reading, spelling and comprehension. This information will be most valuable in increasing the knowledge we have of learning difficulties and the most effective assessment methods to use to benefit the child.

If you have any queries regarding this study please contact Ms. Maria McEvoy at the Psychology Department of Trinity College, Dublin.

Thank you for taking the time to read this.

Yours sincerely,

Maria McEvoy, B.A. (Psychology)

Consent Form

Title of the Study: The Role of Persistent Early Reflexes in Learning Disabilities

Researchers: Maria McEvoy, B.A. (Psychology) and
Dr. Gina MacDonnell, Senior Clinical Psychologist

Dear parent,

Thank you for agreeing to allow your son to take part in our research project and educational assessment.

As part of this assessment we wish to conduct a three minute test of your child's physical reflexes. The psychologist will videotape your son completing three simple tests of balance.

If you wish to permit your son to take part in this phase of the assessment, please sign the consent form below and return to the school as soon as possible.

I do wish my child to take part _____

I do not wish my child to take part _____

(Please tick one)

Signature of the parent or guardian _____

Appendix D: Scoring Guidelines for Reflex Tests

The test for the **Tonic Labyrinthine Reflex** must be administered to the child without socks and shoes and with trousers raised slightly to the knee. The child stands with hands by his side and his eyes closed. The examiner moves the head up and back so that the back of the head approaches the back of the neck. The head is then moved down so that the chin rests on the chest.

Scoring:

0 – no response, **1** – ligament movement of the feet and lower leg, no feet movement, **2** – moves toes, **3** – loses balance, **4** – falls over.

The Schilder Test for the ATNR: The child is requested to upright with feet together and arms held straight out in front at shoulder level with wrists relaxed. Standing behind the child, the examiner asks the child to close his eyes while the child's head is turned slowly to one side and then to the other and to try to keep his arms in exactly the same position while the head is passively moved (tell him only his head should move). The examiner then turns the child's head 70 - 80° (or until the chin is over the shoulder) and holds that position for five seconds and then repeats this movement over the other shoulder.

Scoring: **0** – no movement of arms,

1 – slight movement of arms (up to 20°) to the same side as the head is turned or a slight dropping of the arms

2 – movement of the arms (up to 45°) as the head is turned or marked dropping of the arms

3 – An arm movement greater than 45°, either to the side or down or swaying and loss of balance

Ayers Test II for ATNR: Ayers II involves placing the child in a quadrupedal position and the examiner kneels in front of the child facing him. The child's body weight is evenly distributed between the upper and lower extremities, knees five to six inches apart, arms slightly abducted, elbows extended and slightly abducted, wrists hyper-extended in the neutral position, hands flat with fingers extended, head in neutral position and neck relaxed.

Scoring: **0** – no elbow flexion, that is, full elbow extension is maintained and no visible muscle tone changes are noticeable.

1 – slight muscle tone changes are minimally visible but there is no elbow flexion

2 – slight visible elbow flexion is less than 30°

3 – elbow flexes 30° – 60° with little shoulder depression and no noticeable trunk movement

4 – elbow flexes more than 60° causing depression of the shoulder with trunk movement but balance is maintained and the contralateral leg does not leave the surface.

5 – obvious loss of balance occurs when elbow flexes more than 60°, forearm may touch surface, contralateral leg may leave surface and quadrupedal position may be lost causing collapse of the body position.

Sample of Scoring Tables

Name:
Number: 1

TEST	Right 1	Left 1	Right 2	Left 2	Right 3	Left 3	Total Right	Total Left
Schilder								
Ayers II								
	Up 1	Down 1	Up 2	Down 2	Up 3	Down 3	Total Up	Total Down
TLR								

Name:
Number: 2

TEST	Right 1	Left 1	Right 2	Left 2	Right 3	Left 3	Total Right	Total Left
Schilder								
Ayers II								
	Up 1	Down 1	Up 2	Down 2	Up 3	Down 3	Total Up	Total Down
TLR								

Name:
Number: 3

TEST	Right 1	Left 1	Right 2	Left 2	Right 3	Left 3	Total Right	Total Left
Schilder								
Ayers II								
	Up 1	Down 1	Up 2	Down 2	Up 3	Down 3	Total Up	Total Down
TLR								

Name:
Number: 4

TEST	Right 1	Left 1	Right 2	Left 2	Right 3	Left 3	Total Right	Total Left
Schilder								
Ayers II								
	Up 1	Down 1	Up 2	Down 2	Up 3	Down 3	Total Up	Total Down
TLR								

Name:
Number: 5

TEST	Right 1	Left 1	Right 2	Left 2	Right 3	Left 3	Total Right	Total Left
Schilder								
Ayers II								
	Up 1	Down 1	Up 2	Down 2	Up 3	Down 3	Total Up	Total Down
TLR								