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Alternative therapies in the treatment of developmental dyslexia in preliterate children: An evaluation of retained reflex replication, vestibular system stimulation and related claims regarding early indicators.

by

Elaine Claire Smith

A dissertation submitted for the degree of Doctor of Philosophy of the University of Dublin, Trinity College, Dublin 2, Ireland.

This research was carried out in the School of Psychology.

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Declaration

I declare that this work has not been submitted previously as an exercise for a degree at this or any other university. The scale of the study necessitated two principal researchers, of which I was one, who shared all aspects of recruitment, testing, training and administration. However, the literature review, data-analysis and thesis write-up is entirely my own work. The Trinity College library may lend or copy this thesis without restriction.

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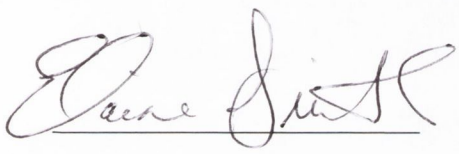

Elaine Claire Smith

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Thesis Summary

This study is concerned with the remediation, prediction and causal theories of developmental dyslexia and adopted a longitudinal, multi-factorial design in order to explore three principal claims. Exclusive methods of defining and diagnosing dyslexia, as well as dissatisfaction with narrow causal theories, have given (re)birth to alternative thinking about the disorder.

The thesis begins by introducing the reader to some conceptual points about the disorder, covering its definition, diagnosis, the problems with each of these, and its symptoms. It also explains the constitutional and genetic nature of the problem given the family-history based recruitment method subsequently employed. This is followed by a chapter devoted to three of the most popular and currently accepted causal theories of dyslexia which include the phonological deficit hypothesis, the magnocellular deficit hypothesis and the temporal processing deficit hypothesis. A critical review of each of these theories concludes that either they do not progress beyond a merely descriptive account of the disorder (as opposed to a causal one) or they fail to account for the complex array of difficulties with which dyslexic people present. This paves the way for a new theoretical suggestion – the cerebellar deficit hypothesis which goes further to address both of these matters than any of its predecessors. However, before this can be explored in full, it is necessary to introduce the reader to an overview of normal motor development in children as this underpins one of the aims of the research and is linked to a "normal" cerebellum. A summary of motor development is presented including primitive reflexes, postural reactions and voluntary movement as well as some of the sensory systems which afford this normal development (i.e., the vestibular system, proprioceptive system and visual system). This is followed by a chapter which explores the link between motor abnormality and dyslexia. This looks at the link between motor and cognitive development followed by some of the original theories in this area from the 1950s and 1960s, including "lateral dominance", "neuro-maturational theory" and "neurological organisation". It then considers some of the earlier work linking abnormal motor development / skills to dyslexic children and the attempts to remediate their difficulties by involving the children in specially designed motor programmes in order to stimulate neuro-sensory systems or inhibit primitive reflexes which are blocking the way to normal motor, and thus, cognitive, development. This leads to a review of recent studies which have revived an interest in motor theory and dyslexia and highlights their methodological problems. As the necessary background material is now covered, the next chapter explains the cerebellar deficit

hypothesis, preceded by a neuro-anatomical review of the cerebellum and its functions. The aims of the study are presented in the subsequent chapter, of which there are eight, covering three main issues. First, are motor programmes, which are designed to improve or encourage motor development / skills, useful in alleviating the symptoms of dyslexia? Second, can tests of cerebellar and motor skill be used to identify dyslexia in a young sample? Finally, is this evidence consistent with the cerebellar deficit hypothesis? Chapter 7 covers the research literature relevant to the tests and intervention programmes used in the current study which are divided into five sets of tests (i.e., balance tests, reflex tests, visuo-motor tests, fine-motor tests and phonological tests) and three motor programmes (i.e., an experimental balance programme, an experimental reflex-inhibiting programme and a placebo-controlled "pseudo programme"). These are described more fully in terms of procedure and materials necessary in the Methods chapter which also explains the design and describes the participants of the study. The final two chapters present the results and discussion. Little support was found for the idea that motor tests can be used for predicting which of a young sample of children are at risk for dyslexia, despite earlier claims to the contrary using older children. There is also no evidence for programme efficacy in reducing the severity of dyslexia compared to those in the placebo-control or control groups. The discussion reviews these findings and makes suggestions as to how this should affect current knowledge and practice. Limitations of the current study are presented and suggestions made for refining research in the area.

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"It is exceedingly difficult for the normal person to picture all the social difficulties which word-blindness can cause. Even when his condition is only moderately severe, the word-blind person is aware of it daily and in innumerable ways. His difficulties extend from trivialities which are of little practical importance but are embarrassing, to serious occupational disabilities"

Hermann (1959)*

* A quote from the book "Reading Disability" by Danish neurologist Knud Hermann (1959). Found in Arkell (1996).

1. Chapter 1: Introduction to dyslexia

1.1 Conceptual points

Reading, writing and spelling are requisite skills in Western culture, essential for maximising success professionally and personally. Though complex in nature, most of us assume fluent control of them by adulthood, and yet some people never develop such dexterity with written language. Of these, some will reach a level of acceptable efficiency, though they usually work harder to achieve it; others, however, may fail to attain a level of functional literacy at all.

Two points are necessary in order to clarify terms throughout the coming text. Firstly, although problems with any of reading, writing or spelling could constitute the principal part of a child's written language difficulty, they rarely exist in isolation from each other. A distinct problem affecting one of the three is more likely to be the result of some acquired disorder; brain injury resulting from either external trauma (e.g., a traffic accident) or biological factors (e.g., tumour, stroke). This is referred to in the neurological literature as aphasia or alexia though such aetiology is not the concern of this work. With regard to developmental dyslexia, however, it is usual to see some combination of difficulty with all three. Because reading relates to the other two in some way, such problems are often referred to in a global sense as "reading disorders". Secondly, because reading is a skill we learn as children, it is usual for developmental disorders affecting reading to be diagnosed at this time; as such, most of the literature and research on reading disorders concerns children. Thus, throughout the text regular reference will be made to children, although it is acknowledged that the primary disorder does not necessarily disappear once the child reaches adulthood, despite the coping mechanisms they may develop as adults.

The term "reading disorder", however, is a vague one. It encompasses the full population of those who have difficulty with reading, including those labelled as "backward readers", "garden variety poor readers" and those suffering from "developmental dyslexia" (also known as "specific reading disorder", "specific reading disability" and "specific learning disability"). The current work is not concerned with the first two, but focuses specifically on children who may be developmentally dyslexic. The term "developmental" recognises the congenital and neurological nature of the disorder; it is part of who the child is and always has been; it is an inherent, innate and genetic condition, rather than, as previously mentioned, an acquired

disorder. For simplicity's sake the thesis will refer throughout to "dyslexia" and where it does so it should be assumed that it is the developmental form that is under consideration rather than an acquired equivalent.

1.2 Dyslexia explained

1.2.1 A brief introduction

The term "dyslexia" is fraught with difficulties. Its definition and diagnosis do not lead one from the other. The descriptive criteria, as listed by various definitions, are not enough to guarantee a diagnosis of dyslexia because this is reserved for people who demonstrate an *unexpected* problem with the written word. This is where the idea of dyslexia as a neurological condition deviates from the commonly-accepted and socially-constructed idea of dyslexia as a discrepancy disorder between attainments and aptitude.

At its most basic, dyslexia is a developmental language disorder, where the primary symptom (frequently demonstrated in linguistic and cognitive studies) is a deficit in the phonological coding of written language, caused by a deficit in phoneme segmentation skills (Pennington, 1991). This easily detectable problem is not sufficient to warrant a diagnosis of dyslexia because in its full manifestation, the range of symptoms with which dyslexic people tend to present goes well beyond a problem with mere phonics or literacy. It is only because our society attributes such fundamental importance to the smooth acquisition of literacy that people are primarily identified in this way. This is not to criticise the stronghold academia has on the term; it is economic practice to ignore even abnormal variations in behaviour if they are not dysfunctional. However, this study explores the possibility of developing a more global assessment of a dyslexic's problems by employing alternative screening / testing procedures which catch all dyslexics, whilst still allowing for the socially-constructed definition of dyslexia (as a discrepancy) to identify brighter dyslexics if need be.

Although dyslexia has been defined in terms of insufficient phonological processing abilities, it is also generally accepted that dyslexia is the result of neuro-developmental deficiencies deep within the brain, ultimately the result of some genetic anomaly. Because reading, writing and spelling are modern additions to our species' repertoire of skills (having been invented as recently as 4000 years ago) it is highly improbable that, what-ever this anomaly, it would affect only those particular skills. As

there can be no genetic basis for these precise skills, the sub-skills underlying them must depend on fundamental sensory and motor processing functions that originally evolved for other purposes (Stein, 2004). Thus, we would expect there to be many difficulties commonly seen across the dyslexic population and which may or may not have anything to do with reading. This is, in fact, what the literature suggests.

1.2.2 Labelling

Diagnosing a disorder such as dyslexia should not be undertaken without a meaningful and working definition of the disorder upon which the diagnosis is based. However, defining, and even labelling, a syndrome such as dyslexia is not an easy task. Many different labels have been used over the years as approaches, fashions, political-correctness and policy changed.

Dyslexia literally translates as "difficulty with words" and was originally referred to as "word-" or "letter-blindness". The former was first used by Kussmaul in 1877 in reference to an adult female who suddenly acquired a difficulty in reading text after an attack of apoplexy. Subsequent medical practitioners commented on similar problems with a gradual move to developmental similarities. A German ophthalmologist, named Berlin, was the fist to use the term "dyslexia" in 1884 with regard to adults (Hallahan and Mock, 2003), and in 1896 both Kerr and Morgon reverted to the term "word-blindness", or "congenital word-blindness", after encounters with children of normal intelligence who demonstrated an inexplicable difficulty in learning to read (cited in Pennington et al., 1991; Selikowitz, 1993). The founder of the International Dyslexia Society, Samuel Orton, used the complex term "strephosymbolia", meaning "twisted words" in reference to the disorder.

The medical monopolization of dyslexia persisted well into the 1970s using labels relating to theories of causation (Szklut and Breath, 1985). "Minimal brain damage" and "minimal brain malformation" were common in the 1940s (depending on whether causation was attributed to environmental or genetic influences), yet at that time, no evidence of structural damage could be found to justify the former and, even now, analysis at the cellular level is not possible using CAT or MRI to justify the latter. "Minimal brain dysfunction" suggested a neuro-chemical problem yet this neither had ever been shown to be the case (Selikowitz, 1993). As far as 1978 Critchley, was insisting that dyslexia remain a medical responsibility (in Snowling, 1987); he

commonly referred to dyslexics as "patients" and regarded the doctor as the most suitable professional capable of diagnosing dyslexia. However, in 1977 the British Medical Association advised its members that dyslexia was not a medical problem (Doyle, 1996) and it has since become the ward of educationalists.

The educational profession approached the condition with terms relating to function rather than cause (i.e., "reading disorder). From the term "learning disabilities", coined by Kirk in the 1960s, evolved "specific learning difficulty" in the UK and "specific learning disability" in the US which was preferred to "dyslexia" throughout the 1970s and 1980s (BPS report, 1999). These days "learning disorder" has re-appeared in the US (Rittey, 2003) and "specific learning difficulty" in the UK, both of which act as umbrella terms under which dyslexia may be found alongside other conditions such as dyspraxia, dysgraphia and dyscalculia.

Up to 37 other terms, listed by Doyle (1996), include such diverse-sounding labels as attentional dyslexia, congenital word blindness, hyperlexia, mind blindness, semantic processor dyslexia, legasthenia and strephosymbolia. Thus, in the attempt to define the same learning impairment, we have been left with a range of varied and inconsistent terms. Many of these are now used interchangeably and synonymously, causing much professional confusion.

1.2.3 Definitions of dyslexia

There have been numerous definitions of dyslexia over the years. A precise definition is important because it guides diagnosis and treatment and, therefore, should aim to encompass all of those with the disorder. In this case it should do this by recognising the constitutional nature of the disorder and describing behavioural consequences in the light of varied theoretical causalities. This would allow for those of all IQs to be diagnosed as dyslexic. However, many definitions, despite acknowledging a biological cause, persist in making it more exclusive to some groups of children by adding exclusionary criteria (Rack, 1994). Led by Batemans's definition of learning disabilities in the US, dyslexia first became linked to the idea of an attainment-aptitude discrepancy[1] in the mid 1960s.

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[1] This is underlined where relevant.

"Children who have learning disorders are those who manifest an educationally significant discrepancy between their estimated potential and actual level of performance related to basic disorders in the learning process, which may or may not be accompanied by demonstrable central nervous system dysfunction and which are *not secondary to generalised mental-retardation, educational- or cultural-deprivation, severe emotional disturbance or sensory loss*"[2].

(Bateman, 1965, in Hallahan and Mock, 2003)

Although a discrepancy can occur at any level of intelligence, it is more meaningful and easier to interpret when the child is of average or above-average intelligence because the problem can be dissociated from other causes (e.g., mental retardation). This is the discrepancy criterion upon which all current practice is based, and which serves to bias diagnosis toward more intelligent children. Frith (1999) refers to this as the first paradox of the discrepancy definition of dyslexia. Irrespective of whether or not it is functionally worth identifying low IQ children as dyslexic, it is becoming controversial not to be able to do so ("The dyslexia myth", channel 4, 08-09-05 raised this very point).

The definition of the World Federation of Neurology (1968)[3], probably the most widely accepted definition, was one of the earlier ones to include such criteria:

"A disorder manifested by difficulty in learning to read *despite conventional instruction, adequate intelligence and socio-cultural opportunity*. It is dependent upon fundamental cognitive disabilities which are frequently of constitutional origin."

(Critchley and Critchley, 1978)

The term "Specific Reading Disability" (SRD), used in 1972 by the British Department of Education and Science (DES), (now known as the Department for Education (DFE) implied the same thing:

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[2] Italics explained on next page.
[3] In Critchely & Critchely, 1978

"SRD is a problem (in children) whose reading (and probably writing, spelling and number) abilities are significantly below the standards which their abilities in other spheres would lead one to expect"

(Doyle, 1996)

More detailed, though saying the same thing, is the definition of the International Dyslexia Association (2002) and shared by the National Institute of Child Health and Human Development (NICHD):

"Dyslexia is a specific learning disability that is neurological in origin. It is characterized by difficulties with accurate and / or fluent word recognition and by poor spelling and decoding abilities. These difficulties typically result from a deficit in the phonological component of language that is often unexpected in relation to other cognitive abilities *and the provision of effective classroom instruction.* Secondary consequences may include problems in reading comprehension and reduced reading experience that can impede growth of vocabulary and background knowledge."

(IDA, 2004)

In addition, there exists the paradoxical method of defining something by focusing on what it is not, rather than on what it is, which serves to exacerbate the exclusivity of the disorder. By excluding those with sensory problems, emotional problems, poor environmental circumstances and so on, it is understandable that dyslexia has acquired a reputation as a "middle-class" disorder. Such criteria[4] can make it difficult for children with an added disability or disadvantage, but who are nevertheless dyslexic, to get an appropriate diagnosis. This is apparent in the definition put forward by Critchley and Critchley (1978):

"A learning disability which initially shows itself by difficulty in learning to read, and later by erratic spelling and by lack of facility in manipulating written as opposed to spoken words. The condition is cognitive in essence, and usually genetically determined. *It is not due to intellectual inadequacy or to lack of socio-cultural*

[4] These are presented in italics where relevant.

opportunity, or to faults in the technique of teaching, or to emotional factors, or to any known structural brain-defect. It probably represents a specific maturational defect which tends to lessen as the child grows older, and is capable of considerable improvement, especially when appropriate remedial help is afforded at the earliest opportunity."

Some definitions are better worded, suggesting that such factors can co-occur, as long as they are ruled out as the primary cause. For example, in the US the "Individuals with Disabilities Education Act" (IDEA, 1997) defines "specific learning disability" as:

"A disorder in one or more of the basic psychological processes involved in understanding or using language, spoken or written, which the disorder may manifest itself in imperfect ability to listen, think, speak, read, write, spell or do mathematical calculations. Such a term includes such conditions as perceptual disabilities, brain injury, minimal brain dysfunction, dyslexia, and developmental aphasia. *Such a term does not include a learning problem that* is primarily *the result of visual, hearing, or motor disabilities, of mental retardation, or of emotional disturbance, or of environmental, cultural or economic disadvantage*"

(In the Task Force on dyslexia, 2002)

This definition requires a distinction to be made between those whose problems have an obvious cause and those who may have both dyslexia and an additional problem (e.g., mental-retardation), but the fundamental problem of how to do this remains.

1.2.4 The case in Ireland

The term "Specific Learning Disability" (SLD) was adopted in 1993 by the Special Education Review Committee (SERC) of the Department of Education and Science and is primarily a definition of elimination. They define an SLD as:

"Impairments in specific areas such as reading, writing and arithmetical notation, the primary cause of which is not attributable to assessed ability being below the *average range, to defective sight or hearing, emotional factors, a physical condition or to any extrinsic adverse circumstances.*" (Task Force Report, 2002)

This incorporates much of the baggage of previous definitions and as a result the Department commissioned a Task Force on dyslexia, the report of which was published in 2002. This offers a broader definition; its inclusion of cognitive characteristics and additional behavioural consequences allow for the application of disparate causal theories:

"Dyslexia is manifested in a continuum of specific learning difficulties related to the acquisition of basic skills in reading, spelling and / or writing, such difficulties being unexpected in relation to an individual's other abilities and *educational experiences.* Dyslexia can be described at the neurological, cognitive and behavioural levels. It is typically characterised by inefficient information processing, including difficulties in phonological processing, working memory, rapid naming and automaticity of basic skills. Difficulties in organisation, sequencing, and motor skills may also be present."

The recommended use of this definition has not yet been officially adopted by the Department of Education and Science. However, it too requires the use of a discrepancy and is exclusionary in its definition.

1.2.5 Ideal definitions

Perhaps the best definition is that of the National Joint Committee on Learning Disabilities (NJCLD[5]) in the US, which explicitly acknowledges the distinction between intelligence and dyslexia whilst still recognizing their possible co-occurrence. The commonly used term of "learning disabilities" in the US is laid out as follows:

"A heterogeneous group of disorders manifested by significant difficulties in the acquisition and use of listening, speaking, reading, writing, reasoning, or mathematical abilities. These disorders are intrinsic to the individual and are presumed to be due to central nervous system dysfunctions. Even though a learning disability may occur concomitantly with other disabling conditions (e.g., sensory impairment, mental retardation, social and emotional disturbance) or environmental influences (e.g.,

[5] Founded in 1975, the National Joint Committee on Learning Disabilities (NJCLD) is a national committee in the United States of representatives of organizations committed to the education and welfare of individuals with learning disabilities. (http://www.ldonline.org/about/partners/njcld)

cultural differences or insufficient/inappropriate instruction), it is not the direct result of those conditions or influences"

(in Szklut and Breath, 1985)

Dyslexia, as defined by this, is recognised as a constitutional disorder, incorporating a range of characteristics which are accounted for neurologically, thus, allowing for future developments of causal theories and explanatory models. Whilst recognising that dyslexia must be distinguished from confounding factors, it nevertheless acknowledges the possibility of their presenting together, thereby theoretically allowing all relevant children to be identified.

Both the British Dyslexia Association (BDA) and the Dyslexia Institute (1989) also have inclusive definitions:

"A specific difficulty in learning, constitutional in origin, in one or more of reading, spelling and written language which may be accompanied by difficulty in number work. It is particularly related to mastering and using written language (alphabetic, numerical and musical notation) although often affecting oral language to some degree"

(The BDA - Doyle, 1996)

"Organising or learning deficiencies which restrict the student's competencies in information processing, in motor skills and working memory, so causing limitations in some or all of the skills of speech, reading, writing, essay writing, numeracy and behaviour"

(The Dyslexia Institute - Doyle, 1996)

The latter is a particularly nice example as it is not only inclusive but makes allowances for a wide range of behavioural characteristics based on the kinds of weaknesses dyslexic people generally show.

1.2.6 Diagnosis

Although the DES and the Dyslexia Institute in the UK define dyslexia in an inclusive way, this does not necessarily correspond to practice. Based on the previous definitions, a formal diagnosis requires the following salient features:

- Written language problems in the bottom 10%
- A within average-range IQ
- Normal sensory functioning
- Normal emotional functioning
- An adequate educational environment

In Ireland in 1993, SERC formalised the diagnostic assessment for dyslexia with a list of recommendations and requirements stating that:

1) Assessment by a psychologist on a standardised test of intelligence should place general intellectual ability in the average or above average range (i.e., a threshold requirement).
2) Performance in basic skills in relation to objective criteria, such as standardised tests, should be at a very low level. It would be expected that some 2% of the overall pupil population would be found in this category (i.e., a discrepancy requirement).

(Task Force Report, 2002)

This practice is based on the idea that a full assessment should give a reliable indication of the aptitude and attainments of the person in question. The probability of there being a discrepancy by chance between these two scores is low and it gets lower as the size of the discrepancy increases. Thus, a specific learning disability (i.e., dyslexia) is inferred on the basis of a discrepancy, given that intelligence is normal. The severity of the disorder (mild, moderate, severe) depends on the size of this discrepancy. IQ testing is the usual method by which we can determine aptitude and eliminate low-intelligence as a possible cause of reading failure. More importantly, this also determines the expected reading ability of the individual. In children, assessment of aptitude usually consists of an intelligence measure such as the Weschler Intelligence Scale for Children, 4th

edition (WISC - IV) (Wechsler, 2003). In adults, the Weschler Adult Intelligence Scale, 3rd edition (WAIS - III) would usually be used (Wechsler, 1997). Both of these consist of 11 different sub-tests which form a verbal score (or IQ) and a performance score (or IQ) and contribute to the formation of an overall, full-scale IQ. The individual tests can yield further information, forming a profile of strengths and weaknesses, which may be of benefit when recommending remediation plans.

In addition to intelligence testing, tests of reading, writing and spelling need to be administered in order to measure any aptitude / attainment discrepancy. Such skills may be tested using any standardised test, such as, for example, the Wide Range Achievement Test, 3rd edition (WRAT-III) (Wilkinson, 1993), the Woodcock Reading Mastery Test (WRMT) (Woodcock, 1998) or the Weschler Objective Reading Dimension (WORD) (Wechsler, 1993). These are designed to measure reading recognition, speed, comprehension, decoding, spelling and arithmetic computation.

Despite the criticisms that such a discrepancy (based on an average IQ) invites, the fact is that a very often the acquisition of appropriate help depends upon it.

1.2.6.1 The problem

It could be argued that the use of a discrepancy (with IQ in the average range) and exclusions is not morally justifiable even if it is understandable. It is not justifiable on biological grounds because dyslexia is generally accepted to have a genetic basis and to be independent of IQ. It is not justifiable on moral grounds because it is biased against certain sections of the population, denying them, in some cases, the diagnosis and services that higher IQ individuals receive. Having said this, there is no commonly accepted way of clearly and accurately differentiating between a reading disorder which is due to "dyslexic" (i.e., neurological) causes alone and a reading disorder which is due to a low global IQ. As mentioned previously, detection of phoneme segmentation problems can be indicative of dyslexia and treatment could theoretically be prescribed on this basis alone. However, this does nothing to address the root of the problem, and given the current climate of obsession with neurobiological and genetic causes of such disorders, there is a movement to identify those at risk of dyslexia with such causes in mind.

In order to address the misconception that dyslexia only affects intelligent children, a change is needed in the way dyslexia is referred to and understood. Confusion abounds

in the literature because of the number of different uses of the term. There is the underlying neurological / developmental condition which is referred to as dyslexia; there is the academic symptom-led idea of dyslexia (i.e., phonological problems), and finally there is the socially-constructed discrepancy-definition of dyslexia (Table 1[6]). Consistency is needed with regard to the terminology used which, once in place, will make it easier to integrate the different approaches, explanations, theories and models from genetics, neurology, cognition, environment, behaviour and so on. In order to make sense of the wealth of information we need a way of conceptualising the disorder in question.

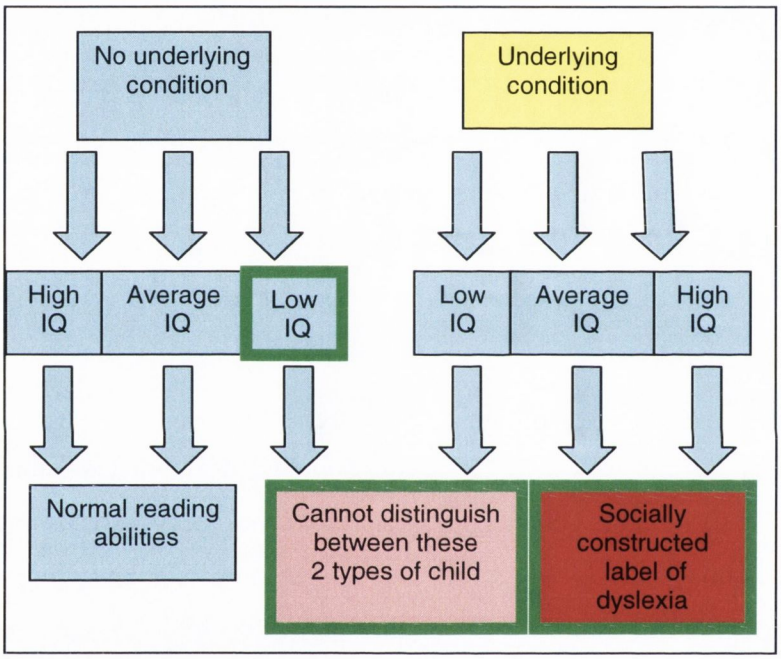

Table 1: Different conceptions of dyslexia.

What this table illustrates is that dyslexia, as defined by the geneticists and the neurobiologists, is an underlying condition (yellow box). However, only those children with this underlying condition of average or above IQ can be *currently* officially diagnosed as having the condition (red box – the socially constructed definition of dyslexia) because general learning difficulties can mask the dyslexic difficulty. In addition to these, there are those (e.g., Fletcher et al., 1994; Shaywitz et al., 1992) who argue that it is the symptoms of dyslexia that matter, regardless of cause, and that all

[6] Having read and researched the relevant literature, his table represents my own understanding of the way in which reading difficulties are understood.

children showing them should be treated equally (green-surrounds). They claim that reading ability is underpinned by phonological ability and that those with reading problems represent the lower end of the normal distribution of phonological ability. Thus, there is no need to invoke the use of the discrepancy criteria to determine who is dyslexic and who is not.

A potential answer is to coin one term for the neurological condition and to specify other characteristics on top of this. In this way, all children with the neurological condition would be referred to with one label ("dyslexia" could be retained for this) and further assessment could reveal the IQs or other pertinent information of the individual. This would provide sub-groups of the neurological condition. If "dyslexia" were not used to label the underlying condition, then it could be used to refer to just those of average or above-average IQ (as currently the practice) – as long as there was some system and label for identifying everyone else as well. If everyone is aware of such terminology and is consistent with its use, some of the confusion would be reduced. The problem with this is little in the way of replicated, empirically-demonstrated methods for identifying the relevant underlying, neurological disorder.

The current study aims to contribute towards this change in thinking about dyslexia by empirically examining the possibility of using a screening method for detecting the underlying condition. This would allow us to treat all children presenting with reading difficulties from this group in the same way, and to diagnose them according to the underlying condition and not according to a discrepancy. Testing for a discrepancy may have its place - allowing for further clarity with regard to the child's strengths and contributing towards individualized treatments - but in an ideal world, where "cause" and not "symptom" was treated, discrepancy testing would not be the focus of diagnosis. It is also important to stress that although reading might be a principal symptom of dyslexia, that there are other symptoms. Given the suggestion that the underlying condition is neurological, one would expect that the causes, and range of resulting symptoms, may lie outside the domains of reading and phonology. Such an approach, therefore, would permit identification of those who may have other difficulties resulting from the underlying cause which could then also be tackled.

1.2.6.2 Summary of the problem

As mentioned, a precise definition is important because it guides diagnosis and treatment, but producing an acceptable and precise definition has proved to be a difficult task. The principal problem is one of exclusion where diagnosis is biased towards more intelligent children and middle-class children. It may also run the risk of excluding those with other handicaps because, although many definitions state that dyslexia may be diagnosed if these other conditions are identified as separate entities, it is not always possible in practice to differentiate where one disability ends and another begins.

With regard to intelligence specifically, definitions have ranged from those explicitly requiring intelligence to be in the average range (World Federation of Neurology, 1978) to those requiring a discrepancy in ability and achievement even if not explicitly requesting that this ability be in the average range (DES – UK, 1972; NICHD, 2002). This is also currently the case in Ireland (SERC, 1993; Task Force, 2002) and yet in practice it is necessary for IQ to be in the average range in order for provision of services. The setting of this, and other, arbitrary, precise requirements (i.e., IQ $\geq$85, achievements in the bottom 10%) results in an inaccurate and socially-constructed idea of what dyslexia is, which does no justice to the research confirming a biological basis to the disorder. Furthermore, there is some question about the use of intelligence tests at all – even for those of average intelligence, however, this is another subject and the interested reader is referred to Stanovich (1996), Shane (1996), Siegel (1999) and Ellis (1985).

The main unfortunate consequence of the discrepancy-based assessment means that such children are not identified until they have been exposed to reading instruction for 2-3 years (Tunmer and Chapman, 1996) and have failed to learn appropriately.

1.2.6.3 Ideal practice

Tim Miles, working over the last half a century in the area of dyslexia, always viewed dyslexia as more than a reading difficulty. He maintained that dyslexia was a disorder of neurological basis and that a neuropsychological evaluation should be undertaken together with traditional diagnostic assessments in order to identify it (Frith, 1999). Frith argues that it is time to move away from the restricting definitions of reading failure by reference to arbitrary cut-off points and arbitrary discrepancies; her view is becoming more common today. If, as she suggests, we accept that dyslexia is the result

of a complex causal chain from biology to behaviour, we must think of it as a neuro-developmental disorder and not just as a reading problem.

The second paradox presented by Frith (1999) exemplifies this point and refers to the fact that diagnoses are not always stable. Because diagnosis is anchored to discrepancies and arbitrary cut off points (as berated in Frith's first paradox), and because reading skills may gradually improve, even in dyslexics, it is quite possible that at an older age they no longer satisfy the requirement of being dyslexic that they once did, despite feeling that their difficulties persist. Thus, what is needed is a measure of these underlying difficulties which is distinct from a measure of reading ability.

1.2.7 Characteristics and symptoms

The Irish Task Force on dyslexia (2002) identified the following as the main characteristics and symptoms of dyslexia:

Characteristics

- It occurs across the lifespan and may manifest in different ways at different ages.
- It may co-exist with difficulties in the area of numbers.
- It may be associated with early spoken language difficulties.
- It may be alleviated by appropriate intervention.
- It can increase or reduce in severity depending on environmental factors.
- It may occur in all socio-economic circumstances.
- It may co-exist with other learning difficulties such as Attention Deficit Disorder and may or may not represent a primary difficulty.
- It may range along a continuum from mild to severe and thus, requires a continuum of interventions and other services.

Task Force (2002)

Dyslexic children show deficits in a range or areas, including, reading, phonology, writing, spelling, memory, speed, hearing, vision, balance, learning, skill, genetics, brain structure and function (Fawcett and Nicolson, 2001).

Symptoms

Indicators of a possible learning difference (Ages 3-5 Years):

- Is later than most children in learning to speak.
- Has difficulty pronouncing some, especially multi-syllabic, words.
- Has difficulty separating spoken words into sounds and blending spoken sounds to make words (i.e., has difficulty with phonological awareness).
- Experiences auditory discrimination problems.
- Is prone to spoonerisms (e.g., fips and chish for fish and chips).
- Has difficulty with rhythm.
- Is unable to recall the right word.
- Is slow to add new vocabulary.
- Exhibits delays in acquiring emergent literacy skills (e.g., understanding that written language progresses from left to right, discriminating between letters, words and sentences).
- Experiences problems learning the alphabet.
- Has trouble learning numbers, days of the week, colours and shapes.
- Has trouble learning to write and spell his / her own name.
- Is unable to follow multi-step directions or routines.
- Is developing fine motor skills more slowly than other children.
- May have difficulty telling and/or retelling a story in correct sequence.

Task Force Report (2002)

Indicators of a possible learning difficulty arising from dyslexia (Ages 5-7+):

- Is slow to learn the connection between letters and sounds (alphabetic principle).
- Has difficulty separating words into sounds and blending sounds to form words (phonemic awareness).
- Has difficulty repeating multi-syllabic words (e.g., emeny for enemy, pasghetti for spaghetti).
- Has difficulty decoding single words (reading single words in isolation).
- Has poor word-attack skills, especially for new words.

- Confuses small or "easy" words: at/to; said/and; does/goes.
- May make constant reading and spelling errors including:
    - Letter reversals (e.g., d for b as in dog for bog).
    - Letter inversions (e.g., m for w).
    - Letter transpositions (e.g., felt and left).
    - Word reversals (e.g., tip for pit).
    - Word substitutions (e.g., house for home).
- Reads slowly with little expression of fluency (oral reading is slow and laborious).
- Has more difficulty with function words (e.g., is, to, of) than with content words (e.g., cloud, run, yellow).
- May be slow to learn new skills, relying heavily on memorizing without understanding.
- Reading comprehension is below expectation due to poor accuracy, fluency and speed.
- Reading comprehension is better than single-word reading.
- Listening comprehension is better than reading comprehension.
- Has trouble learning facts.
- Has difficulty planning or organizing.
- Uses awkward pencil grip.
- Has slow and poor quality handwriting.
- Has trouble learning to tell the time on an analogue clock or watch.
- Has poor fine motor co-ordination.

Task Force Report (2002)

Thus, dyslexia involves many different difficulties although not all of them are necessarily evident in each individual. The language characteristics of dyslexia are most obvious; these include problems with reading, writing and spelling, evidenced by visual rotations and reversals, omissions and substitutions, pronunciation and structural analysis problems with subsequent difficulties in comprehension (Critchley, 1970). Yet, dyslexia is a disorder of lifelong persistence of which reading retardation is only one, albeit a principal, manifestation (Ramus et al., 2003).

The list below gives an idea of the feats a dyslexic person may have to cope with.

Short working memory (STM problems): This is made evident in tests of working memory such as digit-span recall. The dyslexic is significantly less able to repeat a string of numbers than is a non-dyslexic and this applies both for digit-span forwards and backwards. Similarly, when asked to repeat a sentence word for word, although dyslexics may grasp the sense of what has been said, many will have difficulty copying it exactly (Doyle, 1996) and the task of repeating polysyllabic words presents equal difficulty (Miles, 1974). Dyslexics may spend an inordinate amount of time learning material which is subsequently easily lost.

Sequencing difficulties: This may result in the inability to remember information in the correct order. This applies to such things as the days of the week, the months of the year or reciting multiplication tables. Younger children have more problems with such tasks although adults will frequently demonstrate similar problems. Some will not even know where exactly to start (for example, in the case of the months of the year, they may not start with January). This can apply also to the number sequence and arithmetic, with confusion over which direction to count or to perform a subtraction. This may be related to spatial difficulties in which the individual may have problems telling left from right, "b" from "d", east from west, as well as more general problems with times and dates (Doyle, 1996). For older dyslexics this may affect the logical sequencing of ideas (Heaton and Winterson, 1996).

Temporal difficulties: In a child this may result in confusion over the time of day, i.e., morning or evening (Snowling, 1987). Sequencing problems may also be evident temporally, with children showing difficulties in repeating sounds in the right order. This may be demonstrated when asked to repeat polysyllabic words such as "contemporaneous" (Heaton and Winterson, 1996).

Gross motor problems: It is common to see difficulties with postural stability and motor coordination in dyslexic children which affects all major activities such as hopping, skipping, catching / throwing a ball, learning to ride a bike or swimming. These may either be too difficult for the child or be clumsily performed.

Fine motor problems: These can make small, everyday tasks such as tying a shoe-lace and holding a pen problematic.

Mixed cerebral dominance: This means that the individual's right or left side may dominate in terms of handedness, leg dominance, eye and ear dominance, varying from one limb or side to the other. In most people one side will dominate for each of these tasks but in dyslexics there tends to be ambiguity of laterality. This means that neither side will become as fluent and automatic in performing the task as it should and, thus, manual dexterity will be less efficient.

Other problems may extend to visual memory, visuo-spatial abilities, attention and auditory perception. Although these problems will have ramifications well beyond the school-room, it is the school-room where the consequences of such problems are most obvious.

The current study is concerned with methods of screening which do not rely upon difficulties in the class-room. We have to look for manifestations of the disorder in the younger child, such as those suggested by the Task Force report (2002).

Stanovich's (1996) criteria for a biologically-based condition suggests that, in addition to evidence of distinct performance, there should also be evidence of heritable and neuro-anatomical patterns. The research exists in abundance for each, some of which is presented here (neuro-anatomical evidence will be discussed at a later stage).

1.2.8 Subtypes

The idea of different subtypes of dyslexia began to emerge in the 1960s (Johnson and Myklebust, 1967) and has received theoretical support since then (Boder, 1971; Mattis, French and Rapin, 1975; Denckla, 1977; Castles and Coltheart, 1993). The basis of subtype justification is that the reading difficulties of dyslexics are not all identical (Frith, 1999); some have problems with all words where others may be fine with regular non-words but have difficulty with irregular words.

The normal development of reading according to Selikowitz (1993) involves three stages; the visual memory (or logographic) stage, the phonological (or alphabetic)

stage and finally the automatic (or orthographic) stage which is the level of adult, competent reading. The first two stages are acquired by children and act as the preparatory stages for the automatic stage. The first of these, the visual memory stage is that in which words are recognised on the basis of their general shapes and characteristics. This works adequately for a limited number of simple words but is not so efficient when reading content expands and becomes more complex. The phonological stage is that in which the rules of letter and phoneme sounds are used to sound out and pronounce words. This application of rules allows for never-before-seen words to be pronounced but because some phonemes correspond to different graphemes, the context may be invoked to guide in pronunciation. In so doing, a lexicon, or memory bank of words, is constructed for familiar words. This allows for the acquisition of automatic reading because now familiar words can bypass the tedious necessity of mental translation from letters to sounds, allowing instead, fast identification of words based upon the visual processing of letter shape and order (i.e., the orthographic processing). Eventually unfamiliar words may also be processed automatically by comparing them to pre-existing, similar words, and all of these processes are linked to the semantic system allowing for the comprehension of each word. This has resulted in suggestions of three main subtypes of developmental dyslexia; visual (surface), phonological and deep dyslexia (Doyle, 1996; Ellis, 1993).

However, the existence of subtypes was not supported by Thomson (1999) in his study investigating persistence of subtype classifiable errors in 11 year-old children over a 2 year period. He suggested that subtypes were, in fact, an artefact of either reading-age and / or the different types of instruction the children received. The most recent evidence on subtypes suggests that at a causal level different types of dyslexia are unlikely to exist, but rather the specific symptoms a dyslexic shows are the result of other factors such as general intelligence, motivation, encouragement, practice and self-confidence (Fawcett et al., 1996). Frith (2001) also suggests that clinical variability may be a result of the interactions of causal factors from different levels (i.e., biology, cognition and behaviour) with the deficit. Subtypes are often discussed in relation to acquired dyslexia in any case and rarely in relation to the developmental form (see Ellis, 1993). Fawcett et al. (1996) found that 25 / 29 (i.e., 86%) of their dyslexic sample showed evidence of motor task (i.e., cerebellar) impairment compared to just 5 / 26 (i.e., 19%) of the control sample with no evidence of different profiles for different groups of children. In addition, most children in Nicolson and Fawcett's (1994a) other

studies demonstrate difficulties across a range of tasks, including not just motor tasks, but also phonological and information-processing speed tasks. This would suggest a certain homogeneity in the cause of the disorder.

The traditional and most effective method of treating dyslexia is the use of phonological teaching. This method is labour intensive, however, which has led to discoveries of "cures" over the years (see Silver, 1975 & 1986 for a review of controversial approaches to treating such disorders). Some of these claims are well-intentioned, attempting to cite research or doing small studies; others are not so conscientious. Most are considered controversial as the evidence to date of "miracle" or quick "cures" has been largely disappointing as we shall see later. The current study explores some currently popular miracle cures with a view to deriving objective evidence, regardless of the direction it takes, to inform the practising fields.

1.2.9 Incidence and prevalence

Orton was one of the first to suggest that dyslexia may affect up to 10% of the population. Prior to this Hinshelwood had put the prevalence at about 1 per 1000 (in Hallahan and Mock, 2003).

A study carried on the Isle of Wight demonstrated statistical evidence for the existence of a *specific* reading disorder (Rutter, 1978). An unexpected hump at the lower tail of the normal distribution of reading ability suggested that there is some other explanation for poor reading than normal variation. These children had many characteristics in common; they were more likely than other children to have had speech and language problems as well as problems discriminating left from right; they were three times more likely to have a family history of such problems and they were comprised largely of boys, in a ratio of about 3 to 1 (Selikowitz, 1993). Thus, they seemed to represent a particular sub-group of reading disordered children.

Exact prevalence depends upon where we put the arbitrary point which separates normality from abnormality (Ellis, 1985) and for this reason quotes vary from country to country. In the UK, estimates are approximately 10% of children. In their Isle of Wight reading study, Yule et al. (1973) found specific problems in approximately 5% of children, although their finding was higher around London, measuring about 10%; similarly Stein (2001) found 9.4% of his sample to have such specific difficulties. Approximately 4% are usually found to have a severe problem,

with a further 6% demonstrating milder symptoms (Cashman and Lyddy, 2003). Badian (1984) reports similar percentages, with the disorder affecting up to 10% of the population, though of these, 3-7% represent severe cases. This is supported by the World Federation of Neurology (1968) which also reports figures of 3-10%. In the US, however, estimates tend to be slightly higher, generally between 10 and 17% (deFries, Fulker and LaBuda, 1985; Shaywitz, 1998), although estimates as high as 30% have been reported (Eisenberg, 1978, in Pennington, 1991). The variety in definitions has already been discussed and serves to demonstrate the difficulty that might be involved in estimating true prevalence or incidence. As mentioned, the IQ discrepancy surrounding a diagnosis of dyslexia is controversial, with some invoking it and others not. This could account for one reason why estimates differ not just world-wide but within countries as some regard all reading disabilities equally and refer to them as a homogenous group where-as others believe in clarifying the distinctions between backwards readers and dyslexic readers. Miles (1996) even takes issue with the use of the IQ discrepancy, defined by him as "poor readers in relation to intelligence" (p. 88) because it makes no allowance for "certain specified tasks" (other than reading) at which dyslexics are also weak (e.g., responding to tachistoscopically presented digits and slower single word reading than reading-age matched controls). How then we determine prevalence and incidence depends entirely on what our definition is, and this point should be constantly borne in mind.

There have been no specific Irish studies to determine the prevalence of dyslexia within the Republic. The 1998 National Assessment of Reading Achievement found that approximately 10% of Irish 10-11 year olds had serious literacy difficulties. This corresponded to 11% of boys and 7% of girls, where "serious literacy difficulties" referred to a level of reading 2 or more levels below current class level (Cosgrove et al., 2000). Following this, the 2002 Task Force on dyslexia was appointed to address the issue of dyslexia within the country. This reviewed the range of educational provision and support services available to such children, assessed the adequacy of current educational provision and support services and made recommendations for the development / adjustment of existing policy approaches, educational provision and support services. Based on international studies the Task Force estimated that between 6.5 – 8.5% of Irish 14 year olds have literacy difficulties that are likely to put them at an educational or social disadvantage. Thus, at this time we can only assume that rates of dyslexia in Ireland are similar to those found in international studies.

1.2.10 Gender differences

General Population: As with most neuro-developmental disorders (e.g., autism, Asperger's Syndrome, Tourette's Syndrome, Attention Deficit Hyperactivity Disorder, dyspraxia) there seems to be a higher proportion of dyslexic males than females within the general population, with males outnumbering the females by approximately 4 to 1 (Vogel, 1990). One of the largest and most well-known family reading studies to date, the Colorado Family Reading Study (CFRS)[7], reported a referred sample of reading-disabled children with a gender ratio of 3.3 to 1 for males to females respectively (deFries, 1991) although a more recent study in 2000 by Flannery et al. found that reading disability was twice as common in boys as girls. This study was part of the National Collaborative Perinatal Project (NCPP) and was set up in response to the claims that referral bias accounted for a higher number of boys than girls suffering from dyslexia. However, Katusic et al. (2001) found that boys were 2-3 times more likely than girls to evidence a reading problem regardless of identification methods used.

Within Families - Siblings: There is no doubt that language disorders, including dyslexia, tend to aggregate in families. The fact that affected children have close family members with similar problems was first noticed and documented by Morgan in 1896 (cited in Stein, 2004), followed by similar observations by Thomas (1905), Fisher (1905), Hinshelwood (1907; 1911), Stephenson (1907; cited in Pennington et al., 1991) and Orton (1927; cited in Gallagher et al., 2000). From the Colorado Family Reading Study deFries (1989) showed that for dyslexia specifically, the sex ratio in families is more balanced. He found a male-female ratio of 1.5–1.8 to 1, which he claims is characteristic of family samples. An updated report in 1991 by Pennington et al. revealed a similar male-female ratio of 1.2–1.7 to 1. Indeed Shaywitz et al. (1990) claim to have found an equal ratio between the genders when comparing research-identified boys to research-identified girls. They claim that unequal prevalence is the result of referral bias, occurring when samples rely on school-identified children.

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[7]The Colorado Family Reading Study (C.F.R.S.) was established in 1973 with the multiple aims of: assessing familial transmission of reading disorders; assessing reading deficits in close family members of reading disordered children and constructing a test battery to differentiate reading disordered children from controls (deFries, 1991).

Within Families – Parents: Another report from the Colorado Family Reading Study, conducted by Vogler et al., (1985), looked at the predictive importance of affected parents for the two genders. Here the risk for males is increased by a factor of 4 - 10% and the risk for females is increased from 8 - 13%. Thus, while the incidence of reading disabilities may be lower for females than males in the general population (2.2% and 7.8% respectively, where the overall incidence is 5%), the presence of a parental history may increase the *relative* risk more so for females than for males; the gender of the parent, however, seems to be irrelevant.

Explanations: There have been several attempts to explain why we see a lower prevalence of dyslexia in females compared to males, both in the general population as well as within affected families. Pennington (1991) suggests that from a sociological view-point males are more likely to misbehave as a result of their problems than are females which brings them to the attention of diagnosticians. He also points to the biological advantage females tend to have for language skills which may serve to reduce the rate and or severity of dyslexia occurring in females. This ties in with the theory of Feldman et al. (1995) who suggest that the severity of dyslexia in males compared to females is possibly the cause of a reported higher prevalence of dyslexia among males. There is also some evidence that genes carried on the X chromosome may be responsible for this imbalance; where girls have two X chromosomes (one from each parent), boys only have one, which comes from the mother. As Selikowitz (1993) explains, should an X chromosome have some genetic fault, then a girl can use the corresponding gene on her other chromosome. Boys, however, do not have an alternative X chromosome and, thus, are left with the faulty genes. Snowling (2003) points out too that the behavioural manifestations of dyslexic (reading) disorders can vary in severity depending on the language and phonological skills the child has to begin with. In addition to this, protective factors include the timing and quality of the teaching such children receive, differences in print exposure as well as their own attention abilities. All of these individual differences may compensate for a poor phonological pathway within dyslexic families, especially if there is any truth to Selikowitz's argument. This would make assessing the continuity within families difficult to properly assess.

In summary it seems that, for each gender, there exists a substantial increase in the risk of developing dyslexia both when the parents are themselves dyslexic and when a sibling is dyslexic, compared to those children from unaffected families.

1.2.11 Genetic basis of dyslexia

1.2.11.1 Familial Aggregation: Hereditary and Genetic Studies

Family Studies: In looking specifically at dyslexia, Scarborough's (1990) prospective study in which she followed children from "dyslexia at risk" families, containing dyslexic parents, until they were 8 years-old, found that the chance of developing reading disabilities was 65%. Lahey and Edwards (1995) were similar with their finding of 60%. However, the earliest retrospective study was that conducted by Hallgren in 1950 who suggested that the risk of dyslexia occurring in first-degree relatives of affected children was approximately 41% (in Pennington, 1991), compared to the general population risk of about 10%. Bishop and Edmundson (1986) found that 46% of their children had a family member with similar difficulties. Although Gallagher et al's (2000) dyslexia study found 57% to be delayed in literacy development at age 6 years compared to just 12% in the control group, both Gilger et al. (1991) and Pennington (1990) put the familial risk rate for dyslexia at the lower percentage of 35- 45% from the Colorado studies. When looking at the predictive validity of family history, Scarborough (1990) determined that it accounted for 30-36% of the outcome for dyslexia in children.

Twin Studies: The use of classical twin methodology provides a solid way for estimating the overall heritability of such disorders. Both monozygotic and dizygotic twins share the same environment, thus, if dyslexia were caused by environmental factors alone we would expect both types of twins to present similar concordance rates of dyslexia. However, monozygotic twins share more than just their environment; they share 100% of their genes compared to 50% in dizygotic twins. Therefore, if dyslexia were a genetic disorder we would expect a higher concordance rate between monozygotic than dizygotic twins.

Hallgren's 1950 study made use of monozygotic and dizygotic twins to assess the probandwise concordance rates for dyslexia. He reported 100% concordance rate for the former and 52% for the latter. Selection bias could have resulted in these particularly high findings (cited in deFries et al., 1997). Vandeberg (1967), however, estimated the concordance rate of dyslexia for monozygotic twins to be 92%, whereas for dizygotic twins it was just 49% (cited in Lewis and Thompson). Bishop, North and Donlon (1995) found a smaller difference between the concordance rate for the two types of twins; monozygotic twins were 70% and dizygotic twins 46% but only for

males. Female twins' concordance rates were very similar at 44% and 43% respectively (cited in Spitz et al., 1997). Between 1982 and 1991 a twin study was initiated in Colorado as part of the reading study already underway there. Monozygotic and dizygotic twins, at least one of whom had a reading problem, were subject to an intense battery of psychometric tests. The results supported the hypothesis that genes play some part in the aetiology of dyslexia, as the concordance rate for monozygotic twins was 70% compared to 48% for the dizygotic twins (deFries, 1991). Based on a carefully devised multiple regression technique, which took into account the fact that scores for dizygotic co-twins should regress more towards the mean of the population than should that of monozygotic co-twins, deFries et al. (1987) assessed the heritability of reading and spelling, suggesting that about 30% of reading ability is due to heritable factors, though Pennington (1991) claims that with additional data these estimates have now risen to 50%. This was confirmed in 1989 by Wadsworth et al. who also found that half of the reading performance of reading-disordered co-twins was due to heritable factors[8].

Genetic Studies: Though an important methodological tool for the indication of a genetic cause, Gopnik and Crago (1990) point out that a family study cannot *prove* the genetic hypothesis. What is needed for this is direct evidence of a difference in the genetic structure that distinguishes the dyslexics from the non-dyslexics. As previously acknowledged, because reading, and thus, dyslexia, is not an innate skill, there is no genetic code for it per se; it is more the case that abilities underlying the process of reading are impaired and thus, location of these genes might contribute to our understanding of what is going wrong in dyslexics. The principal methods of gene location are summarised by Stein (2004) and listed below:

1. Candidate Studies: Candidate studies involve a lengthy and complex process of determining which genes may be responsible for a disorder and examining them from affected families to see if they are mutated.

2. Linkage Studies: Linkage studies, like association studies (see below), identify chromosomal sites containing single nucleotide polymorphisms (SNPs), to see if these occur more often in dyslexics than control readers. If they do it could indicate that a nearby gene is, at least partly, responsible for reading. Linkage studies determine

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[8] When such studies refer to the heritability of reading it is acknowledged that the genetic basis underlying such abilities / disabilities is not for reading *per se*, but for those skills underlying reading.

whether dyslexic members of families are likely to evidence these SNPs more often than would be expected by chance alone. Linkage studies are employed as an initial step to identify a genomic region which may be related to a given disorder. This region may contain hundreds of potential candidate genes however, so once an area is identified, then association analyses are conducted to locate which of these genes is mutated (Francks, et al., 2002).

3. Association Studies: Association studies are based on the idea that the same genetic mutations are exhibited by unrelated, dyslexic individuals. Few of these studies have been conducted with regard to dyslexia research because they depend upon a concise idea of a gene's localisation on a chromosome.

There are several different chromosomes which are thought to play a role in dyslexia; Stein (2004) suggests that there are probably 5 to 10 genes acting together to cause the disordered sub-skills of reading (i.e., phonological skills, orthographic skills and other aspects of auditory, visual, memory and motor processes).

Chromosome 1: Rabin, Wen, Hepburn et al. (1993) found evidence from 9 three-generational families of genetic abnormalities on chromosome 1 and a translocation of chromosomes 1 and 2 was found in a family reported by Froster et al. (1993).

Chromosome 2: Three studies have found evidence of a genetic link to chromosome 2: one in Norway, one in the US and one in Canada. Fagerheim et al. (1999) conducted a study on an extended family in Norway; Petryshen et al. (2000) found evidence from 96 Canadian families and Francks et al. (2001) conducted a dizygotic twin study in which 119 pairs were involved. All of these studies showed positive evidence for anomalies on the short arms 15 and 16 of chromosome 2 (Fisher and Smith, 2001).

Chromosome 6: There is strong evidence that dyslexia is linked to the short arm of chromosome 6 (C6). Cardon, Smith et al. (1994) reported evidence for a linkage on C6 in the Human Leukocyte Antigen (H.L.A.) region both for normal siblings and dizygotic twins, one of whom had reading disabilities. Where these reading disabilities were more severe, there was even stronger evidence for this site and many other studies have replicated this finding (Grigorenko et al., 1997; 2000; Fisher et al., 1999; Gayan et al., 1999). The studies seem to suggest that C6 is related to phonological decoding and orthographic coding (Francks et al., 2001), rather than single-word reading (Fisher and Smith, 2001). In addition, a specific gene for the magnocellular layer of the brain's

visual processing system, has been found in chromosome 6 (Simpson, 2001). Disruption to this layer in dyslexics forms one of the biggest causal theories for the disorder (e.g., Stein and Walsh, 1997) and so anomalies here could provide strong support for this theory.

Chromosome 15: Smith, Kimberling, Pennington and Lubs (1983) reported the first credible evidence of possible linkage on C15, again from 9 three-generational families suffering from dyslexia. However, this region was not replicated in further linkage studies (Lubs, Duara, Levin et al., 1991, and Lubs, Cross-Glenn, Duara et al, 1993), even when the initial family data were included (cited in Rabin, Wen, Hepburn, Lubs, Feldman and Duara, 1993). Indeed, it was not even replicated by the same authors (see Smith and Kimberling, 1991, cited in Cardon et al., 1994). It seems that 70% of the highly credible score yielded in this study was in fact due to transmission in only one of the nine families (deFries, 1991). deFries suggests that approximately 20% of families may have linkage to this chromosome, and thus, heritability of dyslexia must be heterogenic (also see Pennington, 1991).

A linkage-analysis study in Denmark also failed to show any evidence for the importance of this chromosome (Bisgaard et al., 1987), however, some other studies have also made use of extended families to demonstrate evidence of abnormalities on this chromosome. Smith, Pennington et al. (1991) subsequently did report a replication of their previous results, and this was repeated by Fulker et al (1991) and Grigorenko et al. (1997) in the US. However, these markers were not precisely localised which makes subsequent comparisons to these studies somewhat difficult (Fisher and Smith, 2001). Other countries have also found some evidence of C15 involvement in reading disabilities, including Germany (Schulte-Korne, 1998), the UK (Morris et al., 2000) and Finland (Nopola-Hemmi et al., 2000).

Chromosome 18: This area is thought to be involved in single-word reading (Francks et al., 2002), the data of which comes from 80 separate families (Fisher, Francks, Marlow et al., 2002) and is the strongest linkage data to date.

The sheer size of the human genome makes it difficult to conclude precisely which chromosomes, and location on them, are relevant when it comes to genetically locating dyslexia, but the complexity of this task is increased by the lack of consistency with regard to definition of the disorder, use of diagnostics tools as well as different sampling strategies and genetic analysis techniques between different studies (Fisher

and Smith, 2001). The research to date gives us very little conclusive results and will probably serve simply to guide future research in this area.

1.2.11.2 Genetic conclusion

Thus, we have seen that dyslexia is both familial and heritable. Early documented case-studies, followed by studies of familial aggregation and later by twin studies, indicated the probability of a genetic basis for dyslexia, with approximately 35-50% of first degree relatives being affected. From the Colorado studies it has been estimated, using the deFries-Fulker regression, that approximately 50% of the variance for reading disabilities are due to genetic factors. This compares with an occurrence in approximately 4% of the normal population which demonstrates the importance of heritability factors. Recent association and linkage studies have made headway in answering questions about where and how these genetic anomalies occur. Genetic markers have been located on several different chromosomes, including 1, 6, 15 and 18. Evidence from Galabuda's dyslexic brain studies (Galaburda and Kemper, 1979; Galaburda et al., 1985) showed that cell migration had gone awry and missed its intended location in the cortex, entering instead the molecular layer and causing mild disorganisation of nearby cortical layers (see Ramus, 2004, p. 722). There is evidence too of a genetic susceptibility in dyslexics (gene DYS1C1) which is responsible for neural migration (Ramus, 2004, p. 723). Given that family history is so obviously an important risk factor, it was deemed a reasonable assumption that in appealing to families with such a history, particularly if dyslexia had been officially diagnosed and particularly if they had a son of the appropriate age, that the current study would be successful in recruiting children who had a genetic predisposition toward developing dyslexia. Furthermore, in light of the findings by Stevenson et al. (1987), who found a relationship between age and disability manifestation, our appeal was open to parents who suffered from, either or both of, reading and spelling difficulties in school.

1.2.12 Co-morbidity

The issue of dyslexia and co-morbidity suggests that there may be an underlying neuro-developmental disorder, the symptoms of which may manifest in different clusters or severities from person to person. In this way we can explain why there are veins of similarities throughout the dyslexic population whilst the specific strengths and

weaknesses of individuals may differ greatly. Depending on the symptoms, a person could be diagnosed with one or more developmental disorders. Evidence for this comes from the fact that although there is strong linkage for reading problems with chromosome 6, this is not peculiar to this disorder. Willcutt et al. (2002) have linked this same site with ADHD, a condition highly co-morbid with dyslexia. Half of those diagnosed with either of these conditions show symptoms of the other condition (Stein, 2004). Similar studies have shown that of those with ADHD, approximately half also fit the diagnostic criteria for Developmental Co-ordination Disorder (DCD) (Barkley et al., 1990; Kadesjo and Gillberg, 2001). Others have reported even higher probabilities; Kaplan et al. (1998) claimed that 69% of their ADHD sample and 63% of their dyslexic sample showed evidence of DCD with Iversen et al. (2005) claiming that up to 70% of dyslexics are at the borderline or clinical range for DCD. MacArthur et al. (2000) showed that about 50% of both dyslexic children and children with specific language impairment (SLI) fulfilled the criteria of the alternative diagnostic category. Furthermore, chromosome 6 is close to an area responsible for dealing with auto-immune diseases. Abnormalities here could result in problems such as hay fever, asthma, allergies and eczema, all of which dyslexic families suffer from at a rate more prevalent than in the general population. Chromosome 6, for example, may be responsible for various sensory, cognitive, behavioural and attentional skills all of which are necessary for the smooth pursuit of every-day living (Stein, 2004). Were a mutation to arise here then these skills could be affected in any combination of ways, resulting in profiles of any number of neuro-developmental disorders (dyslexia, dyspraxia, ADD, ADHD, specific language impairment, etc). Thus, genetic research, as Francks et al. (2002) point out, suggests we should discard the idea of dyslexia as a categorical trait, in favour of a varying continuum.

This use of gene localisation provides the rationale for a new conceptualisation of dyslexia: one that fits Turner's (1997) view of a range of Specific Learning Disorders, of which dyslexia is one. Thus, discrete dysfunctions can give rise to co-morbid problems in one child, or explain why a range of disorders may be commonly found in particular families. In addition, it may account for why different diagnosable conditions show overlapping symptoms. This approach represents the view of certain organisations practising within the field of learning disabilities today, whose ideas and practices shall be explored in the following pages. It also supports the Swedish practice

of automatic attention, motor-control and perception screening for all 6 year-olds entering the school system, in the attempt to catch those at potential risk of neuro-developmental disorders regardless of the specific symptoms they may go on to show (McPhillips and Sheehy, 2004).

2 Chapter 2: Causal Theories

2.1 Theories of dyslexia

Scarborough (1990) neatly captures the inherent difficulties involved when discussing the causes of dyslexia; causes, she says, depend on whether one takes the traditional view of dyslexia as a reading problem, or as a broader condition of which reading problems are simply the most obvious symptom. Because dyslexia is now accepted as a congenital and neurological disorder, it is rarely viewed as the former. A weakness in phoneme discrimination might explain reading disorders, but it will not account for the other problems commonly seen in dyslexics. Such are the issues which must be accounted for when developing a theory for dyslexia.

There is a huge number of theories attempting to account for dyslexia, most of which purport to be causal theories when in fact they are descriptive models. The matter is confused not just by disparate theories which are difficult to integrate but by those which appear to contradict each other (Heaton and Winterson, 1996). Heaton and Winterson point out that multiple causality is not the only problem with which to contend, but that the idea of separate causes resulting in separate subtypes of dyslexia is even more improbable. They suggest a logical structure for the explanation of dyslexia and its myriad of associated symptoms, in which one primary cause results firstly in cognitive or, even physical, abnormalities (regularly mistaken as primary causes themselves), and secondly (and consequently) surface symptoms of dyslexia. These intervening abnormalities, to which Heaton and Winterson refer as "false causes", include such things as short-term memory problems, perceptual problems, maturation delay and eye-movement problems. Thus, their model resembles that laid out below (Figure 1).

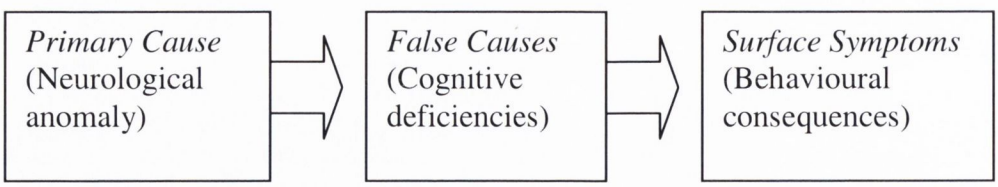

Figure 1: The structure of "explanatory causes" for dyslexia. (Heaton and Winterson, 1996).

The "three level framework" (Figure 2) suggested by Frith (1999; 2001), acts as a map for the causal modelling of disorders such as dyslexia. It allows for the compatible, co-existence of different causal theories, feeding into each other from the Environmental, Biological, Cognitive and Behavioural levels. In this way, theories need not necessarily be mutually exclusive or contradictory.

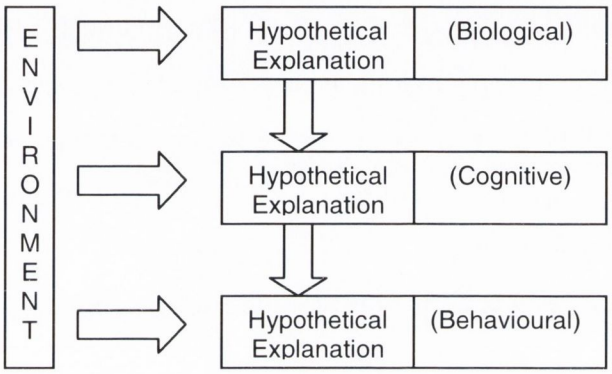

Figure 2: Frith's three-level framework (Frith, 2001, p. 557).

Frith (2001) claims that disorders, such as dyslexia, should be defined not just at the behavioural level but at all three levels for a coherent explanatory theory. Tangible and meaningful observations, such as genetic anomalies and reading / spelling problems, may go at the biological and behavioural levels, with theories at the cognitive level. Environmental factors may act on all three levels. For example, environmental factors are not limited to the cognitive level where teaching, cultural attitudes or socio-economic factors influence grapheme-phoneme knowledge, but may also influence the biological level where our neuro-anatomy, determined by cell migration during early fetal development, can be affected by hormones, drugs, stress, or infection. To endorse biological explanations, therefore, is not to exclude environmental determinants.

Here, three main causal theories for dyslexia will be introduced, which have biological, cognitive and behavioural elements; the phonological deficit hypothesis (i.e., a cognitive / behavioural level hypothetical explanation); the magnocellular deficit hypothesis, and the temporal processing deficit hypothesis (biological / cognitive level hypothetical explanation). The final hypothesis, the cerebellar deficit hypothesis (biological level), is presented in a later chapter.

2.2 Phonological Processing Deficit Hypothesis

Despite differing views of dyslexia and its causes, the existence of a phonological difficulty is rarely disputed (Ramus et al., 2003b). Phonological processing involves the analysis of spoken words into speech sounds and the ability to link these to graphemic symbols. Most studies find that phonological skills are impaired in dyslexics (e.g., Ramus et al., 2003). According to the originators and firm supporters of the phonological core deficit hypothesis (e.g., Snowling, 1987; Stanovich, 1988), the deficit is a reduced sensitivity to the phonological structure of language (Hagtvet, 1997), thereby affecting the ability to manipulate the sounds of spoken words both consciously and unconsciously (Castles and Coltheart, 2003), with the eventual result being grapheme-phoneme correspondence difficulties.

There is an abundance of evidence in the literature to support the idea of a phonological deficit in dyslexics.

Cross-sectional studies have long demonstrated the correlational relationship between phonological awareness and reading problems in dyslexics. Bruck (1990) replicated previous studies (e.g., Bryant and Bradley, 1978; Snowling, 1971) when she found that dyslexic children failed to show age appropriate levels of phoneme awareness; she also found that this persisted into adulthood despite improvements in word recognition, and attributed it to problems in the use of orthographic information. Similarly, Pratt and Brady (1988) demonstrated significant differences between dyslexics' and non-dyslexics' ability to manipulate phonemes, using both children and adult groups. This was independent of IQ and was not due to developmental delay or inadequate teaching. More recent studies show similar results (e.g., Catts et al., 2002 in tests of phoneme deletion).

Neurological studies have provided contradictory evidence of neuro-anatomical a/symmetry differences between dyslexics and non-dyslexics when engaged in phonological tasks (see Habib, 2000) and must be cautiously interpreted. For example, Galaburda et al. (1985) showed evidence of Planum Temporale (PT) symmetry in their dyslexic sample; similarly, using Magnetic Resonance Imaging (MRI), Larsen et al. (1990) demonstrated that, of their dyslexic sample, all those with phonological deficits showed symmetry in the Plana Temporale compared to just 30% of controls. However, reviews of the literature (e.g., Eckert and Leonard, 2000) have generally concluded that PT symmetry is probably not indicative of dyslexia specifically; if it does discriminate

between normal and abnormal language behaviours, these are more likely to represent oral language deficits rather than written language deficits (Plante et al., 1991). Leonard et al. (1993) and Green et al. (1999) suggest that there may be anomalous asymmetries extending beyond the PT. For example, within the left hemisphere, the PT plus the Planum Parietale (PP), referred to as the PT+ (or Caudal Infrasylvian Surface – cIS), has been shown to have leftward asymmetry in more than half the dyslexic sample. In addition to which, dyslexics and non-dyslexics have been shown to have proportional differences of the PP within the PT+ (Eckert and Leonard, 2000) and different surface areas of the PT+ (Green et al., 1999). In addition to the lack of consistency with regards to what constitutes the planum and the measurements used, is the concern that apparent differences between dyslexics and controls may be an artefact of averaging across brains with different left / right asymmetries (Beaton, 2002).

Longitudinal and intervention studies are primarily responsible for suggesting a causal link between phonological deficits and dyslexia. Many studies have demonstrated early phonological deficits to be good predictors of those who develop reading problems (Passenger et al., 2000; Hagtvet, 1997; Scarborough, 1990; Bradley and Bryant, 1983). Scarborough's young participants from dyslexic families produced more phonological errors in spontaneous speech at 30 months (as evidenced in pronunciation accuracy) and low phonemic awareness at age 5 years than did children from normal families. Of those in the risk families, identified by her as having pre-literacy problems, 65% went on to develop reading problems (compared to just 5% in the normal families). Hagtvet's study showed phonemic awareness to be the single strongest predictor of reading at age 8-9 years and Bradley and Bryant (1983) showed sound categorisation (i.e., rhyme and alliteration) to be a powerful predictor.

Following work by the likes of Kuhl (2004) who helped map the timeline of language development in infants, studies using even younger children have been possible. "At risk" children from dyslexic families (i.e., 6 months old) have been shown to take significantly longer than control infants in speech perception of long sounds, with the former taking significantly longer to categorise such sounds than the latter (Richardson et al., 2003). Temporal differences at this preverbal stage, were also found by Paavo et al. (2002), who found that such children differed from controls both in initial and change-detection responses, when measured using event-related brain responses to consonant duration changes of pseudowords (using both normal and rare duration changes).

Intervention studies have claimed to test the causal link through assessing the efficacy of phonological training programmes on reading skills over the years. The best of these, according to MacMillan's (2002) thorough review of the literature, were those by Santa and Hoien (1999), Greaney et al. (1997), Greaney and Tunmer (1996) and Lundberg et al. (1988). The first two of these focused explicitly on children with, or at risk of, reading disability; the former concentrating on phonological skills involving word sort techniques[9] and the latter on systematic strategy training in the use of rime spelling units. Each reported statistically significant achievement gains compared to control or alternative methods. A more recent study with dyslexia "at-risk" children, conducted by Elbro and Petersen (2004), also claimed that their phoneme awareness programme yielded significantly better word-reading results than did the control conditions.

Even within the literature supporting the idea of a core phonological deficit, however, there exist disparate theories as to the core phonological impairment underlying this grapheme-phoneme correspondence difficulty. The deficit may be due to a problem decomposing spoken words (i.e., an even more specific meta-phonological problem) or, alternatively, it may be due to a deficit in speech representation or the ability to create and manipulate speech. The range of impairments has included problems with phoneme, onset, rime and syllable awareness (Bruck, 1992), as well as receptive vocabulary, object naming, syntax comprehension, syntax production, phonological production, syntactic awareness, syllabic awareness, rhyme awareness and phonemic awareness (in Scarborough, 1990). Others (Vellutino, 1979; Catts et al., 1999) claim that there is a more general linguistic deficiency.

Hagtvet (1997, 2003), however, is cautious of the search for "unique" predictors of reading skill, and stresses the importance of interrelationships between different language skills (e.g., syntactic and semantic variables as well as phonological ones), thereby supporting the idea of a more general language problem. Hagtvet's own study reflected findings of Bradely and Bryant (1983) and Scarborough (1990), which demonstrated that although phonological difficulties were indeed powerful predictors of reading development at the group level, they were less so at the individual level, requiring a composite of linguistic variables for more reliable prediction. Hagtvet

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[9] Word Sort: A phonics approach to learning in which children categorise words on the basis of shared phonological, spelling and meaning components (Joseph, 2002).

suggests the approach taken by Tunmer et al. (1984) which considered the broader role of cognitive-linguistic deficiencies and their relationship to reading abilities. In Scarborough's study, for example, different patterns of difficulties were found at different ages. In addition to phonological errors there was evidence of poorly developed syntax (30 months), impaired receptive and expressive vocabulary (42 months) and poor active vocabulary (5 years). Furthermore, Lewis et al. also concluded in their longitudinal study, that children with phonological and other language problems performed more poorly later on in various measures of reading, than did those just with phonological difficulties. In short, there was evidence of language problems outside of phonology. In addition to this, MacMillan's (2002) review concludes that none of the claims linking early rhyme to reading employ a sufficiently strong methodology to support their claims. A similar review by Castles and Coltheart (2003) also failed to find strong support for a causal relationship between phonological awareness and reading. A recent review by Vellutino et al. (2004), however, despite acknowledging that more general language deficits such as visual, semantic and syntactic deficits may be linked to reading disorders in some children, maintained that the evidence for phonological coding deficits was still the strongest available with regards to a causal theory of dyslexia. Yet other studies have determined that, at the very least, there is a reciprocal relationship between phonological awareness and reading ability, such that acquisition of the former increases exposure to, awareness of and performance on, the latter (e.g., Perfetti et al., 1987; Wagner et al, 1994), although this has been refuted by Bruck (1990) who found no evidence of improvement in adults. In addition, the phonological theory of dyslexia does nothing to explain the other deficits and characteristics of dyslexia (i.e., sensory and motor deficits) and indeed it has been suggested that the theory is a tautology rather than an explanation of the problem (Ramus, 2003). It is quite possible, that a third factor, such as general learning parameter, as suggested by Byrne et al. (2000) can influence the development of both reading and phonological awareness. Such conclusions can only support the need for a more global and methodologically rigorous assessment of skills.

2.3 Visual Deficits and the Magnocellular Deficit Hypothesis

Despite the support received by the phonological deficit theory, there are those who believe in a more fundamental deficit. The blurring and moving of letters and words seems to be a common complaint of the dyslexic, and, as such, has stimulated much

exploration of visual aetiology. The idea that dyslexia was caused by a perceptual deficit was dominant in the first half of the last century (e.g., Hinshelwood, 1917; Orton, 1937, in Dockrell and McShane, 1993). More recently theories of visual processing deficits (e.g., Lovegrove et al., 1980) have elaborated upon this idea. Lovegrove found differences between dyslexics and non-dyslexics on tasks of visual acuity, with dyslexics showing reduced contrast sensitivity at low spatial frequencies and luminance. It was not until later, however, that the Magnocellular Deficit Hypothesis (Stein and Walsh, 1997) attempted to explain why this might be.

The magnocellular stream, (M-stream) is one of the two functionally separate visual pathways running from the retina of the eye, through the lateral geniculate nucleus (LGN), the striate occipital cortex, the superior colliculus, the parietal cortex (involved in visual attention) and culminating in the cerebellum (involved in eye and limb movements). The striate cortex (i.e., the primary visual cortex) also projects to the association cortex of areas 18 and 19, which are connected to parts of the brain responsible for memory, pattern recognition, language and symbols. The M-stream consists of large cells and responds primarily to form (gross blobs and shapes), movement (flickering), depth, and small brightness differences. It processes visual information which is fast and transient, with high contrast sensitivity and low spatial selectivity (Livingstone et al., 1991). The parvocellular stream (P-stream), on the other hand, feeds into the temporal cortex and is responsible for the perception of finer detail, colour and high contrast but the cells are smaller than those of the M-stream and conduct information more slowly. Thus, the streams provide a regional functional neuro-anatomical organisation for the earlier visual processing theories (Eden et al., 1996). Visual processing consists of fixations (sustained activity) and rapid movements (transient activity); sustained activity is controlled by the parvocellular system and inhibited by the magnocellular system, whereas transient activity is controlled by the magnocellular system and inhibited by the parvocellular system.

The fine time balance between each of two fixations, referred to as "meta-contrast" by Breitmeyer (1989), consists of sustained and transient actions of the visual system (belonging to the parvo- and magno-cellular systems respectively). An appropriate delay allows the faster transient channel to suppress (inhibit) the sustained channel's prior fixation so that a new image may be processed – a term commonly referred to as "saccadic suppression". If the two fixations occur too close or too far from each other then either the subsequent fixation is of no use as an informative signal

(i.e., prime) from the transient to the sustained system or the sustained system retains the retinal information of the prior fixation (noise), creating confusion for the subsequent sustained system (Breitmeyer, 1989; Lovegrove, 1990). Thus, an impaired transient channel would interfere with saccadic suppression resulting in an unstable visual world (Breitmeyer, 1989). This would be particularly troublesome in a high saccadic activity such as reading. "Dyslexia" could be explained as the fusing of rapidly successive stimuli into one single presentation (Livingstone et al, 1985) and this could exacerbate the poor reading skills of those who may already have a difficulty with coding individual words (Borsting et al, 1996). Such is one explanation for the magnocellular deficit theory of dyslexia. Alternatively, Stein (2001) maintains that utrocular control is impaired in dyslexics. This means the motion signals of dyslexics' eyes are not sent back to the eye muscles properly, and as such, it is difficult to maintain binocular fixation upon targets. This results in an apparent movement of the stimuli which is a particular problem for reading when the eyes are converged at 30cm.

Breitmeyer (1989) suggested long ago that deficits at the early level of the visual pathways probably reflected deficits at higher levels of the cortical M-stream. Areas responsible for language and speech, which are important in reading, lie near the recipient end of this stream (i.e., the Medial Temporal (MT) area). Stein and Walsh (1997) and Livingstone et al. (1995) also suggest that the parietal cortex may magnify the magnocellular distortions of the LGN, thereby relating the differences observed in laboratory conditions to the field of interest (i.e., reading). The experimental research suggests a correlational link between at least some dyslexic groups and visuo-motor problems. Fischer et al. (1993), Biscaldi et al. (1994), Eden et al. (1994) and Biscaldi et al. (1998) have all investigated eye movements as a basis for discriminating between dyslexics and controls. The main differentiating variable across these studies seems to suggest that dyslexics have more problems with saccadic control, evidenced by significantly more regressive saccades, slower Saccadic Reaction Times (SRTs), the number of late saccades, poor smooth-pursuit etc. More specifically, psychophysical studies have investigated contrast sensitivity to link these differences in poor readers to the magnocellular system. Lovegrove et al. (1980; 1982) and Martin and Lovegrove (1984; 1987) found reduced sensitivity to low-spatial-frequency stimuli in poor readers and Borsting et al. (1996) claimed that those "dysphoneidetic" dyslexics who evidenced problems in word analysis and synthesis skills (i.e., grapheme-phoneme problems) could be distinguished on the basis of their poor sensitivity to low-spatial-

frequencies. Finally, although Sperling et al. (2003) supported the findings of Borsting et al. (1996) in that a sub-group of dyslexics showed evidence of magnocellular deficits, they correlated this to orthographic ability and not phonological ability. However, their defence of an M-system deficit is clear, particularly because they eliminated the potential confounding effects of the P-system by isolating the deficient tasks to the M-system[10]. Because the M-stream is responsible for motion sensitivity and consequently stable binocular fixation, this may allow for the development of adequate orthographic skills (Stein, 2001) and is a theory consistent with the findings of Sperling et al. (2003) who linked M-processing with orthographic skill.

Independent of this research, Livingstone et al. (1991) found neuro-anatomical differences between the dyslexic and controls' brains, with magnocells in dyslexics measuring 20% smaller than in controls. In addition, their electrophysiological evidence showed dyslexics to have reduced Visually Evoked Potentials (VEPs) in response to low contrast visual information, compared to controls. Examining the same brains, Galaburda et al. (1994) reported fewer large and more small cells in the left side of dyslexic than non-dyslexic brains which could support the idea of a transient system deficiency. They conjectured that, in turn, auditory system impairment on the left side could interfere with sufficient phonological acquisition for smooth literacy skills. In addition, Finch et al. (2002) found evidence of increased purkinje cell areas in both the anterior and medial posterior cerebxellar cortex, and in the inferior olive of Galaburda et al.'s (1985; 1994) dyslexic sample who had more large neurons and fewer small neurons compared to controls. They point out that this could be suggestive of a deficiency in the input to the cerebellum (which does after all constitute the end site of the magnocellular pathway).

However, the evidence is equivocal. Livingstone's VEP study was not replicated in a subsequent study by Victor et al. (1991). The early findings of Eden et al. (1994) showed that although these visual abnormalities did indeed discriminate

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[10] They did this by modifying a psychophysical paradigm based on that designed by Rogers-Ramachandran and Ramachandran (1998) which stimulates both the magno- and parvo-cellular systems separately. This study used 2 sets of adjacent fields, composed of black and white dots, on which a shape (e.g., heart) was perceptible. By reversing the polarity in counter-phase (i.e., flickering and alternating the colours) at high speeds they could measure the magnocellular system when using black and white colours for the shapes, and measure the parvocellular system when using read and green colours. In this way, Sperling et al. (2003) could differentiate individual and group performance on one system from performance on the other system, thereby, pinpointing a difference specifically to the magno-, and not the parvo-, cellular system. Other studies did not always do this.

between dyslexics and non-dyslexics, they did not discriminate between dyslexics and other poor-readers. Also, as Biscaldi et al. (2000) point out, there are other explanations for the existence of abnormal eye-movements than the magnocellular deficit hypothesis (e.g., an impairment in the superior colliculus or a problem with the neural substrates that control spatial attention). Even more boldly, Skottun (1997; 1999; 2001; 2005) claims that within the visual system there is currently no evidence to support the theory that it is the magnocellular system which is primarily responsible for the dyslexic's visual processing problems (this, he claims, is only a speculative assumption at present). Studies thus far have been correlational only, and in fact, if anything, it is more likely to be a suppression of the parvocellular system than the magnocellular system which is the principal deficiency in dyslexics' visual processing. Cornelissen et al. (1995) also suggest that dyslexics' reduced sensitivity to luminance contrast could just as equally be due to abnormal P pathway input as to M pathway input. Their research has demonstrated no (or little) reduction in high-spatial-frequency (i.e., parvocellular) information during saccades, which would have been expected given its supposed suppression, but rather, a reduction in sensitivity to both low-spatial-frequency information (e.g., Burr et al., 1994) and non-colour stimuli (Volkman et al, 1986), implicating a suppression of the magnocellular system rather than the parvocellular system in *normal* visual processing, thereby undermining the very basis of the magnocellular deficit theory. Backing this up, Gross-Glenn et al. (1995) could find no evidence of low-spatial-frequency differences between the groups of dyslexics and controls. Amitay et al. (2002) tested dyslexics on dissociated M- and P-stream tasks to discover whether those with magnocellular deficits were specifically impaired in this system. Although previous studies had found evidence for magnocellular deficient sub-groups of dyslexia (e.g., Borsting et al., 1996) Amitay et al. found no such evidence for any such sub-group, showing that those who performed badly on magnocellular tests performed badly on all perceptual tasks.

Furthermore, Stein and Walsh (1997) also point out that although the M-stream does not have an anatomically distinct auditory pathway, there is an auditory subsystem which analyses acoustic transients in much the same way that the visual information is processed. Because phonemic discrimination depends upon rapid auditory transitions, this may also contribute to linguistic (i.e., phonological) problems (Livingstone et al., 1991). In addition, the cerebellum, responsible for motor co-ordination, receives information from the magnocellular system. Far from being just a theory of visual

processing dysfunction, therefore, Stein and Walsh (1997) seem to be merging the magnocellular hypothesis into a general timing hypothesis (Stein and Walsh, 1997). This is wise given that the literature generally criticises the theory on the basis that it is overly focused on the visual aspect of a more global sensory impairment in the ability to process brief and rapidly emitted stimuli (e.g., Amitay et al., 2002). Cornelissen et al. (1995), for example, suggests that dyslexics' poor ability to detect coherent motion could reflect deficiencies in the parietal visual pathway (e.g., MT or MST). This moves the focus of a visual deficit away from the magnocellular system and suggests that a more general theory of temporal deficit may do better in explaining the disorder; one which attributes phonological difficulties to a problem of hearing sounds and allows for the independent existence of a visually based reading deficit. It may also do better as a unifying theory of dyslexia, given its ability to account for phonological, visual, auditory and motor deficits of the disorder.

Miles (1994) suggested the need to develop a taxonomy with regard to dyslexia, that allowed for the classification of the disparate symptoms based on a causal theory. In a similar vein, Fawcett and Nicolson (2001) point to the typical movement of scientific explanation from descriptive (as above) to explanatory theory. The following theories (the temporal processing theory of dyslexia and the cerebellar deficit hypothesis (CDH)) are two such causal theories (although discussion of the CDH is presented in a later chapter).

2.4 The Temporal Processing Deficit Hypothesis

The Temporal Processing theory and the Magnocellular theory have very similar premises, the main difference being that the latter restricts its deficit to the M-stream, whereas the former can be applied to all neural systems which engage in rapid temporal processing. The notion of a temporal processing deficit was first proposed by Zurif and Carson (1970) when investigating cross-modal and intra-modal skills of dyslexic versus control children. Tallal and Piercy (1974) extended this, suggesting a general, rapid frequency changing deficit in language-impaired children. The poor readers of their aphasic participants had significantly more problems than did control children in processing fast information (i.e., Temporal Order Judgement tasks [TOJ]) where interstimulus intervals were short (i.e., 20-40ms). This poor perception of rapidly following signals is just one way this deficit can manifest (Mody et al., 1997). Such

children can also have difficulty perceiving brief signals and if the two are combined, as is the case in consonant formant transitions followed by a vowel (e.g., /da/); they are, as Mody et al. put it, "doubly disadvantaged". With regard to reading-disabled children specifically, Tallal (1980) suggested that their poor phonics skills could be specifically due to a problem affecting the rate at which they can process information, rather than just a temporal *order* deficit and certainly rather than a specific *verbal* deficit. These auditory perceptual deficits relate to the difficulty in learning the sounds-symbol relationships essential for adequate phonics skills. This is ultimately responsible for problems in learning to read because such children lack the acoustic cues necessary to discriminate the temporal order of brief (i.e., milliseconds) linguistic events (Witton et al., 1998; Temple et al., 2000). Kujala et al. (2000) also found that dyslexics had particular difficulty discriminating temporal non-linguistic sound features surrounded by other sounds, as is the case with phonemes in words.

As would be expected, studies are not limited to the auditory domain; language-impaired children are also more likely to show deficiencies in other sensory domains compared to controls. Zurif and Carson's (1970) early study showed dyslexics performing significantly worse than controls in auditory, visual and cross-modal domains. The tactile system is also reputedly mildly deficient in dyslexics (e.g., Stoodley et al., 2000; Grant et al., 1999). In addition to finding differences between dyslexic and control samples, within group studies have also found processing deficits of rapidly transient stimuli across modalities (e.g., Van Ingelghem et al., 2001). Witton et al. (1998), for example, showed that low frequency FM sensitivity not only correlated highly with dynamic visual-motion sensitivity, but that together they also both predicted significant amounts of the variance in phonological skill (i.e., non-word reading), for both dyslexic and non-dyslexic samples. Laasonen et al. (2001) found congruous results in their tests of uni-modal and cross-modal temporal processing acuity and temporal order judgement tasks, when they compared dyslexics to controls. This applied throughout the lifespan and was also related to phonological processing.

Thus, the temporal processing deficit hypothesis not only explains problems with reading but also problems with spelling, because the accurate encoding of sound guides not just reading, but letter position which is also guided by efficient visual temporal processing (Witton et al., 1998). This led to the conclusion that the brain is wired, not for language specifically, but for the processing of temporally fast information.

Using the antibody (CAT-301) McGuire et al. (1989) stained and traced a distinctively similar population of neurones which included the magnocells of the LGN but also parts of the motor cortex, pre-motor cortex and supplementary motor cortex of the frontal lobe as well as throughout most of the parietal cortex. The common feature of these neurones is their heavy myelination which allows for the rapid conduction of axon potentials, and as such, the accurate coding and transmission of rapidly changing, dynamic stimuli (Tallal, 2000). These areas are all interconnected and are responsible for somatic and ocular movements, somatosensory function and visual motion (McGuire et al., 1989). It has been suggested that because both dyslexia and auto-antibody production have been traced back to the same chromosome (i.e., chromosome 6; Cardon et al., 1994), that a form of auto-immune disorder, which might attack the myelin of rapid processing cells, could explain why such cells appear smaller in dyslexic samples and also why allergies (e.g., asthma) are a common co-morbid problem with dyslexia (for a review of this see Stein, 2001). It is known that myelin formation develops over time, with different functional systems developing together (Flechsig, 1927). Thus, an auto-immune attack on myelin may not necessarily affect all myelinated cells but could be restricted to some functional area. This is often seen in Multiple Sclerosis (MS), one of the most common forms of autoimmune attack on the myelin sheath, where the motor or visual cortex is usually affected.

Further support for the theory comes from studies looking at training exercises designed to improve temporal processing in language-impaired children. One classic study by Merzenich et al. (1996) used computer game-based training exercises, which involved perceptual identification of sound sequences, and phonetic recognition of target position where sound frequency changes were slowed and amplitude changes increased. They found significant improvements for all children between pre- and post-training performance on the Tallal repetition test (a test of temporal processing ability) with a large reduction in both interstimulus interval and tone duration, both of which showed dyslexics to be approaching normal levels of performance. Looking at reading-impaired children specifically, Kujala et al. (2001) also made use of audio-visual programmes in the attempt to improve dyslexics' processing skills. A controlled trial revealed significant improvements in the experimental group on behavioural measures including, not just faster response-times to non-verbal sounds, but also on reading skills, and this was correlated with functional neural changes also. In this case, the acoustic training programme did not make use of any linguistic stimuli, providing

further evidence of the important role of non-speech representations in language processing (Kujala et al., 2001).

Recent experimental and fMRI studies (e.g., De Martino et al, 2001; Temple et al, 2000; Belin et al, 1998) show support for the temporal processing theory. De Martino et al. (2001) demonstrated that (verbal) stimulus order and brevity differentiated between dyslexics and non-dyslexics (though brevity may have been confounded by syllabic complexity) and these were correlated with phonological skill. No differentiating effect of inter-stimulus duration was found. However, a further study, using rapid non-verbal acoustic stimuli, designed to mimic the structure of speech syllables, has also investigated, and found, differences between such groups (i.e., Temple et al., 2000) and has functionally linked the stimulus processing to the left prefrontal cortex (i.e., Brodmann areas 46/10/9 along the middle and superior frontal gyri). Consequently it is suggested that this area may be involved in reading skills. Activation of this area improved in dyslexics once they were given training in rapid auditory discrimination although whether this was correlated with a change in reading skills directly does not seem to have been explored. This prefrontal area was also implemented in rapid (but not slower) acoustic processing by Belin et al. (1998) and although Temple et al. did not show activation in the auditory regions of the temporal cortex, Belin et al. did find bilateral temporal activation of the auditory cortex which, in addition to stimulating part of the prefrontal cortex, was asymmetrically larger in the left hemisphere when stimuli were rapid. The two areas have also been linked in neuro-anatomical studies of the rhesus monkey. For example, Romanski et al. (1999) found that different streams of auditory information targeted different areas of the prefrontal cortex and suggested that these represented distinct functions; furthermore they suggested that temporal- and frequency- related information represented earlier, more crude information, reaching the prefrontal cortex before the arrival of more complex information from higher associative auditory areas, much as the parvo- and magno-cellular systems in the visual cortex are responsible for distinct information and are thought to arrive at shared destinations at different times. Kujala et al.'s (2000) study also suggested that the information processing deficiency in dyslexics occurred at the level of pre-attentive analysis of the acoustic features. Thus, there seems to be dissociation between verbal and temporal processing, with temporal processing occurring at an earlier or pre-linguistic stage of speech (Belin et al., 1988).

Much of the criticism of the temporal processing theory concerns how accurately the underlying impairment has in fact been identified. Studdert-Kennedy et al. (1994; in Miles and Miles, 1999) make two points relating to this. Firstly, Tallal and others, when testing, either with verbal or non-verbal acoustic tones to represent phonemes, have presumed that these phonemes are distinct units, without any consideration of "co-articulation" (i.e., that process whereby the initial consonant anticipates the vowel even when the consonant is being produced (Fitch et al., 1997) such that they blend into each other rather than being distinct). Secondly, it has been suggested that it is not temporal order but rapid discrimination of similar stimuli which is difficult for such children This was a confounding variable in many of the studies which made use of consonant-vowel syllables such as /ba/ and /da/ to test children's discriminating skills. Only when the syllables were phonetically similar did the children have difficulty with reporting order. Thus, Mody et al. (1997) suggest that the deficits found in such children are neither general nor temporal, but speech-specific.

Despite finding support for Tallal's rapid temporal order processing deficiency, Reed (1989) questions Tallal and Piercy's (1974) study which found deficiencies of language-impaired children in verbal and non-verbal, same-different judgement tasks where temporal order was not even a factor. She suggests that a more basic perceptual deficit could explain not just rapid temporal deficiencies, but also the other deficiencies evidenced by her reading-disabled group (e.g., word identification, sharpness of phonological category boundaries). Similarly, Mody et al. elaborate on Tallal's (1980) own declaration that her sample's deficiencies may not be due to auditory temporal perception, but to a deficit at a more basic perceptual level. Both Mody et al. and Bishop et al. (1999) suggest that the deficit lies in the correct identification of rapid tones, rather than the identification of their order. Nittrouer (1999) was unable to find any deficits in her reading-disabled sample which could be linked to rapid processing or even any difficulty with the consonant formant transitions reputed to be so difficult for this group.

Furthermore, as Reed goes on to point out, the current deficit is not universally found in reading-disabled children, a fact commented upon in many other similar studies (e.g., Laasonen et al., 2001). Because not every dyslexic reader is impaired on tasks of temporal acuity this cannot be a sufficient causal explanation for the disorder. Bishop et al. (1999) concluded from their study of language-impaired children that whilst auditory temporal deficits may explain part of the problem in some children (i.e.,

those already at genetic risk of developing it), they were neither necessary nor sufficient to cause the problem. In addition, Conlon et al. (2004) found that although their measures of temporal processing did distinguish between good and poor readers, the latter group were not necessarily dyslexic, instead having poor literacy skills generally.

Thus, the temporal processing deficit hypothesis in itself may not provide the comprehensive explanation that it sometimes purports to.

The need for a more general theory, which could integrate all of these ideas into one unified hypothesis is desirable. A major attempt to do this is the cerebellar deficit hypothesis. However, before presenting it, it is necessary to outline some further theoretical issues which may be relevant.

3 Chapter 3: Terms, concepts and normal development

Chapter 4 reviews some of the background and current research which suggests a link between motor abnormalities and impaired cognitive functioning, including dyslexia. Chapter 5 reviews the emergent causal theory of dyslexia, which involves the cerebellum. Before looking at either of these, however, it is necessary to introduce some of the terms and concepts which appear in the motor development literature as this will maximise understanding in the following chapters. An introduction to the importance of the cerebellum is given in terms of the role sensory integration plays on motor development and co-ordination.

3.1 Normal motor development

Motor development can be divided up into three broad stages. The first consists of a repertoire of innate movements or "primitive reflexes". The second consists of a slightly more mature set of reflexes known as "postural reactions". Finally, there is the acquisition of voluntary and controlled movement. This skilled movement, according to Laszlow & Bairstow (1985), allows the child to explore his environment, to interact with it and become physically and socially competent.

On the basis of this development, Gesell (1988) proposed 3 major principles of motor development:

(1) The principle of developmental direction, which suggested that development was proximo-distal in that it proceeded from the mid-line of the body out, and it was cephalo-caudal, in that in proceeded from the head down.

(2) The principle of functional asymmetry, which suggested that although development was largely symmetric, there were some exceptions to this, such as the Asymmetric Tonic Neck Reflex (ATNR) which promoted hand and eye dominance necessary for a richer degree of interaction with the world.

(3) The principle of individuating maturation, which suggested that there is a specific, natural order to development and external influences, such as the environment, are unlikely to affect that.

3.1.1 Primitive reflexes

Primitive reflexes are innate, stereotyped and automatic (and hence involuntary) behavioural responses to sensory stimuli which emerge during fetal and preterm life

(Allen, 1996; Capute et al., 1978). They are immediate in their elicitation, being executed without cortical involvement (Goddard, 1996) and provide the motor co-ordination necessary for survival mechanisms which assist the child in nourishing and protecting itself. They are brain-stem mediated (Zafeiriou, 2004) and as the infant grows older, neurological maturation (in the form of cortical inhibition) allows these reflexes to become integrated into voluntary movements. Thus, although being "hard-wired" they can adjust to a changing environment (Leonard, 1998) with the child adapting them gradually to more complex functions (Fiorentino, 1972).

Each reflex is usually elicited by a particular stimulus which causes certain patterns of extension and flexion (Folio & Fewell, 2000). However, elicitation in this form should not be apparent beyond 6-12 months of life and, if present, usually indicates some kind of central nervous system (CNS) immaturity (Goddard, 1996). This immaturity may result in later developmental dysfunctions because it delays both general development and motor development. Persistent reflexes can lead to deformity or may become a self-perpetuating movement ritual (Holt, 1991). As such those presenting with them should be carefully monitored.

Primitive reflexes can be classified according to whether they are "phasic", in which case they allow for muscle and joint mobility, or "tonic", in which case they allow for stability by affecting the distribution of muscle tone (McCormack & Perrin, 1997); tonic reflexes will last as long as the stimulus lasts (Holt, 1991). Although there are approximately 44 commonly-known reflexes (and Illingworth (1987) reports up to 70), only some of these are explored here. The better known reflexes include those listed in Table 2 (a description of the response and its adaptive purpose is given below; details on specific elicitation used in the current study are given in the "Methods" section).

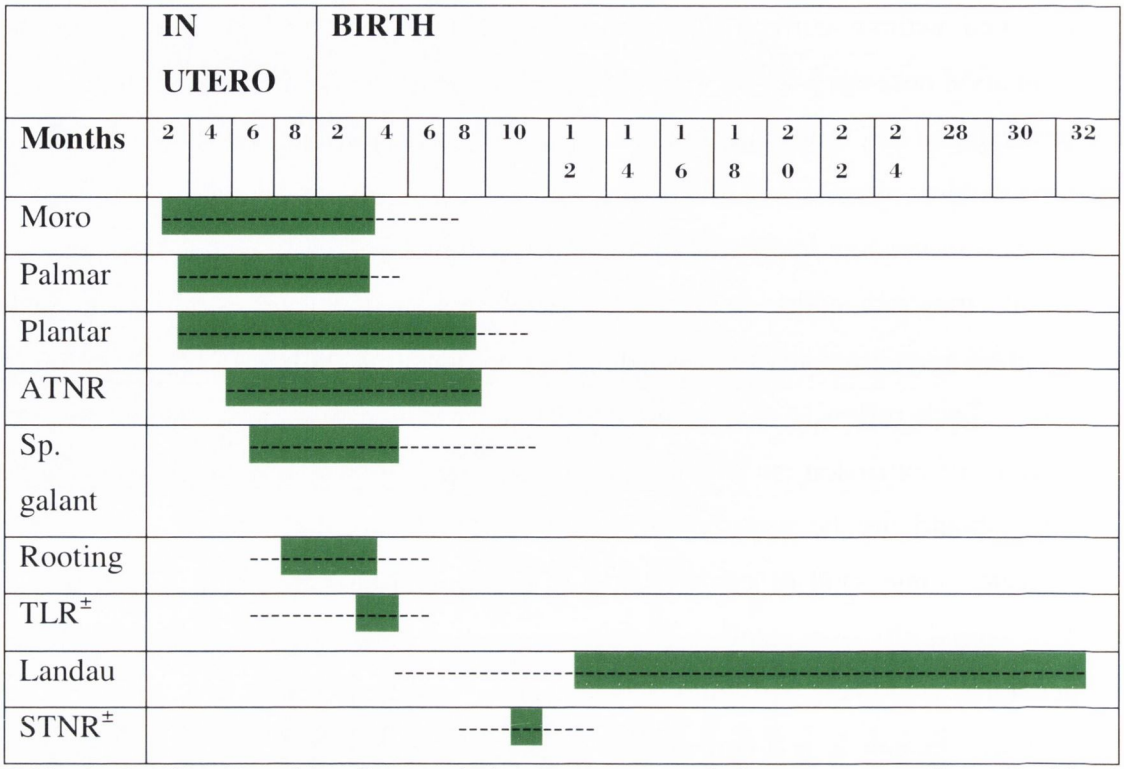

Table 2: Profile of early reflexes; their emergence and disappearance (from Goddard, 1996, p.3).
---- Usual presence of reflex
---- Possible presence of reflex.

The Moro: The Moro is the first reflex to emerge in uterine life, seen at approximately 2 months after conception. It is a protective, phasic reaction usually in response to a sudden stimulus (i.e., noise, light) and representing an early form of "flight or fight" (Goddard, 1996). The abduction (extension) of limbs outwards, accompanied by a deep breath in, is followed by adduction (flexion) of the hands towards the midline and exhalation of the breath (Allen, 1996; McCormack & Perrin, 1997). It may be artificially elicited in the new-born by holding the child upright and allowing the head to fall backwards slightly. This should be inhibited between 2 and 4 months after birth.

± The literature varies in terms of whether the Tonic Labyrinthine Reflex (TLR) and the Symmetric Tonic Neck Reflex (STNR) count as primitive or postural reflexes. The aim of this work is to replicate previous work and explore the claims of commercial centres, not to resolve this argument.

The Palmar: Another protective, but tonic, reaction - the palmar reflex is a form of grasping reflex. It acts as both a protective mechanism (i.e., against falling) and a nourishing one (massaging the mother's breast during feeding). Stimulated by pressure to the palm of the hand, flexor tone causes the fingers to clench together (McCormack & Perrin, 1997). The refined manipulative abilities of the thumb and forefingers may only develop properly once this has been inhibited around 2-3 months after birth.

The Plantar: Similar to the palmar reflex, this reflex is seen when pressure is applied to the sole of the foot. The toes curl inwards, also perhaps in the attempt to secure safety by clinging to the mother. This reflex should be inhibited between 9 and 12 months after birth.

The ATNR: The "Asymmetric Tonic Neck Reflex" is stimulated by turning the head of the infant to one direction. This will cause the limbs on that side to which the head is turned, to extend, with flexion of the opposing limbs (Allen, 1996). Beginning in utero, it stimulates the balance mechanism, something which is reinforced during the birth process as the infant twists its way down the birth canal. It allows the infant to support its own weight against gravity at a non-volitional stage and may contribute to the free flow of air / milk for breathing and feeding purposes. It may be responsible for future laterality development, allowing as it does, for limb dissociation and lateral weight shift (McCormack & Perrin, 1997). Around 1.5 – 2 months after birth, and triggered by the ATNR, infants start to focus on their hand; this continues until they are 4-4.5 months when the fascinations wanes as the reflex becomes inhibited (White et al., 1964), but it is important for establishing short distance visual fixation.

The Spinal Galant: This phasic reflex is present at birth and is elicited by tactile stimulation to one side of the back, causing the hip (and leg) to lift in that direction. It may help in movement down the birth canal (Goddard, 1996).

Rooting: In support of access to nourishment, the rooting reflex is present at birth and is stimulated by tactile contact with the cheek / mouth, which causes both orientation of the mouth towards the source of stimulation and a sucking motion of the mouth. It is inhibited about the fourth month after birth.

TLR: The "Tonic Labyrinthine Reflex" is elicited by movement of the head forwards or backwards. Changes in position are detected by the vestibular system of the inner ear. Movement of the head will stimulate the vestibular system, triggering defensive (i.e., protective) responses to protect from the results of falling. This means that when the head goes forwards, it causes all of the limbs to contract, where-as

movement of the head backwards causes them to extend, as the child's weight is pulled in these relative directions. In the older child who is tested in the standing position, this will be apparent by a step out of place or loss of balance when the head goes back and a bending of the knees when the head goes forwards. The forwards motion continues until the fourth month after birth and the backwards up until the third year of life.

Landau: These final two reflexes are not "true" primitive reflexes because they are not present at birth; they are what Goddard (1996) refers to as "bridge" reflexes because they do not last throughout the life-time. The Landau can be elicited by suspending the infant prone in the air in which case the limbs will extend; retention of this reflex will affect the infant's balance. It is inhibited by three years of age. Both the Landau and the STNR play a part in suppressing the TLR, and will remain persistent themselves if this is not inhibited properly.

STNR: The "Symmetrical Tonic Neck Reflex", like the ATNR, is stimulated by movement of the head in the quadruped position. However, rather than left and right, the head's being extended or flexed causes the body to dissociate into two halves, top from bottom. When the head is extended (above the level of the spine), the arms straighten and the legs contract, where-as when the head is flexed (below the level of the spine) the legs straighten and the arms contract. It helps the infant raise up into a creeping, quadruped position, though it cannot yet move from this position. This reflex only emerges after birth, at about 6-9 months. It is inhibited soon after that, between 9-11 months and is adaptive in that it allows exploration of the ground and whatever may be above the head (Kohen-Raz, 1996).

3.1.2 Postural Reactions

Postural reactions are involved in the maintenance of balance and are necessary for later voluntary movement such as standing and walking. They consist of righting reflexes, in the quadruped position, and equilibrium reflexes, in the bipedal position (Goddard, 1996). They are not generally seen as "true reflexes" given that they usually act as a whole and have input from many sensory systems (joints, tendons, muscles, skin, eyes and labyrinths) (Zafeiriou, 2004). They emerge between 3-12 months after birth, just as the primitive reflexes are becoming less dominant, and are present for life. Their function is in helping the infant adjust its new bodily movements (such as sitting-up and crawling) to gravity, space and motion (Morrison, 1985, p.3). The child is then equipped to develop more and more complex postural skills. The sensory systems

which allow for the development of voluntary postural control are the vestibular, proprioceptive and visual systems. These shall be explained more fully presently.

3.1.3 Voluntary Movement

Although there are a multitude of infant behaviours which appear between birth (where movement has hitherto been described in terms of reflexes) and perfect, adult control of motor skills, only those relevant to the current study are presented. As such, those motor skills, mastered by children between the ages of 3 and 7, shall be described. The kinds of movements evidenced tend to fall into one of two categories; static or dynamic tasks. Static balance is defined as the ability to maintain a stationary position, for a specified period of time - the centre of gravity maintains the base of support (Thomas et al., 1988, p. 81-82). Dynamic balance is defined as the ability to maintain a balanced position, while moving through space – the centre of gravity is shifting constantly to remain inside the base of support (Knight & Rizzuto, 1993, p.1296).

Each stage of motor development is built upon previous patterns of movement, which are then elaborated to make more complex movements (Fiorentino, 1972). Gabbard (2000) divides motor development into a continuum of skills where different phases of ability relate to different chronological stages. "Reflexive" and "rudimentary movements" belong to the "prenatal" and "infancy stages", which last approximately 2 years. Following this is the fundamental movement stage, appropriate for those between the ages of 2 and 6 years (i.e., the "early childhood" stage). This is then followed by "sport skill", "growth & refinement", "peak performance" and "regression", which correspond to the stages of later childhood (i.e., 6-12 years), adolescence (i.e., 12-18 years), adulthood (18-30 years) and older adulthood (70+ years). It is on the "early childhood" stage that this study focuses. Table 3 gives a brief overview of the development of motor tasks in children.

TASK	AGE					
	5-6 months	6-7 months	10-11 months	1-4 years	4-5 years	5-6 Years
Rolling (front-back)	✓					
Rolling (back-front)		✓				
Sitting		✓				
Crawling			✓			
Walking				✓		
Running				✓		
1-leg balance					✓	
Jumps forward					✓	
Walks on a beam					✓	
Skipping						✓
Hopping						✓
Spring jump						✓
Stands on left & right leg						✓

Table 3: The acquisition of "early age" motor skills (Holle, 1976; Gabbard, 2000).

Fiorentino (1963) believed in a similar progression of motor development, with each stage controlled by a different level of the brain (Figure 3). The reflexive, apedal, stage, for example, she believed to be controlled by the spinal cord and brain-stem; the quadrupedal righting reactions by the mid-brain (e.g., crawling and sitting) and the bipedal equilibrium reactions by the cortical levels of the brain (e.g., standing and walking) (Fiorentino, 1963, p.5). These translate roughly to the 3 stages of primitive

reflexes, postural reactions and perfected, voluntary movement. It is the first and the latter of these which are of interest in the current study. Although this is now a somewhat outdated idea of motor development and motor control, it is representative of the hierarchical theories popular at the time (see the ideas of Doman and Delacato in section 4.3 and 4.4). A more inclusive theory suggesting a systems model is now proposed (see Woollacott and Shumway-Cook, 1990, p.800 for a summary). This focuses less on passive reactions and more on active movements, and compares the body to a mechanical system subject to gravitational forces in which the nervous system is part of complex system and motor programs may give different movements depending on the body's position.

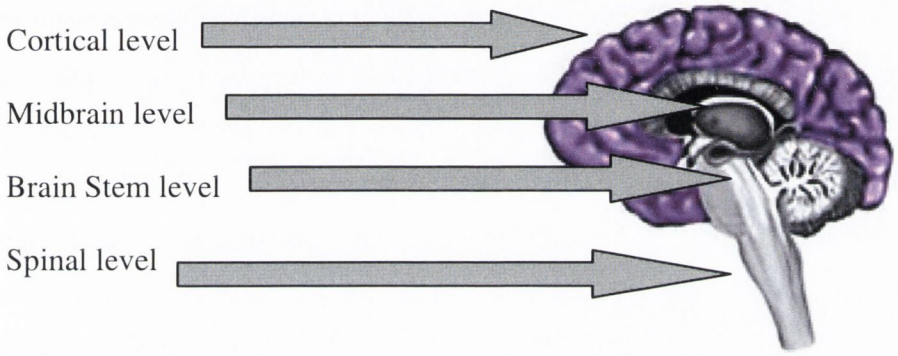

Figure 3: Fiorentino's hierarchical theory of motor development. This suggested that increasingly complex movements corresponded to increasingly sophisticated levels of the brain.

3.2 Retained reflexes

"Retained reflexes", also known as "primitive" or "dominant" reflexes, have been interpreted by some authors as contributing to motor abnormalities either because they prevent the development of higher order reflexes or because higher order reflexes fail to inhibit lower ones. Thus, early reflex theory suggested that if primitive reflexes were not integrated into functional behaviours they would disrupt later motor development. As such, this led to the belief that prolonged, or re-emerged primitive reflexes, after a certain age, were both abnormal and pathological (Miyahara & Reynders, 2003). According to Goddard (1996) they are likely to affect the subsequent normal development of the child, not just in terms of motor skill but in terms of other skills that are based upon, or derive from, adequate motor functioning (i.e., perceptual and cognitive skills). The view of those such as deQuiros & Schrager (1978), and Ayres

(1979), have suggested that "postural autonomy" (i.e., the freedom from the pull of gravity) allows for the development of external and internal schemes of spatial reference, thereby permitting exploration and awareness of obstacles. Berthenthal & Campos (1990) also believed that crawling was the starting point for a developing world of spatial cognition and emotional development, as it afforded exposure and attention to perceptual information (in Thelan, 2000).

The retained reflexes which are of particular interest in the present study (i.e., ATNR, STNR, TLR and Moro) are amongst those Fiorentino (1972) lists as the most likely to contribute to a delay in motor development. A retained TLR, for example, will prevent an infant from lifting its head off the ground and its arms from underneath it and as such will not allow for the development of balance against gravity or facilitate crawling. The child then does not develop stability or a secure reference point in space from which to develop judgements of space and distance. Left from right, up from down and backwards from forwards may be difficult to distinguish from one another. The retained TLR also prevents normal development of other reflexes (i.e., the oculo and labyrinthine head-righting reflexes, without which visual and vestibular information cannot be properly integrated and convergence / re-establishment of binocular vision will be impaired (Goddard-Blythe, 1995). Retained STNR and ATNR will interfere with rolling, crawling and walking skills because the child loses stability on the flexed side. A retained Moro will cause the child to lose balance easily (for example, when they get a surprise or fright).

Of principal interest to educational theory and practice is the possible interference of retained reflexes with literacy acquisition. Goddard (1996) has attempted to explain the kinds of symptoms that result from retained reflexes which may affect school skills; for example, persistence of the Moro reflex makes for a child who is hypersensitive to stimuli. This constant presence of "fight or flight" reactions means he may react either by withdrawing from unsettling situations or by being overly aggressive in them. In terms of learning, he reflects the sensory dysfunctional child, as described by Ayres (1979), in that he is receiving too much sensory stimuli and cannot respond to it appropriately. Neurologically speaking, he is not inhibiting unnecessary stimuli or subsequent reactions (many of the disorders explained by different approaches may in fact be describing the same thing but using different terminology). This will affect his ability to concentrate properly. Goddard describes other long-term effects this may have, including vestibular problems such as motion sickness, visual-

motor / visual-perceptual problems and photosensitivity. She also explains how the "fight or flight" adrenaline response, elicited by the Moro when faced with a bright light, for example, causes the pupils to dilate, which over time causes a conditioned response to any brightness (such as fluorescent lights) (Goddard-Blythe, 1995). These problems may cause the child to have difficulty following letters and words on a page and adjusting to black print on a sharply contrasting white back-ground (and as such, reading and writing). Other problems may include possible auditory confusion leading to poor auditory discrimination (this shall be explored further in chapter 5). Poor co-ordination as a result of vestibular problems makes the child fearful of, and poor at, sport. This child may be often anxious as a result of so many stimuli, which has its own secondary, psychological consequences.

If the palmar reflex persists, fine-motor co-ordination with the hands may be impaired and an inability to make the "pincer grip" will hinder the acquisition of writing and make subsequent neat writing very difficult. This may explain why many dyslexic children show such illegible hand-writing. The plantar grasp, which causes curling inwards of the toes, will interfere with upright stability and walking if retained. This may affect the acquisition of postural stability and subsequent exploration of one's environment as hypothesised by deQuiros & Schrager (1978). A retained ATNR will affect the normal cross-lateral development of creeping, crawling and walking, making the child appear clumsy (these are the children who find it difficult learning to ride a bike or catch a ball). Because of the unilateral nature of this reflex, the child cannot bring both hands together to meet at, or cross, the midline of the body. He can only bring an object close to his face by turning the head in the opposite direction, and thus, cannot examine the object properly which may affect the development of hand-eye coordination as well as pre-academic and play skills (Holt, 1991; McCormack & Perrin, 1997). Unable to pass an object in front of his face from one hand to the other (bilateral movement) also means visual tracking across the midline is untrained or impaired. All of these may affect the child's reading ability, writing ability and attention span (Goddard, 1996). The importance of the ATNR for developing hand-eye coordination was first suggested by Gesell in 1954 (Crutchfield & Barnes, 1993) and supported by Bobath (1980) because it is present during the time that visual fixation upon nearby objects is developing (Holt, 1991). Furthermore, a retained ATNR means that when writing the child's arm will naturally extend away from the copy-book and so muscle tension is required to hold a pencil and write; this results in muscular fatigue,

something commonly reported by dyslexics (McPhillips & Sheehy, 2004). Similar impaired development is seen with a retained spinal gallant reflex, and the two are frequently found together (McCormack & Perrin, 1997). The STNR, if persisting, will prevent the child from creeping, usually causing him to move by shuffling around on his bottom (Portwood, 2003, claimed that 80% of dyslexic infants "bottom-shuffled" instead of crawled). Although the STNR initially trains short distance vision, this is suppressed while long distance vision is developing. However, emergence of the STNR helps to re-establish short-distance vision when focusing on the hands for creeping. In this way, the infant is learning to re-adjust its binocular vision. School-related problems will include an inability to shift focus from the black-board to the page when copying tasks (making the child very slow), as well as the inability to sit comfortably in a chair. The child will sit with legs stretched out so that the arm can bend to write (i.e., "slumped" in the chair), or whose legs are bent double beneath him whilst the arms are stretched out writing. Given the close link between the labyrinthine system, the vestibular nuclei and the eyes, a retained TLR may result in poor sensory integration and thus poor visual-perception and, consequentially, a tendency towards motion sickness. Crawling difficulties mean proprioceptive feedback is also limited and a sense of depth and self in space may be affected. Goddard claims that this will lead to spatial and temporal problems as well as poor sequencing skills. The lack of antigravity extensor control can lead to a forwards or backwards shift of weight making the child appear uncoordinated and clumsy.

Other retained reflexes may also play their part in symptoms seen in the dyslexic child. For example, proper control over swallowing, respiratory and sucking motions allow the child to begin babbling and talking. If retained, articulatory and motor skill difficulties could result in speech and language problems (a common predictor of dyslexia in young children (Hagtvet, 1997).

3.3 Maintenance of balance and posture: The importance of the vestibular, proprioceptive and visual systems

The maintenance of posture is achieved through three major neuro-physiological balance mechanisms: the vestibular system, the proprioceptive system and the visual system. The vestibular system relies on the semi-circular canals and the otoliths of the inner ear; the proprioceptive system relies on sensations received from receptors in the

joints, tendons, muscles and skin and the visual system relies on the retina of the eye and the visual-motor system.

These systems integrate to perform their ultimate functions and do not generally operate in isolation (Howe and Oldham, 1981). For example, the vestibular-ocular reflex is an example of an interaction between the visual and vestibular systems. Ocular nystagmus in one of these reflexes and refers to the involuntary rhythmic eye-movement that occurs when a person spins round then suddenly stops – it is the lingering adjustment of the eyes to tracking the world as it revolves around them.

A defect affecting these systems is likely to produce balance problems for the child. Dysfunctions could lie within one sensory process or within the integrative process (Morrison, 1985, p. 90).

3.3.1 The vestibular system

What it is

The vestibular system is one of the primary peripheral sensory systems contributing to balance (Reber, 1985). It is the first of our sense systems to develop, being operational at 16 weeks after conception and the only sensory system to be fully myelinised at birth (Goddard, 1996). The system provides information about the head position and linear acceleration in space (Umphred et al., 1985) with the main responsibilities of:

- Enabling the body to respond to this information through dynamic and static balance.
- Eye-tracking through maintenance of the fovea on the object of visual fixation.

Other functions to which the vestibular system contributes (according to Goddard, 1996 & Savundra, 1997) include:

- The activity of the autonomic nervous system.
- The level of arousal and mood.
- Motor planning.

How it works

The labyrinth sensory receptors, composed of both the semi-circular canals and otoliths, respond to mechanical responses to any acceleration of the head. The semi-circular canals are responsible for detecting motion, chiefly, changes in direction, including pitch (which refers to neck flexion and extension about a vertical axis), yaw (which refers to right / left head rotation about a vertical axis), and finally roll (which refers to right / left neck side bending about a horizontal axis). The otoliths are responsible for detecting gravity and changes in speed on both the horizontal and vertical planes such as riding in a car or an elevator (Allison & Fuller, 1985).

The vestibular pathway

There are two main vestibular pathways. One is the vestibular-visual-motor pathway which controls eye-movements and the other the vestibulo-spinal tract which controls equilibrium. They start with the labyrinth sensory receptors which stimulate the vestibular nerve (8th cranial nerve). This information is then sent to the cerebellum, where it is integrated with information coming from the visual and somatosensory systems. In response, the cerebellum then accommodates for the appropriate reflexes (e.g., equilibrium reactions), after which the information continues to the central sensory tract and on to the cerebral cortex (Allison & Fuller, 1985).

Its relevance to balance

The vestibular system is important for balance because it is sensitive to the position of the head in space, acting like an internal compass and thereby allowing a distinction to be made between up / down and right / left (Goddard-Blythe, 2000). Therefore, if the head deviates from the upright position, the vestibular system will detect this and make preparations to correct it through the application of anti-gravitational muscles in the way of automatic head and neck righting and equilibrium reactions. It can also distinguish environmental motion from one's own motion and so creates a stable and clear visual gaze when the head is in motion.

Vestibular Dysfunction

Not only may loss of balance and a problem with gaze fixation be the symptoms of peripheral or central vestibular lesions but an abnormality of visual perception may

also result (Savundra, 1997). Further problems include motion sickness, visual-motor dysfunction, becoming easily disoriented, having poor directional awareness, space perception and organisational skills. Children also tend to fear swings and heights (Goddard, 1996). Symptoms reported by sufferers include faintness, giddiness, sensations of swimming or floating, unexpected falls and anxiety / difficulty thinking in certain environments (Savundra, 1997).

3.3.2 The proprioceptive system

What it is

Put simply the proprioceptive system enables the awareness of static position and movement of the body and limbs. According to Savundra (1997) it has the major role in balance and like the vestibular system, the effectiveness of the proprioceptive system depends upon the ability of the brain to integrate information from all the other sensory systems including feedback from muscles, skin, joints, vision, the tactile sense and the vestibular system. Complete loss of proprioceptive input from the lower limbs results in a loss of postural stability which highlights the importance of the role of this system for balance compared to the other systems (Howe and Oldham, 1981).

Its relevance to balance

The proprioceptive system is responsible for sensing body position and ultimately providing the afferent support needed for the muscle contraction that allows for skeletal movement (Ayres, 1972).

It provides information about the position of the body in space, either at rest or in motion, and allows us to perform tasks such as turning on a light switch in the middle of the night (Elleman, 2002). Its input comes from the skin as well as pressure and joint receptors of the foot and other parts of the body such as joints, tendons and muscles. Hence it allows us to make the appropriate postural adjustments without necessitating conscious awareness (Goddard-Blythe, 2000).

Kinesthesia refers only to the sensations involved in active muscle movement and is the conscious awareness of joint position and movement. It is thought that the

sense of kinesthesia comes from the stimulation of the joint receptors rather than from other proprioceptive receptors (Ayres, 1972).

Proprioceptive dysfunction

If children suffer from impaired proprioceptive feedback they will not be very aware of their bodies' movements and as a result will seek out activities that provide extra stimulus impact in order to process information about their body positions in space. Such activities include things like grasping objects very tightly, jumping vigorously for long periods or crashing into things (Brody, 2002).

Goddard-Blyth (2000) claims that those children who demonstrate poor proprioceptive skills are those who also suffer from learning difficulties.
Symptoms of poor proprioception include:

- Poor posture.
- Constant fidgeting or moving.
- Excessive desire to be held.

3.3.3 The visual system

The visual system is responsible for providing information about the environment and movement taking place around us. The visual-motor system passes on information about eye position and velocity of the head which then affects head position which ultimately affects body position (Howe and Oldham, 1981). In addition, the eyes are connected to the other organs of balance and send information to the same balance control centres in the brain. The brain is then able to compare the information from the visual system to that which it has already received from the vestibular and proprioceptive systems. In this way, vision can act as an "objective" source of information when the body shifts orientation and position, and can verify the accuracy (or not) of a movement (Williams, 1983, p. 265).

The types of visual information

The types of visual information necessary for guiding locomotor activities are three fold according to Bruce et al. (1996). Firstly, exteroceptive information is that which guides action in response to the layout of the surfaces in the environment and the

positions of objects or events within it. Proprioceptive information is that which allows movement of body parts in relation to each-other to allow for co-ordinated action, and finally, extroprioceptive information is that which allows movement of body parts in relation to the environment.

The visual pathways

In the mammal there are two principal pathways for visual sight to the brain; the primary or "geniculocortical" pathway and the secondary or "retinotectal" pathway.

Both begin with the retina, which is extremely important in its ability to distinguish between different types of movement, determining whether it is that an object has moved or that the eye has moved relative to the head or whether the eye and head are moving together (Howe & Oldham, 1981). Once light energy reaches the retina it is transformed into neuronal-electrical impulses and then sent to one of the two photo-sensory receptors: the rods or the cones from where it is sent down the optic nerve. The optic nerve fibres account for a huge 38% of all sensory and motor fibres entering and leaving the CNS (Umphred et al, 1985). The fibres from the nasal half of each retina cross at the optic chiasm in the hypothalamus but those coming from the temporal region of the retina pass through without crossing. The pathway is then referred to as the "optic tract." At this point some of the axons terminate in the pretectal area of the mid-brain, involved in reflex responses of the pupil. Others terminate in the superior colliculus of the mid-brain which is responsible for the control of the muscles of the eye. The pathway from the retina to this latter area is known as the retinotectal pathway, the secondary pathway for vision in the brain. This pathway carries enough information to enable some localisation and identification of objects, even in those with damage to the visual cortex who report complete blindness. Therefore, it could be responsible for guiding some actions in an unconscious way (Bruce et al., 1996).

However, in what is referred to as the primary or geniculocortical pathway the majority of the retinal ganglian axons run to and terminate in the lateral geniculate nucleus (LGN) of the thalamus. This then continues as the geniculocalcarine tract where it once again separates onto the top and bottom of the calcarine fissure of the occipital lobes. Primary or non-conscious sight, allowing an individual to see, occurs once the tract reaches the occipital lobes. However, visual perception and hence conscious sight is not possible until the cortical lobes have also been activated, when

meaning can be attributed to the images. The axons also run to the striate cortex in the primary visual cortex, however, it is not until axons from the visual association areas contribute their input that recognition and understanding of images can begin. Axons from the occipital cortex control movements of eyes when we are unconsciously fixating a gaze on a stationary or moving object. The parietal lobes allow for the recognition of identified objects and their positions in space and also of visually guided movement. Finally, the temporal lobes are responsible for storing visual memories.

Within the occipital lobe, areas 18 and 19 are responsible for the production of reflexive visual pursuit where as voluntary movements are controlled by the frontal lobes of the cortex. Blinking and other protective reflexes are controlled by the vestibular system and the brainstem level. The cerebellum is also involved in visual pursuit and eye-blink conditioning (see Woodruff-Pak et al., 1996 and Miall et al., 2001). Movement is much more than just reflex action, a point made by Stejskal (1979; cited in Umphred et al, 1985) who explains how the turn of the eye toward an object leads to a natural tendency of the head to turn, which itself causes the neck to turn. Once this has occurred the whole upper extremities are in a better position to perform complex motor tasks.

Thus, as Bruce et al. (1997) and Kiernan (1987) summarise, the eventual result of the complex process through which the retina transforms fluctuating patterns of light into something meaningful is simply to ensure that this visual information is unconsciously used to appropriately control our actions (i.e., the maintenance of steady postures and movements).

Its relevance to balance

The importance of the visual system can be demonstrated by requesting someone to stand on one leg with eyes open then closed. The loss of balance or sway which results is due to the difficulty of having to balance without visual feedback. The information coming from the environment allows for precise timing and control of movement by offering a means whereby one can "line up" one's body relative to external verticals and horizontals (Howe & Oldham, 1981, Bruce et al., 1996).

Williams (1983) claims that the information provided by the visual system in the control of balance may be more important than the information provided by the

vestibular or proprioceptive systems. Indeed, Bergmann, Bertora, Claussen and Kotz-Claussen (2002) have shown through the development of an electronic platform designed to test balance (or what they term "dynamic posturography"), that 40% of the equilibriometric distribution belongs to the visual system, whereas the vestibular and proprioceptive systems each contribute approximately 30% to balance, although people can function normally even when reliable feedback from one of these three systems is compromised (Hendricks, 2002).

Where postural adjustments and maintenance of balance are concerned, Lee & Aronson (in Bruce et al., 1996) claim that vision can "tune up" the sensitivity of the mechanical systems (i.e., the vestibular and proprioceptive systems) and is a more reliable source of information while the feet and ankles of the child are growing. For example, the visual system can be seen over-riding the mechanical system is adults using the "swinging room" of Lee & Lishman (1975, in Bruce et al., 1996). Here, slight movement of the walls forwards or backwards caused standing adults to sway gently. A common occurrence of a similar effect is that of being on a stationary train which we perceive to be moving when in fact it is the train at the next platform which is moving. These examples demonstrate how visual feedback, through the use of optic flow can over-ride the information we receive from the other systems where novel or demanding conditions are present.

3.4 Sensory integration

The idea that children with learning disorders might have problems integrating sensory information was postulated by Ayres (1972; 1978). She noticed that certain retained reflexes (i.e., ATNR and TLR) were correlated with educational difficulty, as was the presence of abnormal nystagmus (eye-movements) in response to vestibular stimulation. DeQuiros & Schrager (1978) suggested that learning impairment was a result of the inability to inhibit sensory stimuli. Sufferers become confused because their processing system is so overloaded by simultaneous multiple stimuli. This is reflected in a recent study which suggests that the main problem of dyslexics is a general sensory perceptual problem, which makes if hard for such children to pick out the appropriate cues amongst the mass of available stimuli, or "noise" (Sperling et al., 2005). This "noise" is what Ayres described as a "traffic jam in the brain" and labelled "sensory integrative disorder". It does not reflect any physical damage to the brain but

is the result of faulty sensory processing. According to Ayres (1979) the five senses which must integrate properly in order to allow for speech, language, reading and writing, and consequently unimpaired academic learning (among other things), include the auditory, vestibular, proprioceptive, tactile and visual senses (see Table 4 below).

<table>
<tr><th>Senses</th><th colspan="3">Integration of their inputs</th><th>End products</th></tr>
<tr><td>Auditory</td><td colspan="2"></td><td>Speech
Language</td><td rowspan="6">Concentration.
Organisation.
Self-esteem.
Self-control.
Self-confidence.
Academic learning.
Abstract reasoning and thought.
Hemispheric specialisation.</td></tr>
<tr><td>Vestibular</td><td rowspan="3">Eye Movements.
Posture.
Balance.
Muscle Tone.
Gravitational Security.</td><td rowspan="3">Body Percept.
Coordination of 2 sides of the body.
Motor Planning.
Activity Level.
Attention Span.
Emotional Stability.</td><td rowspan="3">Eye-hand coordination.

Visual perception.

Purposeful activity.</td></tr>
<tr><td>Proprioceptive</td></tr>
<tr><td>Tactile</td><td>Sucking.
Eating.
Mother-Infant Bond.
Tactile Comfort.</td></tr>
<tr><td>Visual</td><td colspan="2"></td></tr>
</table>

Table 4: The senses, the integration of their inputs and their end-products (From Ayres 1979, p.60).

As can be seen, different groups of senses may contribute to different functions, and all of these functions combine ultimately in order to allow for the acquisition and development of skills necessary for smooth every-day living (such skills are usually proficient by the time the child starts school).

Vestibular / proprioceptive dysfunctions, for example, may show in the child who cannot sit up in his chair at school, who is clumsy and often drops his pencil (Ayres, 1979). Because he cannot inhibit stimuli or inappropriate responses, he may often be distracted or hyperactive. Because reading and writing depend upon finely-

tuned sensory integration (e.g., space perception, language, memory, visual perception, hand & finger sensations and muscle organisation), disruption of these will affect such skills. Thus, children may be "dyspraxic", "ADHD", "dyslexic", "speech & language disordered" or present with a co-morbid mix of these disorders, depending on how their sensory integration dysfunction manifests. If the cerebellum is engaged in modulating sensory inputs and outputs, it is easy to see how a problem in the cerebellum would result in varied symptoms. Unable either to reject stimuli it does not need, or prevent inappropriate responses, an impaired cerebellum could explain the kinds of "end-products" of Ayres's theory which are deficient in dyslexics (my understanding of this is illustrated in Figure 4).

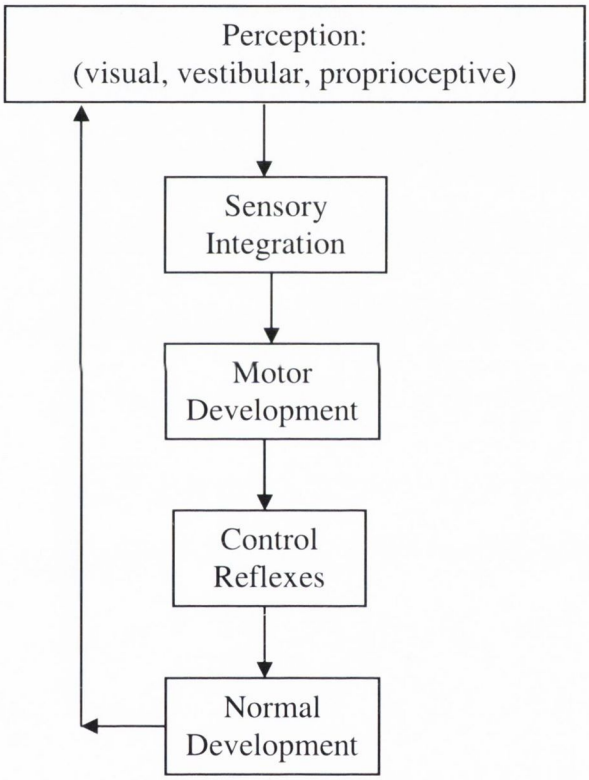

Figure 4: The way in which sensations, perceptions and motor development link together in order to produce the child who is ready to learn.

Thus, the sensory integration provides the foundation from which an infant can explore its environment. This stimulates the motor development sequence, which, once begun, allows for a continuous feedback of stimulation from which the child can learn and

develop further (see figure 4). Morrison (1985) suggests that sensation and perception become integrated through movement.

4 Chapter 4: The link between motor abnormality and dyslexia

The need for a new method of identifying (and treating) dyslexia has previously been outlined (chapter 1). The current chapter reviews the development of an alternative approach to the theoretical understanding and identification of dyslexia, which contributes towards the formation of an alternative causal theory for the disorder (chapter 5).

An exploration of this theory requires an understanding of the different kinds of evidence which contribute to that theory. Although there exists an abundance of anecdotal evidence to back up claims of motor treatments improving the functioning of those with neurodevelopmental disorders (including dyslexia) (see Appendix 2) the examples referenced below include largely academic journal articles and books. Whilst the former are usually peer-reviewed and subject to methodological scrutiny, the latter are not, and yet they are the sources more commonly accessible by the general public. Much of the evidence supporting these ideas appears experimental but has been presented in non-peer reviewed books. Often it has been conducted by teachers or others ignorant of the importance of randomly assigned control studies in the drawing of general, or valid, conclusions. Many of the small-scale studies which found support for "neurological rehabilitation" (e.g., Masterman; Floyd-Miracle) were published in Delacato's 1966 book and as such should be interpreted with caution.

4.1 Cognitive and motor development in normal children

Diamond (2000) claims that although motor and cognitive development are often thought to have different maturational time periods, with the former developing sooner than the latter, they both in fact, take equally long to develop. Like cognitive development, the more complex aspects of motor skills such as fine motor co-ordination, bimanual co-ordination and visuo-motor skills, take until adolescence to mature fully, in much the same way that complex cognitive operations do. Ramsay (1984) demonstrated that babbling and handedness develop together, with Annett (1973) finding a correlation between vocabulary and foot movements, and Rudel (1985) finding a correlation between speed of finger, hand and foot movements and verbal fluency. Kohen-Raz (1996) points to the simultaneous development of postural

control with Piaget's stages of development. For example, object permanence is not seen in infants until they have acquired bilateral grasp in the sitting position; speech and symbolic behaviour until they are developing the ability to stand and walk and so on. In fact, "psychomotor school readiness" is that term given by Kohen-Raz (1996) in merging Piaget's ideas of operational thought, in which the child's speech and thought are no longer dominated by his own cognitions, with the ideas of deQuiros and Schrager (1978) and Palagyi (1925). DeQuiros and Schrager believed in the importance of freedom from conscious involvement in balance maintenance, labelling this ability "corporal potentiality" (i.e., postural autonomy); only once this had been achieved could the child engage in higher symbolic operational thought. Palagyi suggested that the ability to conceptualise "virtual movements" (rather than actually performing them) allowed for the appreciation and understanding of an external world distinct from oneself (cited in Kohen-Raz, 1996, p.123). This all contributes towards the development of cognitive skills necessary for school, such as alertness, attention and orientation in space and time. Studies reported by Kohen-Raz (1996, but conducted in 1969), in California and Israel support these claims. From the California study, he reported a negative relationship between kindergarten readiness and postural sway; also, first-grade reading scores were positively correlated with monopedal postural stability. Similarly, the Israel study found that reading scores and postural stability were significantly correlated.

Thus, evidence was emerging that supported the hypothesis of a reciprocal relationship between cognitive and motor skills. The idea that motor stimulation could be used as an influence on cognitive development simultaneously emerged as a treatment for cognitive deficiencies.

4.2 Lateral dominance

The current study is ultimately concerned with whether or not there is a *causal* link between motor impairments and dyslexia (originating from the cerebellum). The existence of a simple association between the two, though it has little bearing on causation, has long been evident and is not in dispute. This relationship dates back to Orton in the 1920s, who was of the view that reading problems and mixed dominance were related (Orton, 1925; 1937). Specifically, he believed that in dyslexic children the left hemisphere failed to become dominant for reading and writing. According to Kephart (1971) laterality is the only thing which allows us to detect the directional

characteristics of letters such as "b" and "d" and as such has consequences for literacy. Orton pioneered the use of "laterality training" which he claimed would stimulate one side of the brain, thereby fostering lateral dominance (Selikowitz, 1993). This could take the form of eye-patches or any particular method which involved training one side of the body over the other. The goal of human motor development was deemed to be functional independence of each side of the body, so that one could engage in automatic balance and smooth motor co-ordination. Free from the conscious adjustment to gravity, deQuiros and Schrager (1978) argued that we could engage in learning other skills and perform the smooth movements necessary for speech and literacy acquisition. Orton's idea was furthered by Delacato in the 1950s, who extended the importance of dominant laterality to the foot and eye. Later, Denckla (1985) claimed to support the idea by showing that 60% of dyslexic children showed evidence of mixed laterality compared to just 30% of normal children.

DeQuiros and Schrager (1978) firmly supported the theoretical importance of laterality and motor development in contributing to adequate learning. They knew that the cerebellum was responsible for coordinating sensory information such as vestibular, proprioceptive and visual stimuli and claimed that such senses contributed to muscle tonus thereby allowing for the development of posture and equilibrium (balance). They observed that perceptual and motor development occurred simultaneously, with the development of one contributing to the development of the other; enabling skills like hand-eye co-ordination to become perfected. A condition such as dyslexia, they felt, resulted from vestibular impairments, which they referred to as "vestibular-proprioceptive dissociation". This would occur if vestibular action was strong on one side of the body and proprioceptive action on the other. Such a child would not be able to maintain the attention necessary to learn because of a constant feeling of instability. Also, impairments in neck and ocular muscles could cause reading problems and poor motor control in general could lead to poor hand-writing. Because speech, reading and writing all require skilled movement, vestibular disturbances could affect them all.

Others, such as Schain (1972), also noted that children with learning disorders appeared to be clumsy when engaging in fine motor skills, such as tying shoe-laces. He also listed (among others) reflex asymmetries, right-left discrimination difficulties, mixed laterality, dysdiadochokinesia and ocular apraxia as common amongst such people.

4.3 The Neuro-maturational theory

Gesell (1938) believed in an orderly genetically determined sequence of motor development controlled in pace by the maturation of the nervous system. He was influenced in his thinking by Coghill, an embryologist who showed that the change in movement patterns of the salamander corresponded to changes in its neural connections (i.e., Coghill, 1929). As such, Gesell pioneered the "neuro-maturational" theory of human motor control, with the view that increased "corticalisation" of the brain eventually resulted in higher-order movement patterns, free from the control of retained reflexes (McCormack and Perrin, 1997; Gesell, 1988). As all responses and actions are subject to the pull of gravity, all development is bound by it, including that of the mind. Such a hierarchical theory of motor development, where increasingly complex reflexes are controlled by higher and higher levels of the CNS, was popular throughout the 20th century (e.g., Bobath, 1971 and Fiorentino, 1972).

4.4 Neurological organisation

Influenced by the neurosurgeon, Temple Fay, Carl Delacato (1959) (a doctor of education from Philadelphia) believed that the process of reading was possible due to perceptual abilities afforded by a "neurologically organised" brain. The development of this neurological organisation, which he claimed was the principal difference between man and lower animals, came from a sequential and successful progression of ontogeny (i.e., origin and development of an individual) and mobility which recapitulated phylogeny (i.e., genesis and evolution of a species). Thus, the neuro-developmental stages (i.e., simple spinal cord and medulla; pons and mid-brain; larger mid-brain; cortical dominance and cortical hemispheric dominance) through which we pass whilst learning to move (homolateral crawl, heterolateral crawl, heterolateral walk and finally perceptual abilities including reading) are similar to those seen in the movements of animals progressing up the animal scale (i.e., low level vertebrates followed by amphibians, reptiles, mammals then primate ancestors, in that order). Failure to read, communicate or move properly, he claimed, was a result of trauma to the development of this nervous system and could be remediated on the basis of a movement programme offering the opportunity to "relive" (i.e., "recapitulate" either passively or actively) the relevant stages of mobility in the attempt to reorganise the brain (Novella, 1996). This reorganisation would begin at the level of arrested development and continue until dominance was achieved with the hands, eyes and feet. He viewed his approach as one

which treated the central cause, regardless of the specific symptom (Delacato, 1959; 1966). Together with his colleague, a physical therapist named Glenn Doman, the pair devised a treatment for the neurologically "disorganised" brain, which they called "Neurological Rehabilitation" (but more commonly known as the "Doman-Delacato technique"). They established the "Institute for the Achievement of Human Potential" in Philadelphia (US), from which they practised their technique. It aimed to help people with neurological disorganisation, by stimulating the older, more primitive part of the brain into activity (Blythe and McGlown, 1979) keeping the correct movement of the arrested stage, before moving on to the next stage. For example, a child who did not crawl properly was required to target the medulla level of the brain by lying on his back and engaging in the movements of the tonic neck reflex by following his hand with his eyes, as it moved up and down (e.g., Noonan, 1966). A child who did not creep[11] properly (i.e., in a cross-lateral manner) would have been encouraged to creep such that opposing hand and knee were used simultaneously (thereby targeting the pons and mid-brain). The child was also to look at the most forward limb in an effort to develop binocularity (and when walking would have to point at the forward limb using the opposing hand). Such tasks were to be taught slowly over the course of six to eighteen weeks. Where the child could not engage in the task alone, his limbs were manipulated by 5 adults stationed at each limb and at the head. They performed the movements for him, supposedly *imposing* them on the central nervous system, a technique Doman and Delacato referred to as "Patterning". Although the latter would be suggested only for the seriously disabled child (usually those with cerebral palsy), the technique was prescribed for children of many disabilities; not just those with brain damage, but those of normal intelligence with learning disorders, those with autism and also those with Down's syndrome.

Figure 4 (from chapter 3) shows how the integration of senses allows for adequate exploration of the environment which both stimulates motor development and improves sensory integration. A child developing normally, in this way, is the child who is "neurologically organised", who has "sensory integration" and who can develop "lateral dominance". He is also the child who has integrated his primitive reflexes and

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[11] The term "creep" was used by such theorists to refer to what we commonly know as "crawling" today.

whose vestibular functioning is normal. All of these terms relate to the same underlying idea.

4.5 Dyslexic children and motor deficiencies

Bruininks and Bruininks (1977) showed that those with learning disorders were less motor proficient than a control group. They were tested on measures of running speed, agility, bilateral co-ordination, strength, upper limb co-ordination, response speed, visual motor control and equilibrium in stationary and dynamic positions. Similarly, Freides et al. (1980) also claimed to find evidence of poorer dyslexic performance on tests of primitive reflexes and gross motor skills (as measured on a tilt board). This latter study suffered from poor inter-rater reliability on many of tests however and the sample size was small (n = 22) (in Morrison, 1985, p.72).

Rudel (1985) found that on most of their measures of fine-motor skill (i.e., palm-reversal, finger-thumb opposition, finger tapping (with the exception of heel-toe alternation)) dyslexics were significantly slower than controls at performing the tasks. Studying 7 and 8 year olds, Denckla (1985) also found a difference between these two groups in tests of toe-tapping and finger-thumb opposition.

Levinson was suggesting a specific link between cerebellar deficit and dyslexia in the 1970s. Frank and Levinson (1973) supported the theory that dyslexia was a form of vestibular disorder, claiming that, of their dyslexic sample, 97% (i.e., 112 out of 115) showed evidence of cerebellar-vestibular ("CV") dysfunction (e.g., balance, coordination, rhythmic problems). This, they theorised, played a causal role in dyslexia, being that normally working cerebellar-vestibular circuits provide a stable motor background by ensuring automatic and well integrated eye, head and neck muscles. Due to the close link between the labyrinthine system, vestibular system, eyes and cerebellum, a processing problem affecting one of these could result in dyslexia if the ocular fixation necessary for sequential scanning of letter and words was impaired to such an extent that the child's visual perception became scrambled. Levinson referred to the findings of Pavlidis (1981) (which showed dyslexics to have disturbances in saccadic eye-movements) to support his claims, and published his own study documenting such differences in 1989. His published data on 4000 of his patients, again claimed that most (94%) showed two or more signs of CV or

Electronystagmographic[12] (ENG) dysfunction (Levinson, 1988). He even claimed that dyslexia could be treated medically, using antihistamines and anti-motion sickness pills such as Meclizine or Cyclizine which resulted in improvements for 75% of dyslexics in various areas including, academia, concentration, behaviour, balance, coordination, speech and "mental-related symptoms" (not specified) (Frank and Levinson, 1976; 1977; Levinson, 1980; 1991). Levinson (1988) is aware of his lack of controlled comparison groups when describing his results of cerebellar-vestibular dysfunction and this is a very legitimate point. His sample is self-selected, having opted to attend his clinic; thus, it is biased toward attracting the very people he claims should be experiencing the problems he describes. It is no wonder then that his percentages were so high. Furthermore, Levinson (1990) later found that his ENG tests failed to distinguish between dyslexics and controls, in addition to which the only study to try to replicate his anti-motion medication findings (i.e., Fagan et al, 1988), failed to find them to be an efficient method for improving reading itself (though it did find some support for the treatment of visual-motor stability during steady fixation). Thus, it appears that either the theory, or the methodology to test the theory, could be at fault.

4.6 The popularisation of reflex theory in remediation

The presence of reflexes in new-born infants has already been discussed and is not of dispute. Also medically sound is the fact that certain reflexes can no longer be elicited in the normally developing child after a certain age. The idea that children with neuro-developmental disabilities (i.e., dyslexia, dyspraxia, ADHD, etc) retain some of these reflexes which prevent their normal motor co-ordination, and therefore, learning skills, has been popularised in the UK and Ireland by certain organisations. Media frenzy in response to largely anecdotal, but some published, evidence has made the sale of "reflex-integrating" therapies a commercially viable enterprise.

The following section presents some of the work produced by various disciplines which has advocated this approach over the past few decades. Silver (1986) points out

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[12] Electronystagmography (ENG): The ENG is a standardized neuro-physiological test in which eye movements are induced and measured under various testing conditions. Fine and reflexive eye movements are controlled by the cerebellum and the vestibular system. As a result, the ENG can help determine whether or not an inner-ear abnormality exists (The Levinson Medical Center for Learning Disabilities, 2000-2001).

that whilst some research is published in reputable journals, much of the support for this approach has been made available to the general public in the form of popular books, newspapers, lay magazines or television. Although this is still the case today, the growth of academic journals, postgraduate study and research grants has also made controlled experimentation of ideas more prolific. The 1960s and 1970s however appears to represent a time in the field of learning disabilities where ideas were embraced and publicised without such stringent criteria and perhaps the growth in public media generally was sufficient to give credence to these ideas. This was also a time of holistic and experiential ideas which provided the mental framework for such theorising. Delacato (1964), Ayres (1972; 1978), deQuiros and Schrager (1978), Holle (1976), Bender (1976) and Blythe and McGlown (1979) represent this period of thought, and their methodology and findings are somewhat questionable. The likes of Kohen-Raz's (1996) studies in the early 1980s marked a change in the approach to studying (motor) treatment efficacy by employing more secure methodology, with the likes of McPhillips et al. (2000) engaging in well-designed studies more recently.

Some smaller studies using learning disabled children were conducted early on by Ayres (1972) who attempted to remediate what she called "sensory dysfunction" by stimulating the senses and inhibiting primitive reflexes at the same time. She did this by encouraging the child to assume positions in the prone, supine, quadruped and sitting positions and focusing on vestibular, tactile, proprioceptive and motor planning stimulation. DeQuiros and Schrager (1978) were similarly in favour of stimulating these senses whilst also making use of occluded vision and suppressing synkinetic[13] movements. Ayres found that the experimental group improved more so on the WRAT reading test than did the control group, however, 6 years later, she failed to replicate this finding (Ayres, 1978). Her groups were selected from "special schools" in the US, without any elaboration on the nature of the learning disability. Academic tests were administered without any reporting of the means, standard deviations or ranges they yielded, and no change in vestibular functioning (as measured by nystagmus) was reported at post-test, despite this representing the crux of these children's problem. The study also lacked any placebo-control group (Morrison, 1985).

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[13] An involuntary movement of one bodily part that occurs simultaneously with a voluntary movement of another part (Reber, 1985, p. 779).

Holle (1976) was experienced in the teaching of both normal and mentally sub-normal (i.e., mentally-retarded) children, and was aware of the importance of sensory-motor development and how it related to the acquisition of literacy in normal children. She too was aware of the impact of a retained ATNR on developmental skills in general (e.g., self-feeding, hand-eye coordination) and devised a rehabilitative programme based on the inhibition of reflexive movements to try to counteract it.

Meanwhile, Bender (1976) was conducting a study to explore the differences in retained reflexes between children with learning difficulties and those who were achieving in school. She claimed that approximately 75% of those with learning difficulties showed a retained STNR, whilst the others did not. She subsequently designed the "Bender-Purdue Reflex Test" (1976) and a treatment programme to go with it, targeting mainly children with ADHD. This is still commercially sold from the Bender Achievement Centre in Indianapolis, Indiana, US, and the theory is exactly the same as that suggested here (i.e., neurological maturation).

The importance of "retained reflexes" was also embraced by Blythe and McGlown in 1979. They used the term "organic brain dysfunction" to describe children fitting the criteria of their disorder. Their idea claimed that persistent, aberrant reflexes interfere with the development of motor behaviour, such as rolling, crawling and creeping and that these movements themselves were necessary for inhibiting former movement patterns and facilitating the next ones. Failure of movements to become inhibited and transformed subsequently affected developmental and academic progress. This idea formed the basis of a motor therapy programme originally known as "Reflex Developmental Patterning" (or the "Home Programme") under the auspices of the "Institute for Neuro-Physiological Psychology" (INPP), Chester, England. Goddard (1996) later joined the organisation, furthering its name and assisting in exporting the practice to other countries. The original premise was that the arrested stage of development must be physically returned to and integrated, in much the same way as a psycho-analyst would require psychological regression to an emotionally traumatic episode before hoping to resolve the conflict (Blythe and McGlown, 1979). Integration of these reflexes, they claimed, was possible at any age. Should they not have been integrated in the normal way at the normal time, or should they become evident due to trauma or pathology, the above programme, performed daily for 20 minutes over 12 weeks, could aid in re-integrating them. Without having done any controlled studies, Blythe and McGlown claimed that a 12 week programme could improve reading-age in

children by up to 2 years. Blythe and McGlown also commented on the general awkwardness of movement in such children. Like Orton before them they noted the frequency of cross-laterality, as well as abnormal crawling / creeping patterns, the eye-muscle and visual discrimination difficulties. Goddard-Blythe and Hyland (1998) claimed that motor development delay loaded as one of two factors on the INPP's screening questionnaire which discriminated between children with specific learning disabilities (SLD) and normal children (the other being phonological development), however, this may not be surprising given that the children with SLD were drawn from INPP's own data-base of children whose parents could well have selected the service in the knowledge of the kind of therapy it offered.

According to Blythe and McGlown, the course of development for a problem such as dyslexia would go something like this:

Reflex abnormalities
Aberrant motor developmental pattern / hand function
Failure to develop laterality or mixed laterality
Eye muscle dysfunctions
Problems of eye and hand co-ordination
Perceptual difficulties
Reading difficulties
(McGlown , 1990, p.203).

A problem such as this, they claimed, should be approached from the cause (i.e., reflex abnormalities); tackling the end result (i.e., reading) would not be the best approach. McGlown's (1990) developmental theory is similar to that of Doman and Delacato, in which a complex hierarchy of mobility involves stages of crawling, creeping, upright and bipedal walking, where each stage contributes to perceptual maturation and neurological integration leading to increased functionality (McGlown, 1990). If the child continues to grow without passing through, and integrating, these stages adequately, then a baseless scaffolding is constructed, which cannot hope to support the child in higher level skills later in life.

The "Bobath Approach" devised by Bobath (1980) was another treatment based on the importance of inhibiting abnormal reflex activity and facilitating higher reflexes and was used with Cerebral Palsied children. This attempted to teach the child normal

movement patterns by improving postural tone (Greenwood et al, 1997) and were initially passively imposed (though they now involve more active approaches). Electromyograph records of "at rest" muscle activity in those with Cerebral Palsy seem to be greater than the muscle activity seen in Bobath's reflex-inhibiting posture in such children, and are similar to the effects of Diazepam[14] on muscle activity) (Holt, 1991). This might explain the attraction for parents of "non-invasive" treatments for their children.

Several studies on young infants, conducted around this time, seemed to suggest that vestibular stimulation was beneficial for improving cognitive skills (as measured by visual alertness to the environment) or other aspects of behaviour. For example, Korner and Thoman (1972) found it soothed infants; Kanter et al. (1976) found it improved nystagmus response and motor performance; and Gregg et al. (1976) found it improved visual pursuit.

It was 1983 when Ornitz warned that developmental research into the rapid expansion of vestibular therapies was urgently needed. The situation has continued, however, relatively unchecked, with treatments and "miracle cures" offered from many different places (see Appendices 26 & 27).

A study conducted by McGlown himself in 1982 (but reported in his 1990 book), claimed to demonstrate wonderful effects from the use of this reflex inhibition programme. Of the 25 learning disabled children involved, all showed a retained ATNR, 21 showed a retained STNR and all showed crawling and visual motor integration problems. The results claimed that the group had fully integrated their retained ATNR after 9 months of treatment, they had inhibited their STNR and they had improved academically by an average of 31 months in reading and 13 months in spelling. However, McGlown reports no control group, no placebo group and no degrees of significance.

A month-long study conducted by Delacato in 1964 (reported in his 1966 book) employed a controlled experimental study (n = 108), to discover whether the programme would improve reading-readiness in the experimental group compared to

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[14] Diazepam: A benzodiazepine commonly prescribed for anti-anxiolytic and anti-convulsant reasons, although it is also prescribed for its muscle relaxant effects (Ashton, 1994).

the control group. Although no formal statistical tests were reported, Delacato claims that the reading-readiness, as measured by the number of children moving from "readiness level 5" to "readiness level 4" (where the former represents "not ready"), and the number of children finding the tests easier on the second session, showed an overall advantage of 221% for the experimental group (Delacato, 1966). This followed the reported success of a previous study conducted by Doman et al. in 1960, which showed that the implementation of patterning for severely brain-damaged children, resulted in an improvement in mobility worth 50% of the "ideal" (i.e., walking). A study conducted by Masterman (1966) claimed to show support in favour of Delacato's method at a six-week summer reading centre for reading-disabled children ($n = 282$). A significant difference ($p < .01$) was reported although this translates to a clinical difference of just less than six weeks' difference in gains between the two groups (experimental group improved in reading by 5.65 months whilst the control group improved by 4.21 months). A similar study employing control groups was conducted by Floyd-Miracle (1966) showing similar findings, however, these and many of the other small studies reported at this time (and conducted mainly by teachers without adequate research skills) employed small samples, Hawthorne effects, experimenter bias and careless assignment to groups. In addition, none considered how anomalies to Delacato's theory could be explained. For example, of those studies that included average / good readers, it was found that 60% had poor neurological organisation (e.g., Rader-Kabot, 1966). Similarly, one study showed that, of those whose reading was poor, 31% had good "neurological organisation" (e.g., Mariam, 1966). Finally, a study by Kershner (1968) found that, although the programme did improve retardates' ability to crawl, the control group actually improved more than the experimental group on tests of perceptual-motor skills.

The only peer-reviewed, published study replicating Doman et al.'s 1960s work, was that by Neman et al. (1974) and this was heavily criticised by Zigler and Seitz (1975). The latter's findings revealed that impressive-looking gains in neurological age were accounted for by a psychometric problem with the Doman and Delacato developmental profile (for example, a raw score of 32 on Doman and Delacato's profile converted to 48 neurological months, and a raw score of 33 converted to 54 months – a seemingly huge leap in functioning). Even in studies where gains were found and statistical significance was reported, the crucial question (relevant to all treatment-efficacy research) of *how* this statistical significance

translated to clinical or "substantive" significance was not considered. Regardless of whether the theory was ever even anatomically tenable (see Cohen et al., 1970) it seems that it never had any real pragmatic utility, as evidenced in further studies by Robbins (1966a; 1966b), O'Donnell and Eisenson (1969), Jackson and Dando (1976) and Sparrow and Zigler (1978). A meta-analysis study of perceptual-motor training programmes in general (Mattson, 1983), showed the same thing and this was later supported by Polatjako et al. (1992). Mattson points out that such treatments are not only a waste of valuable time and money, but they deny the child proper remediation of his condition. Specifically, Robbins (1966b) concluded that creeping was not related to reading beyond chance levels; there were no differences in reading between those who had "neurological organisation" (i.e., lateralisation) and those who did not; there were no differences in reading between groups after implementation of the programme and, finally, the number lateralised after the programme did not exceed the number lateralised before it.

As for the theoretical plausibility of the exercises, the recapitulation theory is considered a far too linear view of human evolution for it to make sense. In addition, the idea that patterning can affect neurological organisation has been branded a gross over-simplification of the processes affecting brain development, failing, as it does, to consider genetic influences, toxic insult and biochemical abnormalities. It also fails to consider the biological sequence of cell growth, migration, organisation and cell death on brain development (Novella, 1996). Like the pharmacological therapy defended by Levinson, "Neurological Rehabilitation" has been described as "verging on outright quackery" (Farnham-Diggory, 1978). Incidentally, the success of Levinson's treatments has been attributed to other possible causes such as eye movements, rather than as he claimed, vestibular dysfunction (Pope and Whitely, 2003, p.116). The review by Silver (1986) echoed Farnham-Diggory's conclusion in its failure to find support for any correlation between vestibular function and academic performance (p.1049) and his methodology has also been criticised. Pope and Whitely point out that it was possibly children's failure to attend adequately to instructions than the existence of a hyporeactive nystagmus that lead to their results on such measures.

The Doman and Delacato theory and technique was embraced and adored during the 1960s, resulting in the establishment of the "Institute for the Achievement of Human Potential" in the US, as indicated earlier. However, given the lack of replication and subsequent criticism of the ideas and practices, the treatment fell into

disrepute. In 1968 just about every relevant US medical academy and children's disability council endorsed a statement rejecting the Doman and Delacato practice, on the basis of its spurious diagnostic validity, poor replication results, financial cost and heavy emotional and physical demands on the children and their families. This included the American Academies for: Cerebral Palsy, Neurology, Paediatrics, Rehabilitation Medicine and Orthopaedics, plus the National Down Syndrome Congress as well as many of their Canadian equivalents (to name but a few). Indeed the American Academy of Paediatrics re-issued this policy statement in both 1982 and 1999 such was their concern over its wide-spread and ill-informed use.

4.7 Recent research on which the current study is based

Despite the criticisms made about the Doman-Delacato technique over the years, there remains a strong similarity between their theory and that underlying current motor-based practices today.

4.7.1 Recent evidence of retained reflexes / balance problems in dyslexic children

The presence of severely retained reflexes in Cerebral Palsied children exists together with a difficulty in learning to read despite normal general cognitive ability (Seidel et al., 1975, in McPhillips et al., 2000, p.538). McPhillips et al. (2000) suggest that a far milder retention of such reflexes can also result in those with specific reading disabilities (i.e., dyslexia), but without the more obvious awkwardness of movement exhibited by Cerebral Palsied children. McPhillips and Sheehy (2004) presented evidence that reading-impaired children exhibit a significantly higher mean level of retained ATNR than do both normal and above-average readers, and also that there are greater numbers of poor-readers showing a retained ATNR compared to average or above readers (46% of poor-readers compared to 15% of average and above readers). They also showed that poor and high reading groups could be differentiated on the basis of general motor-skill scores, however, unlike Fawcett et al.[15] (2001b) they did not find that balance skills differentiated between normal poor readers and dyslexic poor readers and this also applied to evidence of a retained ATNR. McPhillips and Sheehy concluded that it would not be prudent to use persistent reflexes as a causal

[15] Fawcett et al. (2001b): This is reviewed in full in chapter 5.

model for reading difficulties but that given their co-occurrence it may be used as an indication for children at risk of reading delay.

Incidentally, the application of retained reflexes in diagnosing other neuro-developmental disorders has also recently been explored. Teitelbaum et al. (2004) used video-images of infants with autism and Asperger's syndrome (AS), which like dyslexia, require a level of language development before diagnosis can be made. They found evidence of retained reflexes in children who were later diagnosed as Autistic / Aspergic, however, this was not an empirically designed study, containing no control group or large samples; as such no firm conclusions can be drawn from their findings.

4.7.2 Recent evidence of treatment programmes

Kohen-Raz (1996): Kohen-Raz's own large studies, conducted in Israel in 1982 and 1983, on 26 normal, though disadvantaged, classes (n = 760) claimed to find evidence for the efficacy of his "psychomotor school-readiness training programme", in reducing the "risk" of learning disabilities in first-graders. These children were between the ages of 5 and 7 years and as many as 30% were deemed likely to be at risk of learning disabilities, given a recent prevalence study at the time. Following the intervention, the results showed that on measures of reading, 5% and 18% of the experimental groups were measured as "weak" in the 1982 and 1983 studies respectively, compared to 28% and 31% respectively of the control groups ($p = .001$). On measures of maths, 16% and 13% of the experimental groups were regarded as "weak" compared to 29% and 31% of the control groups respectively ($p = .001$). The physical education programme was run for two hours a week for 7 months of the school year and involved several levels of emphasis. These included perception of body awareness (encompassing directional and spatial orientation); static and mobile object interaction; bimanual and synergetic movements which could be ordered to "freeze" suddenly in the attempt to suppress unwanted movements, and added stressors to balance tasks involving occluded and semi-occluded vision. Many of these exercises were designed to stimulate the vestibular and proprioceptive systems, thereby targeting the cerebellum.

McPhillips, Hepper and Mulhern (2000): The seminal article and instigator of much excitement and controversy was that published by McPhillips et al. in the Lancet in

2000. Their study made use of movements, the design of which were based upon the kinds of movements seen in fetuses is the womb during ultra-sound scanning, and neo-natal infants (for example, movement of the head to one side, followed by slow extension of the arm on that side). McPhillips et al. tested the notion that a frequently-repeated set of such movements (i.e., 10 minutes a day for 12 months) would not only inhibit retained reflexes beyond the age at which it was assumed they normally disappear (i.e., approximately 6 months) but by virtue of targeting the cerebellum they would also extend into cognitive functioning and improve reading skills in those who demonstrated unexpectedly low reading ability (i.e., dyslexics). By comparing their experimental "reflex-replication[16]" group with matched controls / placebo-controls in a randomised, double-blind study, they produced findings which supported their hypotheses. From a sample of 60 8-11 year-old children (20 in each group) they showed that there was a significant Group x Time interaction not just for reduction in retained reflexes, but also for improvement on reading scores (as measured by the Neale-analysis (Neale, 1989) and WORD tests[17] (Wechsler, 1993)) and "rapid-naming" speeds, where the experimental group outperformed the 2 control groups by 12 months on the Neale-analysis test of reading, 8 months on the WORD test of reading and 7 months on rapid-naming tests ($p < .001$ for all differences between the experimental and control groups). These findings relate not just to statistically significant findings but to clinically significant findings and support the idea for specific effects of replication of primary-reflex movements.

Jordan-Black (2005): The effects of primary movement were explored more recently using class-room based samples and controls. This study also reported significant differences for reading improvement between the experimental and control samples after a year-long intervention as well as significant differences in mathematics improvement. These children reflected an average classroom with the usual range of abilities and, thus, the study was not confined to dyslexic children.

Reynolds, Nicolson and Hambly (2003): Reynolds et al. (2003) also conducted a study looking at the efficacy of an exercise-based programme for reading disabilities. They

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[16] Commercially this is known as "Primary Movement".
[17] The Neale Analysis & the WORD are both British standardised tests of reading ability.

made use of visuo-motor and vestibular movements such as stretching, coordination exercises, throwing and catching which were also supposed to target the deficient cerebellum. Known then as "DDAT" ("Dyslexia, Dyspraxia, Attention-Deficit Therapy) and now as the "Dores' Achievement Centres" therapy, the complete therapy was, however, deemed "commercially sensitive" and therefore not specified exactly or entirely. The study employed a two-factor design (Group and Time), making use of an experimental and a control group (n = 35). A placebo-control group was not used on the basis that it could not satisfy ethical approval and the logistics of designing such a programme were not possible. Groups were tested on measures of balance stability (as measured by electroneurophysiological tests of dynamic posturgraphy and electronystagmography – that is, balance and eye-tracking); the Dyslexia Screening Test (DST) and standardised literacy tests (the "NFER / Nelson" reading tests and the Standardised Attainment Tests (SATS) of writing, comprehension and maths.

The exercises were performed at home, under parental supervision, by the experimental group in 2 x 10 minute sessions daily for a period of 6 months. The study's results show some similarities to the McPhillips study, in that on sub-test measures of the DST[18] (Fawcett and Nicolson, 1996), the experimental group improved significantly more than the control group on reading ($p < .05$) but not on measures of writing or spelling. The experimental group also shows a significant Group x Time interaction on the electroneurophysiological tests of balance and eye-tracking. Apart from improvements in literacy directly, Reynolds et al. also report significant Group x Time interactions in favour of the experimental group for various cognitive skills underlying reading skills, such as rapid naming, semantic fluency and verbal fluency (as measured by the DST).

The interesting results are those revealed by the standardised tests of literacy (NFER and SATS). These showed that the experimental group improved from testing-time 1 to testing-time 2 by 19 months on NFER tests of reading ($p < .05$), compared to 6 months in the previous year, and significantly on SATS comprehension ($p < .01$) and SATS writing ($p < .05$). However, because they had started their control group on the DDAT programme and 3 months of it had been completed, it was not possible to explore the interaction of group over time on these measures. This is a disappointing draw-back of the study.

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[18] Dyslexia Screening Test: Normed for children aged 6.5 years – 16.5 years.

4.7.3 Criticisms

There are several questions which Lipkin and Schertz (1996) suggest should be answered when assessing such intervention programmes as these, which include the following:

1. What is the population being treated and is this a homogenous group (according to disability, age etc)?
2. Is the intervention well-defined?
3. How is "efficacy" defined?
4. What is the outcome being measured?
5. When is the outcome being measured?

Thus, if we judge these studies on the basis of Lipkin and Schertz's (1996) questions, we can see that there are a number of pitfalls.

Primary Movement

First, with regard to the study by McPhillips et al. (2000), one of the suggestions made by McPhillips himself is that the self-selected nature of the sample used may have something to do with the fact that he found it difficult to identify any dyslexic child as free from retained reflexes. Perhaps, he says, his sample was of children for whom nothing else has worked. Given the lack of IQ data it is hard to judge the quality of the dyslexia involved and some concern is raised regarding this finding by other studies which suggest that perhaps all of us show signs of retained reflexes, and that they are not, in fact, indicative of CNS pathology. Ikai (1950) found evidence of the ATNR reflex in normal males between the ages of 7 and 10 years, as did Parr et al. (1974) who found all of their 84 children between the ages of 3 and 9 to show the ATNR in the quadruped position. Lack of support for the discriminating ability of reflex tests between dyslexic and non-dyslexic children was also the outcome of a recent Irish study (McEvoy, 2002).

Furthermore, the intervention is described but no exact detail as to *how* it works is suggested beyond the fact that it inhibits reflexes which should have disappeared in early life. This applies equally to the DDAT treatment and is the basis for Reynolds et

al.'s own statement that targeting the cerebellum in order to improve literacy may require a "leap of faith" (p. 52). It is, as Reynolds states, an area of limited understanding in general, and as such, difficult to even hypothesise about.

Dores Achievement Centres (DDAT therapy)

A meta-analysis conducted by Farran (1990) found that although efficacy studies which reported long-term follow-ups found 50-80% of students reaching the outcome goal, many of these children were at a border-line to mild level to begin with and so may have skewed the findings (in Lipkin and Schertz, 1996). In the Reynold's et al. study of DDAT therapy efficacy, there existed a heterogenous population; some dyslexic, some dyspraxic and some ADHD. Selection was made on the basis of DST scores of at least 0.4 (the mean was 0.73) yet a score of 1.0 is recommended in this specific test for the child to be deemed "at risk" of dyslexia (they used criteria for the DEST (Dyslexia *Early* Screening Test), designed for children aged 4.5 years to 6.5 years), the "at risk" score for which is set at 0.6[19]). Thus, one questions whether these children are even at risk to begin with (a point was also raised by Stein, 2003). Only 4 had been diagnosed as dyslexic and mean scores on the DST literacy tests showed all groups to be within the average ranges (p. 60). Even the title of the study is misleading; the title suggests that this is a study of an intervention designed for "reading-disabled" pupils and yet the abstract goes on to define the disorders of those in their groups as "dyslexia-*related*" disorders (see also Rack, 2003, p. 138). This includes ADHD and dyspraxia, which, whilst commonly co-morbid with dyslexia, do not necessarily imply reading disability in themselves.

Furthermore, the use of the DST for identifying those with dyslexia is of questionable predictive validity (although it is used in the current "Dyslexia Early Years Project" because of the lack of an alternative). The authors, Fawcett and Nicolson (1996), claim that predictive validity is good for the DEST[20] but this has never been replicated in an objective Irish study. A firmer study of DDAT efficacy would have ensured diagnosis of dyslexia in all students, particularly given that the

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[19] The Dyslexia Screening Test – 2nd Edition (DST-2) does have a lower scoring cut-off of 0.6 for "mild risk" however it was not this version of the test that this study employed. The first edition makes no reference to this cut off level.

[20] More on this in Chapter 8 (Methods).

mean age was 9 years, 4 months (i.e., an age which is suitable for formal dyslexia testing).

The intervention is "commercially sensitive" so could not be defined. A vague explanation of its "changing the cerebellum" is mentioned, but no full review of the controversy of this explanation as an explanation for dyslexia, and no attempt to explain how exactly the programme might improve the cerebellum, is given.

The outcomes look impressive, with the experimental group improving 19 months over the course of one year. However, there is no comparison on these tests with the control group so it is difficult to draw firm conclusions about the cause of these changes. The control group scores on standardised literacy tests could not be used because they had been engaged in the DDAT programme themselves for 3 months at the point of test administration.

No placebo-control group was used on the weak basis that it would have been unethical. It would have been possible, however, to offer them the same "delayed-treatment" that the control group were offered.

The electroneurophysiological tests, by Reynolds et al.'s own admission have not been subject to studies of standardisation and so no norms are available for performance scores.

One wonders, finally, whether administration and scoring of the SATS and NFER was blind. This is not mentioned in the published article and therefore it is speculation as to whether or not they were. Even the most well-intentioned teacher could sub-consciously mark the experimental group more favourably.

Problems concerning interpretation of the electroneurophysiological and national literacy tests (i.e., lack of norms and lack of control comparison) leave few solid findings. The important one is that of reading on the DST. As Stein (2003) points out, however, the control group's reading was verging on average at the start of the study which means they had less gain to make compared to the experimental group. In terms of other literacy abilities there were no differences on spelling, writing, or phonological segmentation. On tests of nonsense reading, the control group did significantly better than the DDAT group.

5 Chapter 5: The Cerebellar Deficit Hypothesis

One fundamental point to remember when considering the previous literature highlighting the link between movement and learning, is that also made by Kohen-Raz (1996, p.4), namely, that it is not posture itself, but the common neurophysiological functions (behind postural mechanisms and higher cognitive functions) which may be impaired. This leads us to the last causal theory of dyslexia discussed here, the "cerebellar deficit hypothesis". This seems to address two issues; firstly the disillusionment of the current practice of identifying dyslexia, and secondly the wide-spread disagreement regarding causal theories. Its formation represents an attempt to unify disparate causal theories in one framework. The likes of Reynolds et al. (2003) have also used it to provide the rationale for their treatment methods although this is not an argument made by the proponents of the theory themselves.

The structure and function of the cerebellum is first described to give an understanding of the rationale behind this hypothesis.

5.1 Cerebellar neuro-anatomy

The cerebellum is one of two sub-cortical structures that enlarged during primate evolution[21] and is of a much larger size than that of any other species (Leonard, 1998). Located posterior to the brain-stem and below the occipital lobe (Figure 5) its densely packed and deeply folded structure means that it accounts for 10-15% of the weight and 40% of the surface area (Fawcett and Nicolson, 2001) of the brain. Some estimate that 80-85% of the neurons of the entire brain are located here (cited in Herrup and Keumerle, 1997).

[21] The other being the basal ganglia.

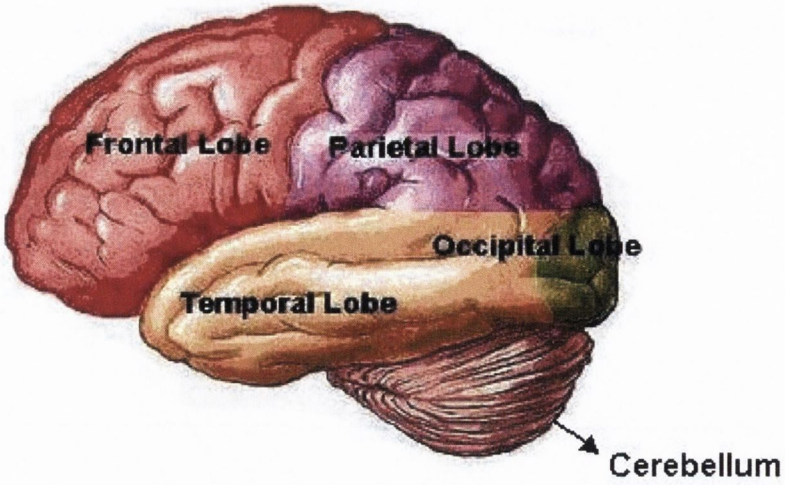

Figure 5: Location of the human cerebellum[22].

5.1.1 The cerebellar cortex and deep nuclei

The cerebellar cortex can be divided up into three phylogenetic structures; the archi-cerebellum, the paleo-cerebellum and the neo-cerebellum, and these approximate fairly well to three separate functional divisions of centrally located neurons, determined by the afferent fibres they receive. Thus, the archi-cerebellum (also referred to as the flocculo-nodular lobe) reflects the vestibular-cerebellum which is connected to the fastigial nucleus in the core of the cerebellum (i.e., the deep cerebellar nuclei). The paleo-cerebellum corresponds to the spino-cerebellum and is connected to the deep interpositus nuclei (consisting of both emboliform and globose nuclei, though it also has some connections to the fastigial nuclei). Finally, the neo-cerebellum corresponds to the ponto- (or cerebro-) cerebellum and is connected to the dentate nuclei, which represent approximately 90% of the cerebellar nuclei (Colin et al., 2002).

5.1.2 Afferent pathways

There are a huge number of afferent fibres which feed sensory information to the cerebellar cortex. Excitatory in nature, these afferent fibres outnumber efferent fibres by 40 to 1; they contain more neurons and synapses than the rest of the brain together (Colin et al., 2002) and represent some of the most powerful excitatory synapses in the

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[22] http://www.techfak.uni-bielefeld.de/GK518/projekte/SOddo/projekt.html]

entire brain (Fabbro, 2000). They enter the cerebellum through the superior, middle and inferior peduncles of the pons and project their axons to the deep nuclei and the purkinje cells of the cerebellar cortex. The vestibular-cerebellum receives afferent pathways from the vestibular nuclei and from the labyrinthine tract; the spino-cerebellum receives them from the dorsal and ventral spino-cerebellar tracts, and the ponto-cerebellum receives fast-conducting mossy fibres from the pontine nuclei (mossy fibres are generally accepted to transmit information regarding the context in which movement takes place). In addition, each of these anatomical areas also receives afferent information in the form of climbing fibres from the contra-lateral inferior olive, located in the medulla. Climbing fibres transmit information about the efficacy of movement so that this can be modified if necessary (Yeo and Hesslow, 1998). Thus, the main afferents are either sensory or cortico-cerebral, (though they also come from the reticular formation and the hypothalamus) (Fabbro, 2000).

5.1.3 Efferent pathways

Efferent pathways leave the cerebellum's vestibular nuclei through the superior and inferior peduncles. The purkinje cells, which are the main neurons of the cerebral cortex and the only source of output from the cerebellar cortex (Allen and Courchesne, 2003), send most of their axons to the deep cerebellar nuclei, though some also go straight from the flocculo-nodular lobe and the vermis (mid-line of the cerebellum) to the vestibular nuclei. Efferent pathways from the purkinje cells are inhibitory in nature. The fastigial nucleus of the vestibulo-cerebellum returns fibres to the vestibular nuclei and the inferior olive, as well as to the superior-colliculus (which serves visual reflexes to moving stimuli). It also sends fibres to the reticular formation, the spinal cord and various thalamic nuclei. The inter-positus nuclei of the spino-cerebellum send fibres to the mid-brain's tegmental tract (i.e., the magnocellular part of the red nucleus) which in turn goes to the inferior olive. In addition it sends fibres to the reticular formation, the peri-aqueductal grey matter, the pontine nuclei and superior-colliculus. The dentate nuclei of the ponto-cerebellum send fibres to the thalamic nuclei, the intra-laminar nuclei, the cerebral cortex and the parvocellular part of the red nucleus. All of these structures essentially act as relay stations, before information is passed to its final destination. The intra-laminar and thalamic nuclei, for example, project to the parietal,

temporal and frontal lobes (motor, pre-motor, cingulate and pre-frontal areas) (Fabbro, 2000). In particular, the medial part of the thalamic nucleus projects to Broca's area (44, 45) in the frontal lobe (Leiner et al., 1989). Between them, therefore, the vestibular nuclei project to the higher centres of the frontal lobe, the spinal tract engaged in motor reflexes, the visual-motor nerves engaged in eye movements and autonomic nervous system involved in arousal and inhibition. The lateral, medial, superior and inferior vestibular nuclei are the main efferents from the cerebellum; the lateral nucleus leads to the trunk and limbs; the medial nucleus leads to the neck and oculo muscles, hence resulting in positional and righting reflexes (Kohen-Raz, 1996).

5.1.4 Circuits involving the cerebellum

Through vestibulo-cerebellar-vestibulo loops, including spino-cerebellar and cuneo-cerebellar tracts, the oldest part of the cerebellum (the vestibulo-cerebellum) is involved in proprioceptive awareness, allowing for the regulation of equilibrium during posture and gait (i.e., balance) as well as co-ordination of head and eye movements (ocular motility) (Fabbro, 2000). The next phylogenetically oldest structure is the spino-cerebellum, which, through the reticulo-spinal tracts and spino-cerebello-rubro-spinal loops, is concerned with many basic aspects of movements including muscle tone, posture and vegetative regulation as well as the control of movement from the level of the spinal cord (i.e., execution of movement). The vermis area controls the proximal muscles and the lateral areas control the distal muscles. The ponto-cerebellum, also known as the neo- (i.e., "new") cerebellum represents that part of the cerebellum which is of exaggerated size in humans, enlarging slowly over our evolution and is principally involved in the control of volitional and skilled movements through the cortico-spinal tract. However, there are also claims of its involvement in language, both in production of language and in more cognitive aspects of language. The superior and inferior parietal lobes, involved in vigilance, focussing attention and the integration of visual, vestibular and somatosensory information send afferent projections to the cerebellum as does the temporal lobe, which also contributes to the integration of sensory processes. Projections from the frontal and prefrontal lobes carry information concerning eye-movement, working memory and the planning of actions; they also carry information from Broca's area (Fabbro, 2000).

While sub-cortical structures were developing during primate evolution, so too were cortical structures. In fact, the phylogenetic development of the neo-cerebellum and parts of the frontal cortex (specifically the prefrontal cortex) occurred in parallel and these are also the last two areas to develop fully ontogenetically (Diamond, 2000). Diamond points out that the cerebellum is responsible for those same cognitive functions as the dorso-lateral prefrontal cortex. As such the two are intricately connected in a system of cerebrocortico-rubro-olivo-neodentato-cerebellocortico loops; similarly, the cerebellum and posterior lobes are connected in a system of cerebrocortico-ponto-cerebellocortico-dentato-thalamo-cerebrocrotical loops (Fabbro, 2000). These are the main circuits involved in voluntary movement and cognitive / language functions and evidence from both neuro-imaging and brain-damaged patients demonstrates that there is co-activation between the cerebellum and the frontal lobes (with high firing of cerebellar neurons) on tasks such as verb-generation, verbal fluency and memory-load, particularly where the task is difficult, novel, unstable, fast and demanding of concentration (see Diamond, 2000, for a review). Thus, as Courchesne (1997) points out, abnormality in one site can set off a chain of adverse affects which alters growth in other sites, which would otherwise have been normal. The cerebellum acts as a sub-cortical information processing link between the temporal and frontal lobes involved in language (Leiner et al., 1989).

5.2 The functional cerebellum

Sensory information travels to the cerebral cortex, connecting to the deep nuclei via purkinje axons, which have an inhibitory effect. The cerebellum essentially acts as a processor for all of this sensory information, filtering out what is not needed and integrating what is, in order to co-ordinate movements / responses. This explains why there are so many more afferent than efferent fibres. Roberts (1967) regarded the cerebellum's function as "improving the smoothness of the reaction to the unforeseen", so while the spinal reflexes are responsible for many vestibular reflexes, the cerebellum's heavily myelinated pathways allow for more organised, well-designed, as well as rapid, adaptive responses (Kohen-Raz, 1996).

The cerebellum is generally accepted to be involved in proceduralising skills; that is, processing commands to execute tasks so that they may then become fluent and co-ordinated (Nicolson and Fawcett, 2001). This is afforded, perhaps, by efficiency in modulation, as described by Ayres (1979). A system which does not "learn" which

stimuli and responses to inhibit, and which to facilitate, is not an efficient modulating system. It is argued that such skills include not only the modification of existing (motor) reflexes but also the learning of novel behaviours (Yeo and Hesslow, 1998).

The "Cerebellar Cognitive Affective Syndrome", described by Schmahmann and Sherman (1998) extended our conceptualisation of the cerebellum's function beyond that of pure motor skills, assigning different topographies different functions. The hemispheres, they suggest, are involved in cognitive functions whereas the vermis is involved in emotional functions and there is plenty of evidence from psychiatric patients to support this (see Rapoport, 2001, for a review). Exaggerated examples of this are seen in children who have undergone surgical removal of cerebellar tumours, where autistic- and attention-deficit- like behaviours have ensued. All parts of the cortical hemispheres are mapped onto the cerebellum (Kohen-Raz, 1996).

5.2.1 Evidence of motor involvement

The cerebellum was once considered to be exclusively involved in motor co-ordination and there is no doubt that it is in part a coordinator for motor activity. The early studies of Flourens (1824) and Luciani (1915) clearly linked the structure to motor behaviour and it is known that the cerebellum projects to the thalamus and then on to areas 4 and 6 of the frontal lobe, which are responsible for regulating movement and posture (Leiner et al., 1989). It is involved in reflexive movement through the vestibular spinal tract; disturbed reflexes are a classic result of cerebellectomy or hemicerebellectomy (e.g., Russell, 1894; Rabacchi et al., 1986). It is involved in motor adaptation, acquisition (novel and / or integration) and classical conditioning (Daum et al., 2001).

5.2.2 Evidence of cognitive involvement

It is only quite recently that the idea of cerebellar involvement in cognitive processing has been embraced, although Gowens first touted the idea of neo-cerebellar contribution to cognition as far back as 1888. Silveri and Misciagna (2000) suggest that this is because the resulting cognitive deficit after cerebellar trauma is usually sub-clinical and obscured by the severity of the motor impairment.

Mice with degenerated purkinje cells can retain motor skills whilst still showing evidence of impaired non-motor skills (i.e., visuo-spatial learning deficits) (e.g., Lalonde & Strazielle, 2003). Using fMRI techniques, clear evidence of an anatomical dissociation has been demonstrated between motor tasks and cognitive tasks such as attention (e.g., Allen and Courchesne, 2003), with attention being localised to the superior posterior cerebellar hemispheres and motor tasks to the anterior cerebellar hemispheres. Allen and Courchesne even demonstrated a correlation between cerebellar lobule size and amount of attention activation. Riva and Giorgi (2000) found clear cognitive deficits (i.e., non-verbal processing, naming and comprehension, executive and time-based tests) in children who had undergone surgical treatment of the cerebellum but who nevertheless showed intact motor skills.

The cerebellum's involvement in speech and language at some level is not in question. There is evidence of cerebellar involvement in such tasks as lexical retrieval (e.g., Fiez et al., 1992), verbal fluency (e.g., Appollonio et al., 1993), synonym generation and syntactic comprehension (Fabbro et al., 2000) and verbal working memory (e.g., Silveri et al., 1998). Fabbro et al. (2000) report that the right cerebellar hemisphere and some structures of the vermis are most commonly implicated in these difficulties. For example, Silveri et al. (1994) and Zettin et al. (1997) reported agrammatic speech in those with right cerebellar hemisphere damage.

5.2.3 Evidence against cerebellum's involvement in pure cognitive tasks

However, Justus and Ivry (2001) claim that the evidence for learning, memory, attention and visuo-spatial processing is less consistent than for speech and language, and that where cognitive effects are seen, these could also be explained by articulatory or temporal aspects of the task. For example, the cerebellum is active when planning or contemplating movement, which blurs the boundaries between what can be considered a motor and what can be considered a cognitive task (Barinaga, 1996). Excision of the vermis in children with cerebellar tumours seems to alter initiation of articulation and the mechanical process of speech vocalisation (see Riva and Giorgi, 2000), supporting Ivry's belief that the cerebellum is really involved in the preparation of movement, with all cognitive work done by the cerebral cortex (Barinaga, 1996). Ackermann et al. (1998) used fMRI to replicate previous PET studies showing activation of the right

cerebellar hemisphere during verbal tasks. However, whereas previous studies had attributed this activation to cognitive aspects of speech production, Ackermann et al. simply requested their subjects to engage in a simple and over-learned task, mentally reciting the months of the year, and as such, hardly engaged in higher level processing. Thus, they attributed activation of the right cerebellar hemisphere to "inner speech" which they claimed subliminally activated orofacial and laryngeal muscles. Given the hundreds of muscles involved in verbal communication, which between them control respiration, phonation, articulation, resonance and prosody (Ackermann and Hertrich, 2000), it is unlikely that speech would escape any cerebellar impairment. Fabbro et al.'s (2000) conclusion that different parts of the cerebellum are responsible for different language skills does not necessarily discount the validity of Ivry's belief. Findings which link more cerebellar activation with more difficult cognitive tasks, are also explained using the "inner speech" explanation; greater semantic activation means greater sub-vocal representation, with more words being prepared for possible articulation (Ackermann et al., 1998). (Their suggestions focus on the "where" of dysfunction anyway; the question of *how* they contribute to such skills is still unanswered).

5.2.4 Models of the cerebellum

Justus and Ivry's (2001) argument concerns the idea that what appear to be non-motor aspects of language could be secondary to some other basic function of the cerebellum. This reflects Topka and Massaquoi's (2002) question of whether a single elementary cerebellar function is common to all cerebellar motor functions. Although different anatomical cerebellar regions have been linked to distinct behaviours, the identical neural architecture across the cerebellum suggests that the signal processing for each area is the same (Bloedel, 1992). This would seem to suggest that the cerebellum aids any area of the brain with which it has reciprocal connections (Allin et al., 2001) and should, therefore, be viewed as a modulator of systems.

There have been many models of basic cerebellar function: performance regulation; learning; timing and sensory integration. However, distinguishing one from the other is not an easy task. The first of these (e.g., Paulin, 1993; 1997) suggests that the cerebellum is responsible for the neural representations of movement by the body,

as well as objects in the environment. The cerebellum as a controller of movement, by comparing intended movement with actual performance (Ghez, 1991), has been suggested. When a neural impulse (programme) is sent to a muscle, a copy of this is also sent to the higher cognitive centres so that any mismatch can be detected. Impaired movement may result either from the generation of an inadequate programme, from error in execution or from unexpected changes in the environment. Failure to make use of the ensuing sensory feedback in order to compare intentional and actual performance, or a problem with the sending / receiving of this sensory feedback, could also result in impaired performance (Laszlow and Bairstow, 1985). However, this "comparison" role could also be secondary to some other basic role. For example, Leiner et al. (1989) suggest that the cerebellum is responsible for improving speed and skill, whatever the performance in question. In addition, Massaquoi and Topka point out that it is often overlooked in studies of cerebellar function that it is the small anterior lobe that is largely responsible for movement control, and as such, there is still the rest of the complex cerebellar system to account for. Courchesne and Allen (1997) suggest a more basic role for the cerebellum, namely that it is engaged in general preparation, especially for predicted stimuli, which affords a quicker and more efficient response. Many studies, most of which use eye-blink conditioning (i.e., a form of procedural learning), have linked learning to the cerebellum. Steinmetz et al. (1992), whose sample had interpositus lesions, failed to eye-blink in response to a simple classically-conditioned stimulus and they insisted this was due to a failure to *learn* the response rather than simply an inability to *perform* the task. Reinforcing this finding are those studies which have failed to find any conditioned learning whilst the cerebellum has been artificially inactivated (see Yeo and Hesslow, 1998) or damaged. Fiez et al. (1993) suggested that the cerebellum functions as an error detector and Marr (1969) that it functions as a modifiable pattern detector, allowing patterns to be modified gradually with experience. Both could contribute to learning by correcting movements in order to reduce error and improve subsequent performance. Allen and Courchesne (2003) have suggested that the cerebellum's role is to predict relationships among sequences of events so that the appropriate responses can be generated as quickly as possible. However, this may also be a result of a more general defect in timing (Lalonde and Botez-Marquard, 2000). The role of the cerebellum's hemispheres as time-keepers, (e.g., Ivry and Keele, 1989), modifying the length and force of muscular contractions to ensure smooth, co-ordinated simple and complex movements,

is another contender. Riva and Giorgi (2000) found that all of the children who had had cerebellar surgery in their study, performed poorly on time-based tests; Casini and Ivry found poorer performances in judging time intervals by those with cerebellar lesions. It makes sense that an impairment in time estimation would affect sound perception and production, and this has been demonstrated using Voice Onset Time (VOT) tasks where more difficulty was exhibited by the cerebellar patient group (than controls) in making voicing distinctions when engaged in auditory perception of similarly sounding words (i.e., "Daten" and "Taten" in the German language (Ackermann and Hertrich, 1997)). However, Mauk et al. (2000) conclude that such theories are not really mutually exclusive; for example, sequence is not the only important consideration in movement because it may not result in movement at the appropriate time (Medina et al., 2000). Both Gao et al. (1996) and Bower and Parsons (2003) suggest that the cerebellum's primary function is in the acquisition of sensory information in order to guide behaviour appropriately and this is why it appears to be involved in not just motor but cognitive tasks. Thus, it seems that if there is one true underlying mechanism, it is yet to be identified.

Another function of the cerebellum which has gained recent popularity (though it was first suggested by Horsely, 1897) is the cerebellum's role in inhibition. Given the amount of sensory information arriving at the cerebellum (as mentioned, afferent neurons outnumber efferent ones by 40:1), plus the inhibitory nature of the purkinje cells, it could be that the cerebellum contributes to proceduralisation and learning by eliminating irrelevant information and responses (see figure 6).

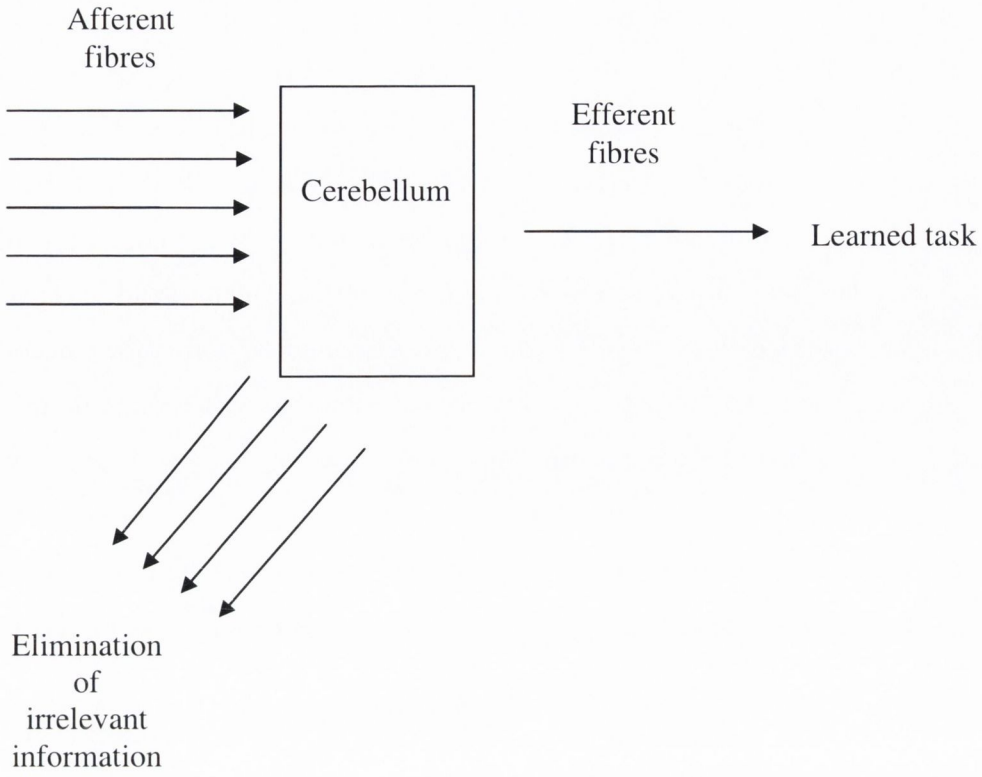

Figure 6: Illustration of how the cerebellum contributes to learning by eliminating irrelevant information.

5.3 Normal cerebellar development

Within the Central Nervous System, the cerebellum takes longer to develop in the embryo than any other neurological structure (Madsen et al., 2002). Emerging from the hindbrain and the posterior part of the midbrain at approximately 32 days gestation, it continues to form until 1 year post-natally. The postero-lateral fissure, dividing the flocculo-nodular lobe (archi- / vestibulo-cerebellum) from the posterior lobe begins around day 45 developing until about day 63 (week 9) around which time the primary fissure begins to distinguish the anterior lobe from the posterior lobe. The mid-line, or vermis (paleo-cerebellum) appears about the eighth week, taking a further 10 weeks to develop more fully. Mass expansion in all directions occurs just after this (at around 20 weeks) with the cerebellar hemispheres becoming obvious as lateral masses of dentate nuclei (the neo-cerebellum). At about 12 weeks, the primitive neuro-cells (neuroblasts) are generated and the deep cerebellar nuclei start to form. The neuroblasts migrate to their respective locations from the ventricular epithelium; firstly, the Purkinje

neuroblasts migrate outwards between the 17-28th weeks, followed by inward migration of the Granule neuroblasts at about 30 weeks. This latter migration continues until after birth, though they then disappear over the first year of life.

The long development of the cerebellum makes it a structure at risk of teratogenic damage (Ciesielski et al., 1997) and it is known to be more vulnerable around the time of birth than other structures of the brain due to the increase and migration of granule cells (Jacobson, 1991), which could have ramifications for subsequent cellular development. Developmental abnormalities, according to Madsen et al. (2002), are best categorised according to gestational timing, and therefore, include disorders of neural tube development, neural / glial development and myelination / cortical maturation. Sporadic cerebellar hypoplasia, as Riva (2000) points out, could result in different developmental disabilities including global delay, learning disability and attention deficits and research has certainly found a correlation between pre-term infants, cerebellar size and subsequent cognitive deficit (Allin et al., 2001).

As Zeffiro and Eden (2001) point out, knowing the neurogenesis and maturation of the cerebellum may help us to understand at what point such neuro-developmental disorders begin in the human brain and why so much overlap between them is seen. As previously mentioned, myelination formation occurs over time, with functional systems developing together (deQuiros and Schrager, 1978). Myelin attack, therefore, could affect a specific set of neurons, such as the vestibular-cerebellar or cerebellar-vestibular nerves, which are usually among the first of the sensory neurons to be myelinated. This would manifest in aberrant control of eye movements as well as balance.

5.3.1 Returning to the issue of co-morbidity

Because of the overlap in symptoms between dyslexics and other neurological disorders, it is possible that they share some cerebellar dysfunction. Studies have found overlap in the performance of children with dyslexia and Developmental Coordination Disorder (DCD) on continuous timing tasks (e.g., Wolff et al., 1990; Portwood, 1999, 2000]); between dyslexics and children with ADHD on motor coordination tasks (Denckla and Rudel, 1978); between dyslexics and children with speech and language problems (Bishop, 1997; Leonard, 1998) and even dyslexics and children with autism /

Asperger's on tests of visual-motion integration (Talcott et al., 2000; Gepner and Mestre, 2002). Furthermore, there is evidence in Autistic brains of vermal hypoplasia and a reduction in purkinje neurons of the posterior cerebellar vermis and hemispheres (Courchesne, 1997) especially lobules VI and VII (Riva, 2000). The effects of acquired cerebellar diseases also support a vermal role in emotional and social behaviours as well as verbal and non-verbal behaviour. In ADHD brains there is evidence of significantly smaller cerebellums than in normal brains, though the cerebellum is not exclusively different in these children. Other areas are also different (Castellanos et al, 1996). Berquin et al. (1998) found a significantly smaller vermis in ADHD boys, particularly of the posterior, inferior (but not superior) lobe, with lobules VIII, IX and X affected (Riva, 2000). It seems plausible, therefore, that depending on the timing of the genetic / environmental teratogenic interaction (i.e., the "window of vulnerability"), the consequences of the impaired cerebellar formation could differ from child to child, or may overlap. This would explain why a range of disorders may cluster in families and also why the disorders are so often co-morbid in one child. Thus, a child showing signs of autism might have experienced teratogenic insult at the very early stage of neural tube closure (Kemper and Bauman, 1998) and the evidence suggests that the window of vulnerability in Autistics occurs around the 5th week of gestation (Courchesne, 1997), thereby affecting the vermis of the cerebellum. Another child in the same family, however, may experience insult at a later stage (thereby affecting the lateral cerebellar hemispheres) and as such show signs of dyslexia. Indeed another child in the same family who was exposed when cerebellar structures were forming in parallel may consequently show multiple neuro-developmental symptoms (e.g., ADHD and dyslexia or ADHD and DCD).

More research is needed to identify the window of vulnerability in dyslexic children, although it seems possible that some impairment implicating the neo-cerebellum, and its neural connections to the relevant areas of the prefrontal lobes, has occurred. Because both of these areas take longer to develop than the rest of the brain, one might hypothesise that for either directly genetic reasons, environmental reasons, or, more likely, some interaction between the two (i.e., genetic vulnerability to environmental stimuli) neuronal migration or development may have disturbed the development of inhibitory efferent projections from the neo-cerebellum. This could have consequences for the subsequent formation of neural circuitry, allowing hard-wired, primitive reflexes to prevail or dominate. It could also have consequences for

the adequate development of those heavily myelinated axons, traced by McGuire et al. (1989) using the antibody CAT-301, of which the magnocells are part. The dentate nucleus which receives projections from the neo-cerebellum contains both magno- and parvo-cells.

5.4 The motor theory of speech

As we have seen from studies such as those reviewed by Riva and Giorgi (2000), parts of the cerebellum (i.e., the vermis) seem to be involved in the articulatory initiation and the mechanical aspects of speech. It is known that the cerebellum sends projections to Broca's area (areas 44 and 45), usually thought of as a motor area, and which is responsible for language articulation. However, although Leiner et al. (1989) ascribe pure cognitive functions, such as word retrieval, to the cerebellum, Justus and Ivry (2001) continue to emphasise its motor basis, through temporal regulation (Ivry and Keele, 2001). With regard to the former, temporal precision is crucial for phonological processing; impairment of this should, therefore, predict impaired speech perception and this is what Ackermann et al. (1997) found when they controlled for confounding factors such as intensity of aspiration noise.

Liberman and Mattingly's (1985) "motor theory of speech" suggests that phonemic awareness depends upon articulatory awareness. That is, the "motions" that people detect when listening to others are then matched to their own performance of these motions as they hear the associated sounds. None of this must necessarily be done at the physical level; such motions can be made at the neurological level, in the biologically distinct module linking speech perception and production. If, as the timing theory would suggest, there is interference in either the detection of others' articulated sequence or one's own sequence of articulation, there should result a faulty representation of sounds. The theory bears a resemblance to, and a development of, the findings that babies can match aurally perceived phonetic information to face and voice (e.g., Kuhl and Meltzoff, 1982) and that this is present from at least 2 months of age, suggesting that our brains may be hard-wired for the processing of phonetic information (Patterson and Werker, 2003). Kuhl and Meltzoff suggest that the baby's tendency to imitate sounds may be due to their knowledge of the relationship between hearing and articulation. A study by deGelder and Vroomen (1998) found that poor readers (i.e., dyslexic readers) were impaired not just on auditory processing but on visual speech processing as well (i.e., the perception of another's mouth movement

during speech); they concluded that poor readers were impaired on speech-processing in general, thus providing support for the motor theory of speech. In a study of dyslexics, Wolff (1999) found that 90% of those with motor coordination problems also performed poorly on tests of motor speech, as measured by repetitious syllable production. Although studies are needed to explore whether dyslexia "at risk" infants are less likely to match this aurally perceived phonetic information to face / voice than are control infants, and longitudinal studies necessary to follow such infants to ages of measurable literacy ability, the motor theory of speech has allowed Fawcett and Nicolson (2004) to suggest a link between motor impairments and the phonological reading / spelling difficulties experienced by dyslexics.

A model of working memory proposed by Baddeley in the 1960s and revised recently (2002) may theoretically support Liberman and Mattingly's theory. Baddeley et al. (1998) suggested that language development (either natively in children or as a foreign language in adults) is determined by rehearsal accommodated for by the articulatory loop, from a phonological store. Important in this articulatory loop is a central speech-motor program. If this is disturbed then language may fail to develop (Caplan and Water, 1995) regardless of the contribution of speech musculature to language (Baddeley and Wilson, 1985). The ideas of a central speech-motor program prevents the model being criticized on the grounds that deaf or mute people develop literacy.

Liberman and Mattingly's theory represents an extension of Baddeley's ideas in terms of phonemic awareness, and subsequent theories since Liberman and Mattingly's idea (e.g., the phonological deficit hypothesis) link phonemic awareness to literacy skills (Figure 7). Evidence of verbal short-term memory problems in patients with right cerebellar damage led Silveri and Misciagna (2000) to suggest that the resulting deficits were not true aphasia but a result of impaired working memory. Botez-Marquard et al. (1996) also found evidence of impaired working memory in patients with severe olivopontocerebellar atrophy (OPCA) compared to those with mild atrophy.

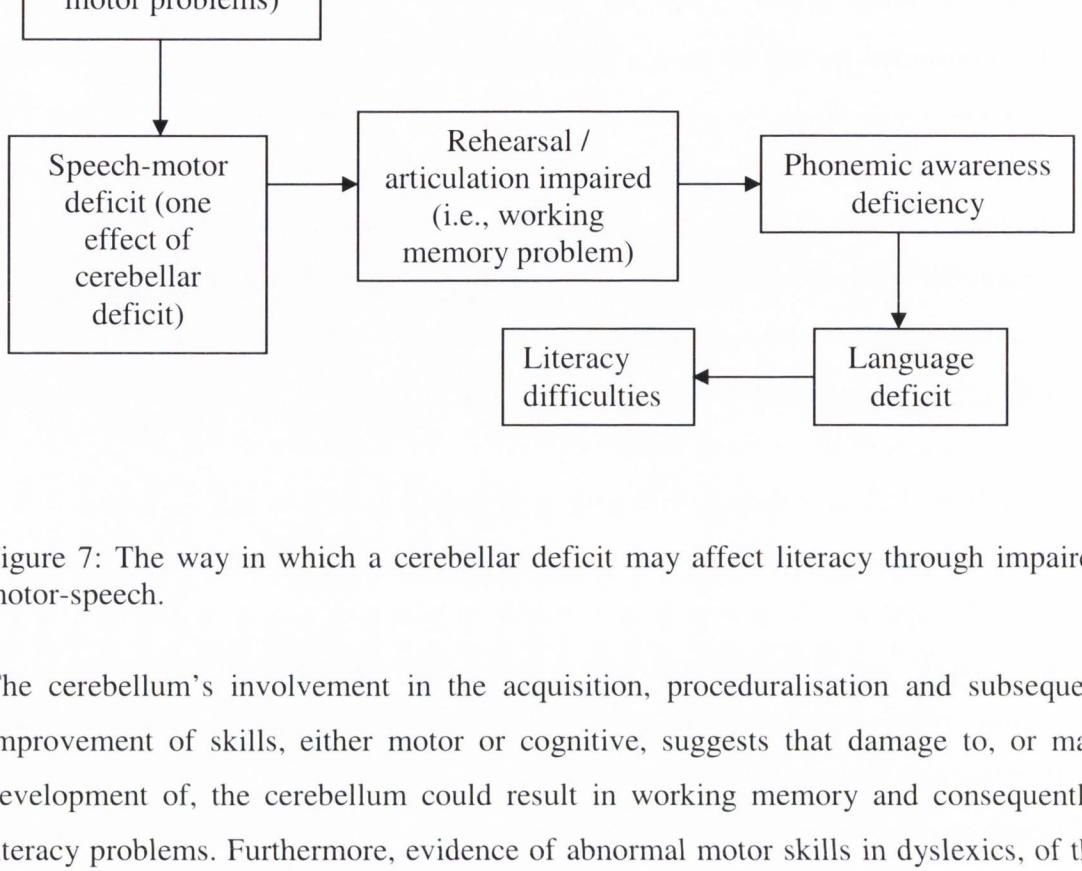

Figure 7: The way in which a cerebellar deficit may affect literacy through impaired motor-speech.

The cerebellum's involvement in the acquisition, proceduralisation and subsequent improvement of skills, either motor or cognitive, suggests that damage to, or mal-development of, the cerebellum could result in working memory and consequently, literacy problems. Furthermore, evidence of abnormal motor skills in dyslexics, of the cerebellum's involvement in motor skills, of the cerebellum's involvement in cognitive skills, as well as an explanatory theory for the cerebellum's importance in language development, have led Nicolson and Fawcett to suggest the cerebellar deficit hypothesis as a conceptual framework for understanding the causal origins of the symptoms of developmental dyslexia.

5.5 The Sheffield hypotheses

The problem with some theories, such as the phonological deficit hypothesis and visual processing deficit hypotheses is that, although descriptively accurate, they limit the dyslexic deficits to a single modality (Nicolson and Fawcett, 1994a) thereby failing to offer a complete explanation for the range of symptoms dyslexics exhibit (Fawcett et al., 1996). Dyslexic and non-dyslexic groups can be differentiated not just on the basis of phonological skill and literacy attainments, but on the basis of general reaction times (as measured by stimulus classification speed) and a wide range of tasks (e.g., phonology, speed, motor skill, visual flicker, binocular control, auditory perception),

with dyslexics doing worse than the controls every time (Nicolson and Fawcett, 1994a; 1994b; 2001).

The cerebellar deficit hypothesis, however, may account for every modality and a range of symptoms, whilst also accounting or allowing for other hypotheses. It grew out of the more descriptive Dyslexia Automaticity Deficit (DAD) hypothesis, which itself incorporates the Conscious Compensation hypothesis (CC). The cerebellar deficit hypothesis, therefore, accounts for the original hypotheses whilst functionally anchoring and explaining the deficit's source to specified locations of the brain.

5.5.1 The Dyslexia Automaticity Deficit and Conscious Compensation Hypotheses

The automaticity deficit hypothesis argues that dyslexic children have more difficulty with general skill acquisition (i.e., learning), than with specific skills such as reading. This is not a new notion – it was commented upon by Morrison in 1985 (p. 89). The conscious compensation hypothesis claims that dyslexics must work harder than non-dyslexics to become proficient at such skills. Automatic processes are those that are independent of the limitations of a central processor (i.e., time or space-wise), which are involuntary and which require extensive practice (Regan, 1981). LaBerge and Samuals (1974) postulated the idea of a reading acquisition model in which the learner passed through two phases - "Attention" and "Mastery" - before acquiring "Full Automaticity" of a skill without any conscious involvement (though Perfetti (1985) proposed that in fact very few people reached a level of Full Automaticity). The suggestion that dyslexics' problems lay in automatising skills was a topic of investigation as far back as 1971 when Eakin and Douglas explored it with reading disordered children and this was supported by findings of Denckla and Rudel (1976) who also found that dyslexic children were significantly slower on tasks of rapid number, letter, object and colour naming than were both normal control and non-dyslexic reading disabled children.

These hypotheses do not deny that dyslexics *can* become skilled, simply that they must employ coping strategies in order to do so; if present, other factors, such as general intelligence, motivation, practice and genetic endowment will determine eventually how well a skill is mastered. Varying levels of the influence of these factors

in different children with dyslexia may, in fact, be the cause of what appear to be different subtypes (Fawcett et al., 1996).

The extra resources necessary for coping strategies only become apparent when the initial task becomes more stressful, as when the task is complex or is subject to time pressures (Fawcett and Nicolson, 1994; Yap and Van der Leij 1994b). For example, dyslexic and non-dyslexic children may perform equally well on any given single-task (due to conscious compensation), but the one explicitly used by Nicolson and Fawcett (1990) was that of motor balance. Motor skills are useful indications of learning skills because of the practice needed to integrate the many subunits into a smooth, unitary movement (Regan, 1981). However, for dyslexic children, the introduction of a second, distracter, task (e.g., counting backwards), affects their CC coping mechanisms to an unsustainable level and, thus, performance on the initial task deteriorates. This type of dual-task performance is a common method of assessing the degree of automaticity of a skill (Jenkins et al., 1994) and demonstrates that it is by "working harder" that the dyslexic children perform adequately on single tasks. Interference with performance is attributed to the reduction of conscious compensation rather than the additional attention requirements of a further task, because results are identical to those found when children are blind-folded (Fawcett and Nicolson, 1992), a measure which, according to Fawcett and Nicolson (1994), prevents conscious compensation without introducing the complications of a dual-task paradigm. In support of this, recent evidence of significant differences between dyslexic and control groups was found in rhythm reproduction tasks when task complexity increased, though in accordance with Nicolson and Fawcett's own studies, performances were similar for simpler tasks of reproducing simple rhythm patterns (Tiffin-Richards et al., 2004).

Smits-Engelsman et al. (2003) support the idea of an automatisation deficit in children with reading difficulties. Their participants were asked to draw a straight line between two targets in two different conditions, firstly in one direction followed by reversing the line (discrete aiming movement), and secondly by moving from one target to the other as fast as possible (cyclic aiming movement). They found that children co-morbid with learning disabilities and developmental coordination disorder performed significantly worse than control children but only on the cyclic aiming movement. Such children, they suggest, need to rely upon feedback during their movement execution in order to plan ahead which places extra demands upon their motor control.

In training programmes designed to test long-term learning and identify the stage of difficulty, Nicolson and Fawcett's (1994a; 2000) dyslexic sample showed problems with initial proceduralisation of skill, the quality of the skill acquired and the rate of improvement of that skill, compared to control samples, although similar patterns of "unlearning" then "relearning" the task, between the 2 groups indicate that the eventual strength of automatisation in dyslexics is unimpaired. Nicolson and Fawcett suggest that it will take a dyslexic 10 times as long as a non-dyslexic to achieve equivalent gains. Furthermore, and crucial in the argument for a causational difference between "backward readers" and dyslexics, is the demonstration of a qualitative difference between reading age controls and dyslexics' performance, not just on those dual-task tests administered by Nicolson and Fawcett, but on other tests of "automaticity" such as Denckla and Rudel's "Rapid Automatised Naming" task. In this task, the child is presented with a series of everyday pictures printed on a page and is required to name them as fast as he can in a sequential manner. This is suggestive of a contributing factor which is distinct from linguistic skills alone.

5.5.2 The Cerebellar Deficit Hypothesis (CDH)

The automaticity hypothesis, however, is not a causal explanation, merely a cognitively descriptive one. As Yap and van der Leij (1994) point out, poor automatisation may itself be a surface symptom reflecting a deficit in some underlying mechanism. The neural mechanisms necessary for a true causal explanation are better provided for by the cerebellar deficit hypothesis. This asserts that dyslexics have some impairment in their cerebellum. The cerebellum is considered the centre of skill learning in the brain allowing new skills to be both acquired and made automatic. Once the cerebellum has done its job during development, the skill in question (i.e., in this case phonological representation) may be accessed without the cerebellum (Zeffiro and Eden, 2001). Thus, trauma to this region would not necessarily result in acquired dyslexia.

5.5.3 Empirical evidence

In recent years much of the exploratory work concerning the cerebellum and dyslexia has been conducted by Nicolson and Fawcett of Sheffield University. They claim that

tasks known to be linked to cerebellar function (even if not entirely specific to it), such as eye-blink and time-estimation have been shown to be deficient in 80% of their dyslexic sample, with no overlap between dyslexics and controls. Time-estimation is one cerebellar task which lies outside of the motor domain and it supports Ivry and Keele's (1989) hypothesis of the cerebellum's role as a time-keeper. They found that patients with cerebellar damage showed evidence of impairment in judgements of time-estimation but not of loudness-estimation (i.e., the control judgement task). Since then, Wolff et al. (1990) have demonstrated a significant difference in the performance of dyslexic and non-dyslexics in their ability to engage in precisely timed bimanual coordination (on fast and asynchronous conditions only), and the Sheffield group have replicated this deficiency. For example, Nicolson et al. (1995) showed that dyslexic children evidenced a dissociation between time-estimation (the cerebellar task) and loudness-estimation, performing significantly worse than their reading-aged controls on the experimental task but equivalently on the control task to their age-matched controls. Subsequently, the simple conditioning-study of eye-blink response as a measure of cerebellar abnormality demonstrated group differences between dyslexics and non-dyslexics in terms of tuning (i.e., timing of blink) and habituation (number of blinks) and could suggest a functional abnormality connected with the cerebellum even if the specific pathway is not clear (Nicolson et al., 2002). This evidence supports their previous studies of cerebellar task differences between dyslexics and age- and reading-matched controls which showed the former to be significantly more impaired on a range of tasks (Fawcett et al., 1996). The tasks used were the same as those described by Dow and Moruzzi (1958) which tested for traditional signs of cerebellar dysfunction. The tasks[23] on which the dyslexics performed particularly poorly (i.e., even worse than their reading) included: finger and thumb opposition; tremor; arm displacement; toe tap; limb shake; dysdiadochokinesia[24]; postural stability and muscle tone.

Nicolson and Fawcett (1999) are careful in their writing to adopt a speculative stance with regard to their theory and the possibility of the cerebellum's co-varying, rather than causal, role in dyslexia, however. Later, Fawcett and Nicolson (1999)

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[23] These are described in more detail in chapters 7 and 8.

[24] Diadochokinesia: Difficulty performing rapid alternating movements as with the fingers or hands (i.e., a soft-sign) (Reber, 1985, p. 230).

conducted a study to test the generalisability of their findings, changing their sample from their normal one (a previously recruited, faithful, and repeatedly tested sample for most of Nicolson and Fawcett's experimental hypotheses) to a larger community-based one (from private dyslexia schools). They reported that of the 59 diagnosed dyslexics, of whom 91% and 92% had reading and spelling difficulties respectively, 68-86% were "at risk" on 3 or more cerebellar tasks. In comparison, of the 67 control children, of whom 24% and 10% had reading and spelling difficulties respectively, only 16-18% were "at risk" on 3 or more cerebellar tests. Thus, although the overall effect size was lower, the results were similar.

Other support for the hypothesis that dyslexics don't automatise reading skills in the way that non-dyslexics do has been reported. Yap and Van der Leij (1994b) and Van der Leij and Van Daal (1999) consecutively showed that dyslexics have a significant deficit in automatic phonological decoding which was exacerbated by time-constraints, as well as a deficit in identification of highly-familiar words and other orthographic stimuli, also exacerbated by speed limitations, where scores were worse than those of reading-age controls. With regard to motor skills, Yap and Van der Leij (1994a) replicated part of the Nicolson and Fawcett (1990) study, showing significant impairment of balance skills in dyslexics (but only on the right leg).

Iversen et al. (2005) also found strong evidence for a relationship between reading difficulties and motor coordination problems. They showed that poor readers, regardless of whether this was caused by dyslexia or not, performed significantly less well on tasks of manual dexterity (particularly eye-hand coordination when measured by control of pen, and by coordination of two hands performing a single task) and tests of balance (particularly fast movements). In fact 60% of the children in their "dyslexic" group and 53% in their "poor readers" group scored at or below the 5th percentile compared to only 13% in the control group. Combined with those scoring at a borderline level, there were approximately 70%, 60% and 26% scoring poorly on overall motor coordination scores in the three groups respectively. Brookes and Stirling (2005) found a significant correlation between reading ability and both posture and complex movements (taken as representative of cerebellar functioning) which involved tests of fine-motor control and balance skill. However, their experimental design did not differentiate between different groups of poor readers

As we have seen earlier, McPhillips and Sheehy (2004) also found that at least one motor-based test of retained reflexes (the ATNR), and other tests of motor

development (i.e., "manual dexterity" and "balance" tasks from the Movement ABC[25] motor impairment test of Henderson and Sugden, 1992) could be used to discriminate between those with reading problems and those without.

Fawcett et al. (1996; 2001) make two further claims; firstly, that such findings as these persist even when the sample is screened for ADHD. Secondly, that cerebellar tests can discriminate not just generally between samples of dyslexics and non-dyslexics but that (static) cerebellar tests discriminate between *types* of poor readers. Sub-groups of poor-readers (i.e., those with low IQ) do not evidence such cerebellar impairment as do poor-readers with an average or higher IQ. This lends more weight to the idea that such tests reflect a distinct neurological condition, perhaps a result of a dysfunction with the lateral parts of the posterior lobe of the cerebellum (Fawcett et al., 2001), as the cause of poor reading rather than poor general ability (i.e., "garden variety poor readers).

5.5.4 Neurobiological evidence

Suggestive evidence of differences between dyslexics and non-dyslexics comes not just indirectly from behavioural data such as described above but also directly from neuro-physiological and neuro-anatomical studies, though imaging studies suffer from a serious lack of consistency.

Rae et al. (1998; 2002) found biochemical evidence of cerebellar differences in dyslexics compared with controls. These differences manifested as reduced overall cellular density bilaterally and reduced neuronal density of the left coupled with an increase on the right (i.e., cerebellar asymmetry). They suggest that the reductions are a result of smaller numbers of larger neurons, which is also consistent with the magnocellular deficit hypothesis. They also report that dyslexics' cerebellar grey matter ratio correlated significantly with nonsense word reading time, seeming to suggest a relationship between dyslexia severity and cerebellar symmetry (Rae et al, 2002) (though Beaton (2004) criticises the study on the basis of unknown co-morbid

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[25] The "Movement ABC" test battery consist of 8 subtests, divided into 3 sets of tests. These are "manual dexterity" which includes peg-board manipulation, threading bolts and line drawing; "ball skills" which includes ball bouncing (off a wall) and throwing a bean-bag into a box; and "balance" which includes balancing a ball on a board, hopping and balancing on a board (these make use of both static and dynamic tasks).

diagnoses; some subjects were thought also to have specific language impairment). Nicolson et al. (1999) point out that this is interesting given the evolution of this part of the cerebellum (i.e., the neo-cerebellum) relative to other primates, and its connection to both frontal motor and language (i.e., Broca's) areas of the brain. However, unlike Rae et al, Finch et al. (2002) did not find any evidence of cerebellar asymmetry. They did find evidence of increased purkinje cell areas in both the anterior and medial posterior cerebellar cortex, and in the inferior olive of Galaburda et al.'s (1985; 1994) dyslexic sample who had more large neurons and fewer small neurons compared to controls. They point out that this could be suggestive of a deficiency in the input to the cerebellum, from the inferior olive, rather than a deficiency of the output to the dentate nucleus. This is consistent with theories of sensory integration which claim that a brain deficient in processing / organising the flow of sensory impulses (in a manner which results in precise information about the person and his world) is a brain which cannot cope with ordinary stresses and demands, thereby resulting in a range of dysfunctional symptoms (Ayres, 1979). This includes vestibular and proprioceptive information as well as visual, auditory and tactile information and would result in poor motor coordination, muscle tonus and spatial awareness, making the acts of literacy both tiring and difficult. Given that the olivo-cerebello-dentate pathway aids the neo-cerebellum to engage in language tasks, it makes theoretical sense that a difference here might also result in dysfunctions at the language level.

Nicolson et al. (1999) replicated an earlier study conducted by Jenkins et al. (1994) in which they used Positron Emission Tomography (PET) to demonstrate cerebellar involvement in motor performance of both pre-learned and new tasks. Although Jenkins et al. found the cerebellum was activated by both of these tasks, it was more evident with newly-learned tasks. Nicolson et al. also found that a small sample of dyslexic adults had significantly less regional cerebral blood flow in the right cerebellum for pre-learned sequence and new sequence tasks than did controls (0.9% and 0.5% consecutively for dyslexics compared to 5.6% and 7.8% consecutively for controls), thus supporting the findings of Rae et al. (1998; 2000). They also showed other functional differences (as is typical with such studies); the controls evidenced more activation around the cingulate gyrus during the pre-learned sequence whereas the dyslexics showed more bilateral activation of the frontal lobes during the novel sequence. This could suggest that the dyslexics are bypassing the cerebellum and relying on conscious strategies for new learning (Fawcett and Nicolson, 2001). Thus,

problems are only evident when tasks must be processed rapidly (or are dual in nature) because this requires the additional involvement of the frontal cortex, which in dyslexics would already be in use.

These are important findings, not simply because they confirm that functional differences in learning do exist between dyslexics and non-dyslexics, but because they link at least part of these differences to the cerebellum. Furthermore, as Nicolson et al. point out, the two differences found support skills relevant to reading; the execution and perfection of an acquired skill. Further support for this connection was produced by Brookes and Stirling (2005) who found a significant correlation between cerebellar soft-signs and reading as well as cerebellar soft-signs and cognitive skills thought to underlie the reading process (i.e., knowledge of common sequences such as days of the week, and picture sequencing).

5.6 Integrating four causal theories

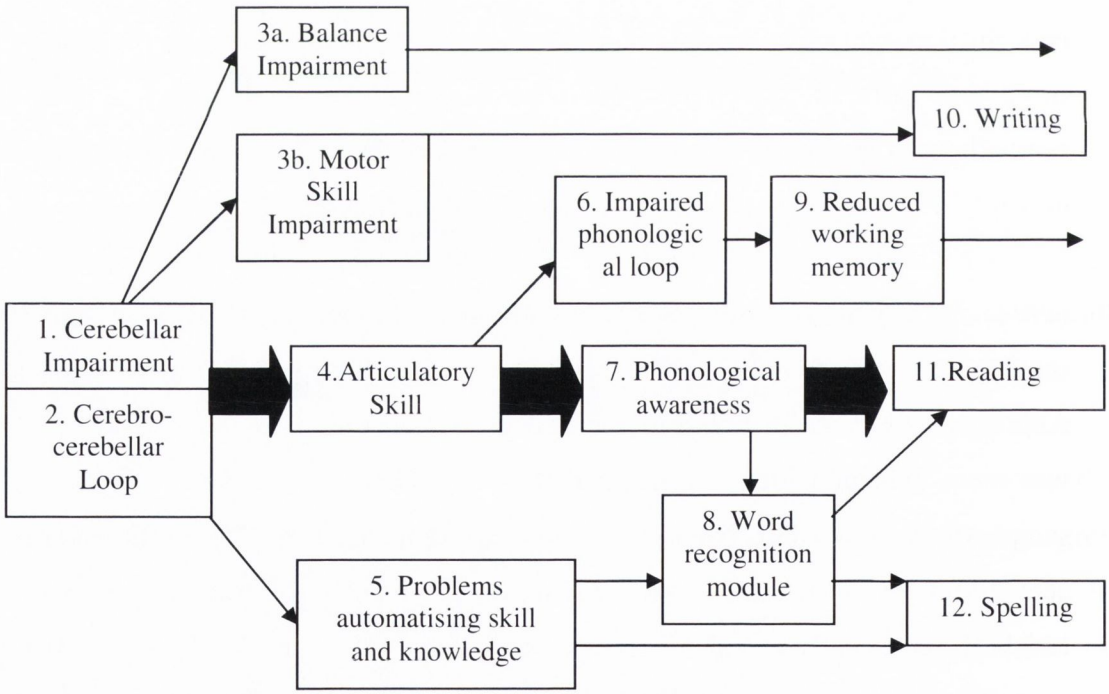

Figure 8: Fawcett and Nicolson's hypothetical causal chain (reproduced from Fawcett and Nicolson, 2001, p.99).

The cerebellar deficit theory is a satisfactory theory for dyslexia because it accounts for such a wide range of symptoms. Being biological in its causal explanation, it subsumes

the cognitive / behavioural phonological deficit hypothesis, and allows for the co-existence of the temporal processing hypothesis and, thus, the magnocellular deficit hypothesis. Figure 8 illustrates how the hypothesis represents a "causal chain" model with three distinct impaired causal pathways resulting from the underlying cerebellar problem (boxes 1 and 2). These pathways include general motor skills (boxes 3a and 3b), speech articulation (box 4) and automaticity (box 5). These allow for a genetically determined, neurologically based, developmental disorder, which manifests in a range of ways, the most obvious of which in today's academically oriented culture, are the educational skills of writing (box 10), reading (box 11) and spelling (box 12).

The difficulties in acquiring and automatising at an early age, the precisely timed muscular movements involved in articulatory and auditory skills, explain not only the common early signs of abnormal development in dyslexic children, such as motor and speech delay, but the later phonological and literacy skills as well. Because of the large attentional load (i.e., conscious compensation) required for the acquisition of each skill, there is little processing of sensory feedback to improve that skill. Thus, the child is at a disadvantage when commencing a subsequent task. The direct result of poorly-timed muscular movements, include poor co-ordination and clumsiness with everyday tasks, such as tying a shoe-lace, learning to ride a bike and more notably, illegible / untidy hand-writing. More importantly from this perspective are the indirect literacy problems, caused by a lower sensitivity to the phonetic structure of language which, combined with the increased need for conscious involvement, result in, firstly, weak acquisition of the basic components of reading (i.e., letter identification, grapheme-phoneme correspondence) and secondly, the poor transition of these to advanced skills (i.e., the blending of phonemes into syllables and words) (box 7). The initial acquisition of these skills is hard enough and yet stopping there is not sufficient; the skills must also be made automatic, improving consistently throughout childhood. Too much attention on any of the components of reading will reduce the quality and comprehension of reading. These two responsibilities of the cerebellum (i.e., learning and perfecting) are what Fawcett et al. (2001b, p.122) refer to as the "double whammy" when it comes to reading because the skill relies on both so heavily.

The production of letter shapes for writing is also a result of weak fine motor coordination and, thus, in need of conscious compensation. Spelling represents a combination of intact phonological skills (box 7), the automatic skill of fine motor timing / coordination for making letter shapes and also the automatisation of

knowledge (box 5). Thus, this may be even more problematic for the dyslexic and is often the most telling and persistent symptom in the older dyslexic (Stevenson, 1987; Thomson, 1984). Thus, the cerebellar deficit theory nicely absorbs cognitively descriptive models of both phonological and automaticity deficiency. In addition to this it also purports to account for the poor working memory often seen in dyslexics' WISC / WAIS profiles and rapid naming difficulties which are consistent with other hypotheses such as the double deficit hypothesis (Wolf and Bowers, 1999). Slow articulatory speed results in an impaired phonological loop (box 6) and, thus, reduced working memory (box 8). These serve to exacerbate the difficulty involved in literacy attainment because a poor working memory is an important skill in holding sounds of letters long enough to synthesise them into a spoken (read) word or building up letters into a written spelling.

Nicolson et al. (2001) suggest that Stein's theory of magnocellular processing deficiency (i.e., temporal processing deficiency) may act either instead of, or in parallel with, the current theory. As Bishop (2002) points out, the two actually work well together given the close neuro-anatomical links from the cerebellum to the magnocellular pathways and in fact there are magnocells in the cerebellum as well as the motor output systems. Furthermore, given the finding by Wolff et al. (1990) that a sub-sample of dyslexics performed significantly worse than controls on cerebellar tests of time-estimation, it is plausible that the important timing control of speech and language processes is interrupted which may contribute to reading disorders in the way that Nicolson and Fawcett describe.

Given the possible role in timing, the inhibitory nature of the cerebellum and the close links between the cerebellum and vestibular system, there does seem to be some kind of justification for the existence of retained reflexes, and thus, disturbed balance skills. Goddard-Blythe (2000) even suggests that some children with dyslexia (usually with a retained spinal galant reflex) will show a higher incidence of bed-wetting than other children their age (over the age of 5). This could be explained by the link between the medial vestibular nerve and the autonomic nervous system, which also would explain abnormal fight or flight responses, such as those experienced by the child with a retained Moro reflex (see chapter 3). The vestibular system and its connections are illustrated in Figure 9.

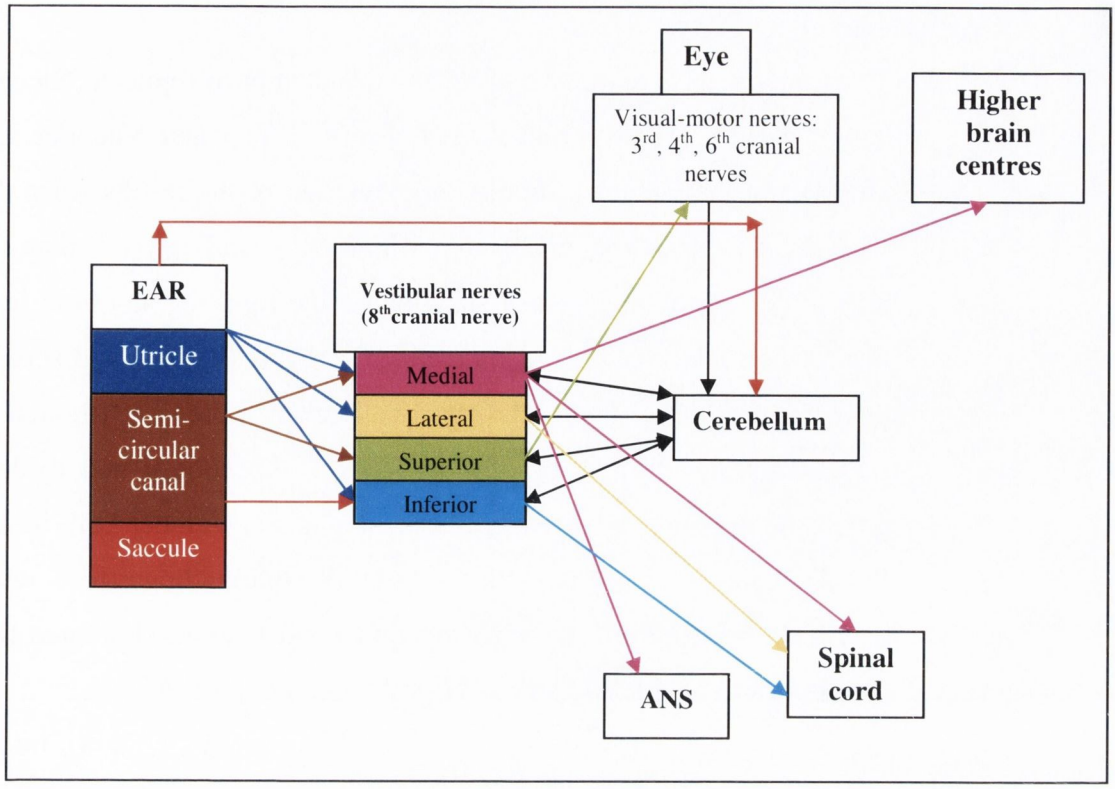

Figure 9: The vestibular system[26].

5.7 Problems with the Cerebellar Deficit Hypothesis

Empirical Problems

The theory espoused by Nicolson et al. (2001) is that cerebellar deficiency impedes articulatory and motor skill development, resulting in motor and language difficulties which manifest as delays in babbling, talking, crawling and walking and eventually problems in learning to read, write and spell (i.e., dyslexia). The theory rests upon a number of premises for which the Sheffield group claim to have empirically and theoretically accounted:

(1) There exists a cerebellar impairment in dyslexics

(2) This cerebellar impairment adequately explains dyslexia

In order for the second to be considered, the first must be upheld. Only once this is done is there any point in examining the second.

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[26] Derived from Goddard (1996, p. 53) and Kohen-Raz (1996, p. 29)

- With regard to the first premise:
    - There exists, to date, only one successful attempt to replicate Nicolson and Fawcett's (1990) original findings (i.e., Yap and Van der Leij, 1994a) and they advise cautious interpretation of the results since they only partially replicated Nicolson and Fawcett's findings (i.e., dual-task differences existed only when standing on the right leg, not both legs). Furthermore, the replication sample size was tiny (n = 28) and some of their videoed material was missing. Finally, there were large (though not statistically significant) differences between the control and dyslexic groups on measures of the single-task for Yap and Van der Leij. Savage (2004) expresses concern about the possible confounding effects which may have been introduced when Nicolson and Fawcett calibrated their two groups to avoid this carry-over problem on the second task.
    - Since the partial replication by Yap and Van der Liej (1994a), no one has managed to replicate the Sheffield findings using the same tests. Both Wimmer et al. (1998) and Wimmer et al. (1999) failed to support previous findings and even cast doubt on the importance of the dyslexics' single to dual-task differences found in Yap and Van der Liej's study. The latter study also found that half of the dyslexic children (i.e., the "non-ADHD" half) in their sample, showed evidence of perfect balancing performance, with no reduction in balance between the single- and dual-balance tasks. Such findings are continuing (e.g., Geuze and Kalverboer, 1994; Kronbicher et al, 2002, Van Daal and Van der Leij, 1999). Furthermore, Fawcett and Nicolson's (1999) generalisability study failed to account for the fact that 28% of the control group had been involved in previous studies using identical cerebellar tests. Without controlling for possible practice effects it is difficult to know whether the differences between the two groups are artificially inflated.
    - Although initial supporters of the importance of soft neurological signs implicating motor skill deficit in dyslexic children, Denckla et al. (1985) later attributed such deficits to ADHD, claiming the presence of such signs in dyslexics was due to high co-morbidity of the two conditions. This was also supported more recently by Wimmer et al., (1999), Van

Daal and Van der Leij (1999) Ramus et al. (2003a; 2003b) and Raberger and Wimmer (2003). Even Wolff et al. (1990), in their study of precisely-timed bimanual coordination, found evidence of sub-groups within their dyslexic sample, with only half of their dyslexic sample showing evidence of this difficulty (and of this sub-sample the results did not correlate with reading impairment). As they point out, this represents some non-linguistic, non-cognitive, non-neuropsychological deficit, but what exactly does it mean? Furthermore, O'Hare and Khalid (2002) found that although tests of neurological functioning (i.e., the Quick Neurological Screening Test), particularly cerebellar-based tests, were useful in differentiating between children with developmental coordination disorder and normally developing children, they were not particularly useful in identifying the subgroup of reading-disordered children within the DCD sample. Thus, the identification of a sub-sample within a dyslexic sample, as described here, is a common finding in research.

- Even accepting that motor skills are impaired in dyslexics, there has been no replication of other cerebellar test deficiencies in dyslexics, for example, time-estimation. Both Stringer and Stanovich (1998) (in Ramus et al., 2003b) and Ramus et al. (2003b) have failed to discriminate between dyslexic and non-dyslexic groups on the basis of time-estimation tasks[27]. This would lead one to conclude that although motor-based tests may be indicative of cerebellar damage, the fact that they are intricately linked to other parts of the brain suggests that they could be equally indicative of damage else-where. This is supported by the apparent consensus since the studies of Fawcett et al., that such impairments may be more indicative of ADHD / DCD than dyslexia.
- Zeffiro and Eden (2001) point out that dyslexics do not mimic the ataxia apparent in those who have sustained cerebellar trauma later in life. However, direct comparisons to acquired injuries, even at the same site,

[27] The fact that there are differences between German and English speaking dyslexics should not be overlooked (i.e., German is far more phonological than is English). In addition, different criteria for these dyslexic groups was employed. Ramus et al. (2003b) used a cut-off IQ of 80 points where-as Nicolson and Fawcett (1999) used a cut-off IQ of 90 points.

may not be meaningful. Within a developmental context, the brain can adapt its wiring in order to compensate for functional losses (Nicolson, Fawcett and Dean, 2001).

- With regard to the second premise:
    - As Ramus et al. (2003b) point out, if there is a causative link between cerebellar impairment, as measured by the Fawcett et al. type tasks (e.g., Fawcett et al., 1996), and phonological skills, then a correlation between the two should hold not just between factors but within groups as well. That is, it would be expected that quality of performance on phonological tasks would match quality of performance on motor tasks. However, no within group correlation data were presented. The same point holds regarding articulatory skill.
    - Furthermore, it must be adequately explained how cerebellar impairment results in dyslexia. Nicolson and Fawcett must be commended for their attempt to do this using the Liberman et al. (1963) motor theory of speech perception, however, Ramus et al. (2003b) have rejected the model on the basis of the initial theory's weak integrity. They claim the theory's basic assumption with which dyslexics' literacy difficulties are explained appears to have been fundamentally flawed because it assumed that internal representation of speech was dependent upon explicit speech articulation. However, as we have seen, internal representation of speech may be accounted for by a biological module linking speech perception and production (Liberman and Mattingly, 1985).

It is an exciting idea that cerebellar processes may underlie reading skill as it suggests that those whose reading impairment is a result of "neurological dyslexia" may be identified on the basis of cerebellar impairment, rather than, as is currently the case, on a reading / IQ discrepancy, the concern over which has previously been expressed. Although the likes of Ramus et al. (2003b) did find pure dyslexics with motor difficulties and do not deny that dyslexics may have trouble with motor control, the literature does seem to support the claims of Ramus et al. that co-morbid cases are (1) more likely to have motor difficulties and (2) more likely to have severer motor difficulties. To echo Bishop (2002), further replication studies of cerebellar findings are

needed which employ properly controlled designs to account for the confusion caused by co-morbid difficulties. A non-specific marker of neuro-developmental problems, whilst useful as an early indicator of potential problems, does nothing to offer relief from the current diagnostic problems surrounding dyslexia specifically. Furthermore, the level of inconsistency in the literature makes interpretation difficult and practical application unjustifiable at present. Thus, further clarification is one of the aims of the current work.

In summary, the cerebellum is traditionally thought of as responsible for the control of learning and motor coordination. On one level it is linked to the very basic aspects of movement (i.e., posture and muscle-tone); it also coordinates head and eye movements. In addition, it allows for the development of skilled movements beyond these levels. More recent research has linked the cerebellum to skills in the cognitive domain, though whether these are truly cognitive or have a motor element in their processing is still a matter of debate. Motor behaviour may be linked to the cerebellum because motor acts are the most overt signs of learning, in terms of acquiring, proceduralising and perfecting skills; more overt than cognitive processes. The inhibition of unnecessary stimuli and response performance possibly allows for the cerebellum's engagement in time keeping and between them these functions ensure that only relevant responses are performed and that these are done so in a smooth and coordinated manner.

6 Chapter 6: Aims

6.1 Rationale for current study

Assuming that the cerebellum is impaired in dyslexics, and that the claims of subsequent treatment efficacy studies (i.e., McPhillips et al. (2000) and Reynolds et al. (2003)) are true, this would suggest that not only is it possible to identify and treat dyslexia using cerebellar theory but, because the treatment is targeting the primary dysfunction, that such children, if treated at a younger age, should no longer show signs of being at risk of dyslexia.

Schiffman and Clemmens (1966) estimated that of those children in whom a learning disability is diagnosed at age 8, 82% can be brought up to their grade-level within a 2 year period. However, this percentage steadily drops and by age 14, just 6% can achieve such gains (cited in Goldberg and Schiffman, 1972). Thus, early intervention is crucial in terms of temporal and financial resources. A review of the literature conducted by Foorman and Torgesen (2001) provides more recent support for the efficacy of appropriate teaching for those at risk of reading failure. The instructional procedures deemed to be most useful incorporate phonemic decoding, fluency in word recognition and text processing, construction of meaning, vocabulary, spelling and writing. Together, they claim that these will bring the majority of children up to their current grade reading level. A meta-analysis conducted by Shonkoff and Hauser-Cram (1987) suggested that treatment programmes are most beneficial if administered to the child under the age of 3 years old. Using such a sample was not possible in the present study given the concentration abilities required to complete the testing procedure, however, by recruiting pre-school and early-school children we targeted participants as young as was possible. The study by McPhillips et al. (2000) showed that replicating primitive (retained) reflexes in much older children (age 8-11 years) had a positive effect on both reducing retained reflexes and improving reading scores. The current study wished to investigate whether a similar (or stronger) effect, could be obtained with a much younger sample.

Added to this is suggestive evidence that those with learning disorders may suffer from elevated levels of anxiety or depression as a result of their disorder. Social and emotional difficulties caused either by the biological disorder responsible for their

academic difficulties or by the added burden put on the individual by their dyslexia, or indeed both (Bruck, 1986) can make life more stressful for the dyslexic. Bryan et al. (1983) and Swanson and Howell (1996) both showed increased test-anxiety amongst students with learning disorders, suggesting an exam-performance double-handicap (Wachelka and Katz, 1999). Ryan (1991) suggests that, as a result of their academic difficulties, dyslexics may have negative thoughts about themselves, may view the present world negatively, and as such, may foresee a life of continuing failure. Heath and Ross (2000) also found increased levels of depression in learning-disordered students. The "negative Matthew effect" is used to describe this downward spiral of achievement deficits and negative motivational spin-offs seen in dyslexics (Stanovich, 1986).

Given the apparent efficacy of the treatments used in both McPhillips et al.'s (2000) and Reynolds et al.'s (2003) studies, it would seem that adoption of an exercise-based remedy for children with dyslexia (or at risk for it), would not only save time and money on the basis of rate of improvement, but would also improve the quality of life and self-esteem of such individuals by targeting them at an early age. Before justifying the adoption and integration of their techniques in the national curriculum, however, the following issues need to be raised:

Replication The level of inconsistency and criticism in the literature warrants concern. There is clearly substance to the argument that dyslexic children show some motor impairment but the extent and severity of this should be clear enough to allow for replication. A recent study by White et al. (2006) demonstrates these problems. Despite their suggestions that reading difficulties in children and sensorimotor difficulties (as measured by auditory, visual and motor tests) are dissociable Bishop (2006) has refuted their claims on the basis of differing findings across authors thereby rendering their conclusions incoherent. It is difficult therefore to come to any firm conclusion about the role of the cerebellum in reading, or the role of motor theory in reading when such different views and findings are constantly being reported.

Generalisability Frequent self-criticism of Nicolson and Fawcett's work concerns the small size of the samples employed in addition to which there may be a certain degree of sample saturation / exhaustion, given the on-going and frequent use of the same sample for testing. Thus, Fawcett and Nicolson (1999) replicated their findings on both

a larger and a novel sample, demonstrating the generalisability of their findings. Concern regarding the inclusion of previously-used control children has already been expressed with regard to causing inflated differences between the treatment and control groups (see Beaton, 2002). The current study attempts to address this by exploring the differences in cerebellar response with a similarly large sample, albeit a younger one.

As causal theory for *dyslexia*

Two Issues arise here:

(1) Tests of cerebellar functioning by Nicolson and Fawcett have thus far only really found cerebellar deficiency in *pure* dyslexics. Other studies suggest such findings are due to co-morbidity of ADHD / DCD.

(2) Tests of Time Estimation, perhaps one of the most pure tests for cerebellar dysfunction (as distinct from motor-based tests which may target other parts of the brain), fail to differentiate dyslexics from non-dyslexics in all but the studies of Nicolson and Fawcett.

Treatment efficacy and test prediction

Both McPhillips et al. (2000) and Reynolds et al. (2003) conclude by suggesting that larger studies are needed in order to replicate their findings. That is a major goal for the current study.

6.2 Aims

The current study was conducted as an objective attempt to test the validity of motor theory as an explanation for the origins of developmental dyslexia and to evaluate the efficiency of motor-theory based interventions in remediating the condition.

Thus, the following were the principal aims of the current study:

6.2.1 Treatment Efficacy

Aim 1

To investigate whether a programme designed to integrate retained reflexes into higher order movements reduces the subsequent probability of risk for dyslexia, as measured by the DST / DEST[28].

Aim 2

To investigate whether stimulating the vestibular system through a programme of gross motor co-ordination reduces the subsequent probability of risk for dyslexia, as measured by the DST / DEST.

6.2.2 Predictive Efficacy

Aim 3

To investigate whether retained reflexes can discriminate between dyslexic and non-dyslexic children by predicting which children of a pre- or early-school sample will subsequently score "at risk" for dyslexia on the basis of the DST / DEST.

Aim 4

To investigate whether vestibular functioning, as measured by a battery of gross motor co-ordination tests, can discriminate between dyslexic and non-dyslexic children by predicting which children of a pre- or early-school sample will subsequently score at risk for dyslexia on the basis of the DST / DEST.

Aim 5

To investigate whether fine motor co-ordination tests can discriminate between dyslexic and non-dyslexic children by predicting which children of a pre- or early-school sample will subsequently score at risk for dyslexia on the basis of the DST / DEST.

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[28] Dyslexia Screening Test / Dyslexia Early Screening Test (Fawcett and Nicolson, 1996; Nicolson and Fawcett, 2004).

Aim 6

To investigate whether visuo-motor skills can discriminate between dyslexic and non-dyslexic children by predicting which children of a pre- or early-school sample will subsequently score at risk for dyslexia on the basis of the DST / DEST.

Aim 7

To investigate whether phonological skills can discriminate between dyslexic and non-dyslexic children by predicting which children of a pre- or early-school sample will subsequently score at risk for dyslexia on the basis of the DEST / DST.

Causal theory

Aim 8

To investigate whether tests of phonological skills are related to performance on cerebellar tests in order to test Liberman and Mattingly's (1985) motor theory of speech as incorporated in to Nicolson and Fawcett's (1990) cerebellar deficit hypothesis.

The study design is illustrated in Figure 10. This is broken up in to the independent and dependent variables. There are four independent variables; the scores of the reflex replication group, the balance group, the placebo-control group and the control group. There are two types of dependent variables; the "major" dependent variable is the score of that test which we used to judge risk of dyslexia (i.e., the DST / DEST). The "minor" dependent variables include the performance on those five sets of tests (and their subtests) which made up our testing sessions. These include the tests of retained reflexes (of which there were 6 subtests), tests of balance (of which there were 8 subtests), tests of visual-motor functioning (of which there were 4 subtests), fine motor tests (of which there were 5 subtests) and tests of phonological skill (of which there were 3 subtests). These "minor" dependent variables were also used as dependent variables at testing time 2 and testing time 3 and also as predictor variables in the evaluation of early signs for dyslexia (at testing time 1). The retained reflexes were used to determine the initial risk for developing dyslexia at time 1. There was one

uncontrolled variable and that was the Conners' Parent Rating Scale – Revised for ADHD screening. This was administered to the parent of the child at testing time 3.

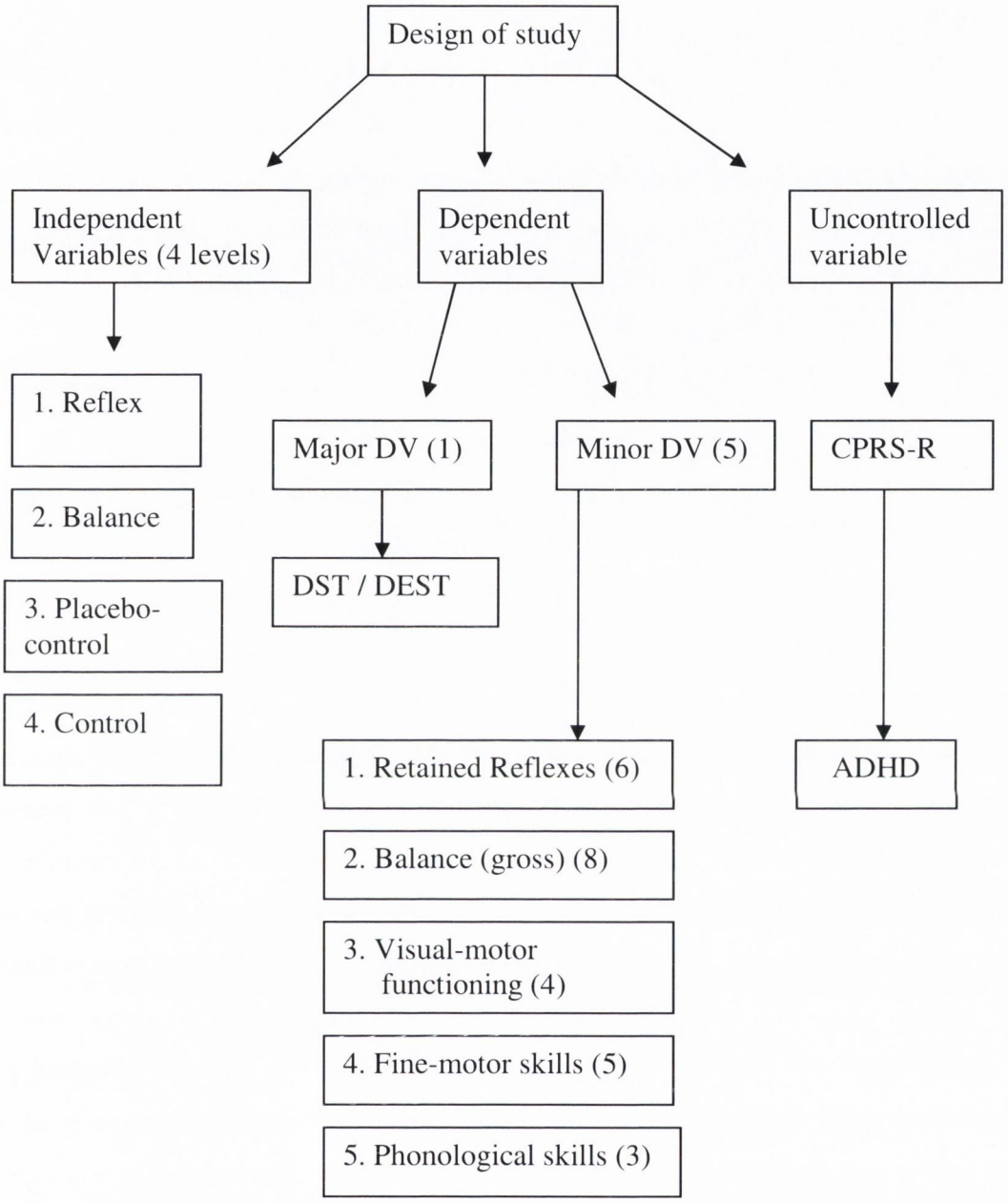

Figure 10: Design of the study

7 Chapter 7: Tests and Treatments

Tests

Chapter 4 explored the development of motor-based practices surrounding dyslexia and neuro-developmental problems, used in previous decades. Chapter 5 introduced a newer theory of dyslexia which, apart from explaining the basis of the specific learning difficulty itself, might explain and justify these motor practices. Chapter 6 now attempts to marry these previous chapters by reviewing motor tests and treatments, based on cerebellar theory.

7.1 Minor predictor variable and predictor variable

Morrison (1985) points out that learning-disordered children often show three interrelated variations in development compared to normally developed children; these include, retention of primitive reflexes, inadequate / non-automatised postural and equilibrium reactions and unstable visual-motor control during motion. All of these have been covered at some level in the current study. As pointed out by Fawcett et al. (2001), standardised motor batteries do not cover the range of cerebellar abnormalities that such studies as this are keen to employ, and for this reason it was necessary to compile a range of suitable tests. The current study includes tests of equilibrium and reflex, as these were the main focus of those studies on which the current one is based (i.e., McPhillips et al. (2000); Reynolds et al. (2004)). Furthermore, Kohen-Raz (1996) suggests that, in order to detect those learning-disordered students with motor impairments, the tests with the most predictive utility are those of primitive postural reflexes, static balance and walking performance. A study by Iverson et al. (2005) found that tests of manual dexterity and balance could discriminate between poor and good readers at age 11, but that tests of ball-catching could not.

In the original battery of tests measuring cerebellar functioning, designed by Dow and Moruzzi (1958), there were 14 separate items belonging to one of three constructs; "maintenance of posture", "hypotonia[29]" and "complex movements" (e.g., finger to finger movements). A recent study by Brookes and Stirling (2005) found significant correlations between the first and last of these with reading and with

[29] Hypotonia: The state of muscular relaxation.

cognitive skills underlying the reading process (e.g., picture arrangement), however, they could not find evidence of strong correlations between "hypotonia" and such skills. The current study did not employ any hypotonia tests, but focussed on those belonging to the other two categories.

Other tests included in the current study's battery were fine-motor tests (e.g., traditional "cerebellar" tests), visual-motor tests and phonological tests. The principal reason for adding phonological tests to the compiled battery was that they act as a good predictor of future reading problems (especially in families with a history of dyslexia) (Muter and Snowling, 1998). Thus, it may be possible to compare phonological performance with various aspects of motor performance.

The attraction of these tests is that they are non-invasive in style and can be fun for the child to do. They can be carried out in an educational setting and, if refined, may take less time to administer than the typical battery of cognitive tests currently required in order to establish the existence of a learning disability.

Thus, the current study employed the following set of tests:

- Balance (Gross-motor) tests
- Reflex tests
- Visual-motor tests
- Fine-motor tests
- Phonological tests

7.1.1 Balance tests

Morrison (1985) stresses that it is not just the inability to inhibit reflexes, but also the retarded development of postural and equilibrium reactions that characterise those learning-disordered children who have neuro-behavioural dysfunction (p. 2).

Rudel (1985) found that dyslexics performed significantly worse on most measures of motor skill, but only up until the age of 11 years; beyond that, as has been demonstrated by Fawcett and Nicolson (1992), dual-task methodology must be employed in order to tease out differences between dyslexics and non-dyslexics. All children in the current study were well under the age of 11, so complicating tests in this way was not necessary.

Kohen-Raz (1996) suggests that for children below the age of five years, using simple "pass / fail" criteria for standardisation purposes is not very reliable; because of this, the current study quantified the quality of performance not on "pass / fail" but on degrees of steadiness according to that scoring-method devised by McPhillips[30] (see Appendix 1, tests 9-14).

Balance is the ability to maintain equilibrium (Crutchfield and Barnes, 1993) and, in this study, two forms of it are tested; static and dynamic balance. Together these test both the vestibular and the proprioceptive systems, and by asking the child to close his eyes on some tests, the contribution of the visual system is also tested. Thus, all of the neuro-physiological systems underlying balance are represented in the following tests.

1. Standing on one leg

By the time he is 4 years old the child should be able to control his balance on one leg, eyes closed; this postural ability indicates that vestibular, proprioceptive and visual information are integrating adequately in the cerebellum (deQuiros and Schrager, 1978). It also represents the later myelination of the ponto-cerebellar tracts around this age, which are linked to the frontal lobes and suggests the child may be ready for learning cognitive-based skills. Perhaps this is why it is felt that children are ready to enter the school system from this age onwards. The Peabody Developmental Motor Scales, second edition (PDMS-2; Folio and Fewell, 2000) lists that by the age of 3.5 years, a child should be able to stand on one foot for 5 seconds and by 4.5 years, for 6 seconds. By 5 years, this has progressed to 10 seconds. McGlown (1990) also suggests a 3-4 year old can remain stable for 6 seconds. Holt (1991) reports that a 4 year old stand for 4-8 seconds and a 5 year old over 8 seconds. This test is also one of the two motor tests that Haslum (1989) reported as correlating most highly with the dyslexic sample in the British National Cohort Study (see Nicolson and Fawcett, 1994).

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[30] "Primary Movement for the Early Years"; a training course run by Martin McPhillips, Dublin, December, 2002.

2. Mann test (Advanced Romberg)

The Mann test is a typical clinical test of balance. Use of the test with eyes closed, in order to account for removal of visual feed-back in guiding balance / behaviour and testing proprioceptive and vestibular stimuli only, is tested in addition to a test with eyes open, to involve the visual system. Kohen-Raz (1996) claims that children can do the normal Romberg (i.e., standing feet together) when 4 years old and with the eyes closed by the time they are 6, but for this version of the Romberg he found that 50% of 5 year-olds and 75% of 6 year-olds could stand, eyes open, for 15 seconds. Of the 6 year-olds, 25% could stand for 10 seconds eyes closed (Kohen-Raz, 1996, p. 212)

3. Tandem walk

This test was used by Frank and Levinson (1973) to identify those dyslexic children they labelled as "cerebellar-vestibular impaired". Ability to walk with feet in the tandem position is one of the capacities checked on the ataxia rating scale (ICARS) devised by Trouillas et al. (1997). McGlown (1990) suggests a 3-4 year old can do this. According to Holt (1991) this ability emerges in the 4th year and can be performed by 90% of 5 year olds. Walking backwards, as on this test, is the second motor test reported by Haslum (1989) to correlate most highly with presence of dyslexia (in Nicolson and Fawcett, 1994). According to the PDMS-2 (Folio and Fewell, 2000) a 3.5 year-old child can walk forward a distance of 4 feet on a 4 inch-wide beam, and a 4.5 year-old can walk, heel-to-toe forwards and backwards for a distance of 5 feet.

4. Walking on tip toe

Holt (1991) reports that a child of 2.5 years should be able to do this for 3 steps forwards and by the time they are 3 years old they should not step off a line which is 1 inch wide and 10 feet long. The PDMS-2 suggests that a child of 2.5 years will walk 5 steps on a 4-inch wide beam and at 3 years they will walk 8 feet.

5. Slalom walk

At 3.5 years a child should be walking forwards on a line for 8 feet and backwards 5 steps (PDMS-2; Folio and Fewell, 2000). The movement requires the child to walk along a single line marking on the ground (Appendix 1, test 5), alternately lifting each leg and crossing it over the line.

6. Hopping on the spot

This skill should have been acquired in children around 3-4 years of age (Parker and Larkin, 2004). By the time the child is 3.5 years he should be able to hop for 3 steps; by the time he is 4.5 years he should be able to hop 7-9 times and by 5 years old, he should be able to hop 10 consecutive times (Gabbard, 2000; Wellman, 1937). Holt (1991) says a 4 year old can hop 5 times and a 5 year old can hop 10 times.

7. & 8. Marching / Crawling

Homolateral Movements: The retention of reflexes could create a barrier to performing homolateral movements in a smooth manner. Bobath and Bobath (1975) and Portwood (2003) point out that some children fail to crawl at all during infancy, preferring instead to shuffle about on their bottoms. This may be due to the fact that normal crawling is impossible or difficult. By asking the children to engage in tasks requiring cross-lateral movement for smooth efficient movement, it was hoped that those for whom it was difficult could be identified. A mature walking pattern should be seen in a normally developing 4-5 year old child (Gabbard, 2000) and the failure to swing arms in opposition with leg action is one of the common walking problems listed by Larkin and Hoare (1991).

Crawling should be difficult for a child with a retained STNR. Movement of the head will, as mentioned, result in contracting the muscles in the direction to which it is moved, and as such, the child's head should either lower to the floor, or the bottom should lower to the heels. In either case crawling may be clumsy. Lateral movement of the head would stimulate an ATNR if it is retained. The PDMS-2 states that an infant of 5-11 months will creep on hands and knees with opposite arms and legs moving together; however, this should be outgrown fairly quickly.

7.1.2 Reflex tests

Dysfunctional Symptoms:

The method of reflex elicitation, as described here, is similar regardless of the author; Capute et al. (1978), Fiorentino (1980), Goddard (1996) and McPhillips et al. (2000) all describe the same processes. The assessment of neo-natal infants was once dependant upon the subjective assessment of muscle tone, however, with time this developed into a more objective method of assessing the presence or absence of primitive reflexes, though without a system for measuring improvement over time (Capute et al., 1978). Capute et al. were among the first to anchor degree of response to the development of a graded reflex scale.

Regardless of the eventual findings of this research, there is no doubt that retention of primitive reflexes generally indicates CNS immaturity or pathology (Kohen-Raz, 1996; Zafeiriou, 2004). Although there are many different primitive reflexes, it would not have been practical to test for them all. The important thing was to look for evidence of a "cluster" of retained reflexes, as single findings are rarely predictive of anything (Crutchfield and Barnes, 1993). In his comprehensive, and clearly-written review of the research literature, Morrison (1985) makes the same point, careful to refer only to those children with *clusters* of signs as suffering from "neurobehavioural dysfunction". Goddard (1996) defines a "cluster" as two or more primitive reflexes each of which elicit 50% or more or the full reflex. There is some disagreement regarding which of the primitive reflexes (either individual or in combinations) are the best predictors of clinical relevance (Zafeirioui, 2004). However, given the attempt here to replicate the acclaimed success of current practices, four particular reflexes were tested in the current study. Of these, three are described by Kohen-Raz as the most prominent. These are the ATNR (Asymmetric Tonic Neck Reflex), STNR (Symmetric Tonic Neck Reflex) and TLR (Tonic Labyrinthine Reflex). In addition to these, the Moro reflex was also included as part of the battery. As previously described, the ATNR, STNR and TLR are all tonic reflexes, whereas the Moro is a phasic reflex. Of the tonic reflexes, the current study makes use of the ATNR and the STNR as proprioceptive reflexes and the TLR as a vestibular (or labyrinthine) reflex (McCormack and Perrin,

1997). All are tested for with eyes open and eyes closed (in order to switch off the influence of the visual system). Goddard-Blythe (2001) claims that of a sample of 54 dyslexics, 100% showed evidence of a retained ATNR and TLR, 72% showed evidence of a retained STNR and 81% evidence of a retained Moro.

Proprioceptive tonic reflexes are those stimulated by movement of the head (up / down or left / right) which affects the tone of the neck muscles, thus, triggering tone redistribution throughout the body. This results in the co-activation of agonist and antagonist muscles (Bobath, 1985). Details of how to test for retained reflexes is given later in the methods section.

1. ATNR

Three measures of the ATNR were taken, as it was not certain whether one test would be better than another in eliciting a retained reflex (if present). These included a measure taken in the supine position, in the quadruped position and standing up position (arms outstretched). In general, the ATNR is triggered by a turning of the head to the left or right, which causes the limbs to extend on the side to which the head is turned, and the contra-lateral limbs to contract. Kohen-Raz (1996) suggests that the quadrupedal position may elicit the reflex in more covert form, which supports Morrison's (1982) findings that this position discriminated between learning-disordered and normal children, where the standing position did not (in Morrison, 1985). Outstretched arms may be subject to the confounding effects of fatigue, and for this reason, the child was given a rest between each 2 sets of head rotation (i.e., eyes open and closed). Given our young sample, it was considered wise to include all three measures.

2. STNR

One measure of the STNR was taken of the child on all fours. This is stimulated by movement of the head vertically. As the head moves upwards it causes the lower limbs to contract and the upper ones to extend. This pattern in reversed as the head is lowered towards the chest. A child with a retained STNR, when raising the head in the table position, will demonstrate a bend in the lower half of the body, such that the bottom and heels will meet, possibly resulting in a "W" sitting position. This is due to the tightening ham-strings, stimulated by the head movement. A

movement of the head downwards will result in the upper part of the body contracting, such that the head and floor may meet (McGlown, 1990).

Vestibular tonic reflexes also redistribute muscle tone, but this is in response to gravity as well as head or body movement (McCormack and Perrin, 1997). Movement activates the labyrinthine system of the inner ear (i.e., saccule and utricle) which in turn ensures the activation of appropriate muscles.

3. TLR

This occurs in response to movement of the head below or above the level of the spine (Goddard, 1996). When the head moves above the level of the spine, it causes the body to extend all over (arms and legs will straighten). The opposite movement will cause the body to flex.

4. The Moro

This is also a vestibular reflex, albeit a phasic one. This is triggered in response to a sudden change in the environment through any of the senses (visual, auditory, tactile or vestibular). The arms will spread up and out, in a symmetrical manner, away from the body and accompanied by a sudden intake of breath, before slowly returning back towards the body.

7.1.3 Visual-motor tests

Fixation, accommodation and convergence should be established in children by the end of the third month (deQuiros and Schrager, 1978). The human baby can fixate upon a target and follow it from the mid-line to 90 degrees by the time it is 2 months old. A month later, it can follow a target 180 degrees across the mid-line of the body. Vision gradually improves and by the time the child is 3 years old the accommodation reflex is developed (though it can still regress at this time) and a year later binocular vision is well developed (Holle, 1976).

Any sensory disorder involving input from the retina or the joint and pressure receptors of the neck will, according to Ayres, upset visual perception (and thus, reading). Visual tests were used by Frank and Levinson (1973) to identify those

dyslexic children they labelled as "cerebellar-vestibular impaired" and as such were included here to give a fuller picture of the child's vestibular functioning.

The maintenance of posture is achieved through the three major physiological balance mechanisms previously discussed (the vestibular system, the proprioceptive system and the visual system). As we have seen, the vestibular system relies on the semi-circular canals and the otoliths organs of the ear, the proprioceptive system relies on sensations received from the joint and pressure receptors of the neck and skin and the visual system relies on the retina of the eye and the visual-motor system. These systems integrate to perform their ultimate functions and do not generally operate in isolation (Howe and Oldham, 1981). For example, the vestibular-ocular reflex is an example of an interaction between the visual and vestibular systems. The nystagmus in one of these reflexes and refers to the involuntary rhythmic eye movement that occurs when a person spins round then suddenly stops – it is the lingering adjustment of the eyes to tracking the world as it revolves around them.

Vestibular Dysfunction

Loss of balance and problems with gaze fixation are not the only symptoms of peripheral or central vestibular lesions; an abnormality of visual perception may also result (Savundra, 1997). Further problems may include motion sickness, visual-motor dysfunction, becoming easily disoriented, having poor directional awareness, poor space perception and poor organisational skills. Children also tend to fear swings and heights (Goddard, 1996). Symptoms reported by sufferers include faintness, giddiness, sensations of swimming or floating, unexpected falls and difficulty thinking in certain environments (Savundra, 1997).

Children with a vestibular-based learning disability will have problems following a moving object in front of their eyes (Ayres, 1979). Goddard-Blythe (2001) claimed that 92% of her dyslexic sample (n = 54) showed such problems. Vestibular reflexes allow for corrective movements to be performed at the appropriate scale and speed. They are triggered on the basis of information from the 8th cranial nerve which leads to 4 vestibular nuclei; the superior, inferior, lateral and medial nuclei. The former receives information from the semicircular canals; the inferior nuclei receive them from the utricle and saccule; the lateral nuclei receive them from the utricle, and the medial

nuclei receive them from the utricle and semi-circular canals. All vestibular nuclei also receive information from the cerebellum (Crutchfield and Barnes, 1993).

The following four tracking tests were tested:

1. Visual tracking
2. Hand-Eye tracking
3. Fixation with eyes
4. Convergence and re-establishment of binocular vision

There are also 4 kinds of vestibular reflexes; the vestibulo-ocular reflex (VOR: horizontal and vertical), nystagmus, visual-ocular reflex and cervico-ocular reflex. The VOR is one of the most important functions of the vestibular system (Crutchfield and Barnes, 1993). The visual-ocular system is tested in the current study by use of 2 tests of visual pursuit; test 1 requires the child to follow a moving object (pencil) with the eyes whilst keeping the head still (the optokinetic reflex); and the test 3 requires the child to move the head whilst focussing upon a stable object (the vestibular-ocular reflex). Evidence of excessive saccadic movement whilst performing these tasks, or an inability to keep the head still or hold gaze upon the pencil suggest that the child may have impaired visual-ocular reflexes. These tests are included on the basis of the vestibular system's close link with the cerebellum, and on the basis of "blurring" being a common complaint of dyslexics when they read. Cermak and Ayres (1984) claim to have found a difference between learning-disordered and normal children on the "Space Visualisation Contralateral Use Test" (SVCU), which measures the child's spontaneous tendency to cross the midline of the body using puzzles. As far back as 1912, Head considered the reluctance to engage in a cross-lateral response (by using the ipsilateral side) as a sign of neuropathology (Head, 1912). These visual tests also test the child's ability to cross the midline of the body by looking at ocular tracking and arm usage across this midline (i.e., test 3). However, Morrison (1985) cautions against over-reliance on the findings for reasons of heterogeneity and poorly-described "learning disorders".

"Visual tracking" is one of the visual-motor tests used by the ICARS (ataxia rating scale) designed by Trouillas et al. (1997). Goddard (1995) suggests that a retained ATNR will cause eye-tracking and pursuit problems because the eyes cannot move independently of the head; if they attempt to cross the mid-line of the body the head or body may "jump" from one side to the other. A retained STNR will have consequences for visual accommodation and binocular convergence (i.e., test 4). All of the visual tests and scoring methods were suggested by Blythe (2001), although the reliability and validity of them is not known.

7.1.4 Fine-motor tests: (Adiadochokinesia x 2 / Rapid alternate movement x 3)

Temporal coordination of antagonistic muscles is necessary to ensure smooth coordination when engaging in motor tasks, particularly fine motor tasks. The result of poor timing will be dysmetria and dysdiadochkinesia, as well as possibly deficient speech production (Ivry and Gopal, 1992). The ponto-cerebellar myelination necessary for smooth pursuit of such movements develops in the fourth year (Yakovlev and Lecours, 1967) and, as such, the tests included here should be suitable for the age group in the current study.

Ivry and Gopal (1992) suggest that fine motor skills may be particularly difficult for a child with poor timing skills. Many of these, as with the balance tests, are taken from Dow and Moruzzi's (1958) test battery for balance and coordination, designed to examine cerebellar function using soft sign indicators. Although this original test had 14 items, not all of these were used due to time and concentration constraints on the part of the child. Despite this, Brookes and Stirling (2005) report that in their study on normal children, reducing this test battery did not reduce the test's validity.

Two tests of adiadochokinesia

1. Finger to nose

This was one of the tests Frank and Levinson (1973) used to identify those dyslexic children they labelled as "cerebellar-vestibular impaired". An inability to smoothly and successively touch the thumb with the fingers of the same hand or touch the tip of the nose with the tip of the finger with eyes closed are generally believed to be

indicative of cerebellar dysfunction. It was shown by Satz (1978) that children who had problems performing these tasks at the expected ages were more likely to show evidence of learning disabilities some years later.

2. Heel on shin

Along with the finger to nose test described above (and drawing of the "Archimedes Spiral"), this test, described by Holmes (1917; 1939) is one of three "cardinal cerebellar signs" (Trouillas et al., 1997).

Three tests of rapid alternate movement

3. Finger tapping

This was one of the tests on which Rudel (1985) found a significant difference between young dyslexic and non-dyslexic children (i.e., under the age of 11 years) and which Satz (1978) used to predict those who were likely to show learning disabilities later on. The ability to perform consecutive finger to thumb movements emerges around the age of 3 years old and improves steadily until the child is about 8 years old (Gabbard, 2000).

4. Palm reversal

Rudel (1985) also found significant differences between her two groups on this test of alternating palm reversals.

5. Finger to thumb

Finger movements, such as successive opposition of fingers and thumbs, has been shown to be deficient in dyslexics (Rudel, 1985; Denckla, 1985). It was one of the tests used in Dow and Moruzzi's (1958) battery and is employed in this study as it was described there. It was shown to differentiate dyslexics from normal readers in Fawcett et al.'s (1996) study and was replicated by Ramus et al. (2003). Seitz et al. (1990) claimed to show unexpected cerebellar activity in dyslexics when they performed this test.

7.1.5 Phonological tests

Articulatory tests were a further aspect of dysfunction found by Frank and Levinson (1973) to be useful in identifying those dyslexic children they labelled as "cerebellar-vestibular impaired". Phonological tests have long been demonstrated to be possible early predictors of dyslexia in children too young for formal testing (Mutter and Snowling, 1998) and deficient speech production is something Ivry and Gopal (1992) suggest might result from poor timing (controlled, they claim, by the cerebellum).

Tallal et al. (1997) conducted a longitudinal study of pre-school language impaired children and showed that although only 17% of them went on to show specific reading difficulties, this was nearly double that of the controls who showed similar problems (9%). As time was a significant factor in Tallal et al.'s study they suggested that perhaps SLI children's deficits become more reading specific over time. This in turn implies there may be a developmental continuum between early SLI and phonological based reading difficulties – resulting from the same processing constraint but with different manifestations at different ages. This interpretation would be consistent with Stevenson's (1987) hypothesis, that an underlying processing problem presents as an SLI in pre- and early- school children, a reading impairment in primary school and a spelling impairment in adolescence. Thus, we see the developmental course of a single deficit. Gallagher et al. (2000) found that those children who demonstrated early language delay in their fourth year were more likely to demonstrate literacy difficulties at age 6 than were controls.

Three tests were included in the phonological battery: letter name knowledge, phoneme deletion and non-word repetition.

1. Letter name knowledge

Letter-name knowledge was tested to inform us of whether the child was familiar with the alphabet and allow us to compare groups at a later stage. Letter naming ability is necessary in order for children to become phonologically aware, thereby gaining reading skills (Blaiklock, 2004), and as such, it was deemed important for indicating whether a child might be at risk of later reading failure. Jorm et al. (1986) have shown that dyslexic readers can be distinguished from normal poor-readers at school entry; the dyslexics' deficiencies were more limited than the poor-readers' who were affected on a wide range of cognitive tasks. For the dyslexics,

however, there were two main predictive factors; "early literacy" which included letter name knowledge and "phonological processing" which included phoneme segmentation (hence use of the phoneme deletion test below). Gallagher et al. (2000) have also shown that letter knowledge at 45 months (i.e., 3 years, 9 months) can predict literacy level at age 6 years. Similar findings have also been shown by Muter and Snowling (1998) who found that letter name knowledge when tested at ages 5 and 6 years, was predictive of reading accuracy at 9 years.

2. Phoneme Deletion

Tests of phonological awareness have been shown to correlate with reading scores, both concurrently and predictively (Blaiklock, 2004). Jorm et al. (1986) also demonstrated the importance of phoneme segmentation for predicting dyslexia versus poor-reading generally (see above). Both phoneme deletion (designed by Muter and Snowling, 1998) and non-word repetition (below) were found by Muter and Snowling (1998) to be the two best long-term predictors of reading accuracy at age 9, when tested at ages 5 and 6.

3. Non-word repetition

Trouillas et al. (1997) also assessed speech when designing their ataxia rating scale, however, this was designed for adults and employed a semi-quantitative evaluation of spoken sentences for dysarthria. A simpler version for children was necessary for the current study and so Gathercole et al.'s (1994) "non-word repetition test" was used which contains a list of 50 nonsense words, between one and four syllables in length. The non-word repetition test has been strongly linked to the important language abilities of vocabulary acquisition, reading and language comprehension of the early school years and involves the sub skills of phonological working memory, phonological analysis and articulatory output processes. It also invokes the use of pre-existing word knowledge such that similar sounding words may help repetition (Gathercole et al., 1994).

7.2 Uncontrolled variable

Conners' Parent Rating Scale – Revised (CPRS-R)

Co-morbidity of dyslexia and ADHD, plus findings by Denckla et al. (1985) necessitated the inclusion of this screening test to control for the confounding effects of ADHD. Furthermore, one of the criticism made by Morrison (1985) in his review of studies of sensory integration efficacy was that, although it is well-known that children with learning disorders very often have behavioural problems, outcome measures for such problems are rarely measured in the way that academic ones are (p.154). He also suggests that the way in which such studies might work is by reducing hyperactivity and improving attention and concentration (i.e., an indirect effect) rather than necessarily changing brain function.

7.3 Independent Variables: Treatments

There are many exercise-based "treatments" marketed as methods for alleviating developmental disorders (e.g., INPP, NDT, Primary Movement, Dores' Achievement Centres, BIRD, Bender Achievement Centres), and some which are marketed for the average school child (e.g., Brain Gym) simply to encourage their capacity to learn. Few have tested their claims empirically and of those that have done, the methodology has left some room for improvement (i.e., McPhillips et al., 2000; Reynolds et al., 2004).

The "Institute for Neuro-Physiological Psychology" (INPP), based in Chester, England, has already been introduced. It was followed by INPP (Ireland), from which evolved Neuro-developmental Therapy (NDT), advocating the same treatments based on the same theory as previously discussed. Simultaneously, "Primary Movement", was set up in Northern Ireland by Martin McPhillips, though he too was originally trained by INPP. Thus, many of the currently commercially available therapies have the same foundation and theoretical route. McGlown split from the original team (i.e., Blythe and McGlown) in 1983 and founded the "BIRD" (Brain Injury Rehabilitation and Development) charity to treat brain injuries and learning disabled children in much the same way; this is now known as "Developmental Reflexive Rehabilitation". The Dores' Achievement Centres have begun appearing in the last several years and also advocate movement therapies. These are not based on the idea of replicating primary reflexes, but on stimulating the vestibular system and ultimately the cerebellum.

Thus, a fundamental aim of the current study is to evaluate these long-established ideas through two experimental treatments, that of vestibular / proprioceptive stimulation (i.e., Balance) and that of reflex / synkinetic inhibition.

Goddard's (1996) idea which suggests that now-developed, higher centres of the brain can consciously compensate for the impaired cerebellum through continued effort, represents a "second chance" for the brain to register the movement patterns which should normally have inhibited these primary reflexes. Because, as Ayres (1979) points out, the cerebellum is essentially an outgrowth of the vestibular and reticular systems, and because the cerebellum is engaged in sensory-integration, movement therapy is particularly beneficial in helping the cerebellum, and consequently improving specific skills in which sensory-integration is important, such as reading and writing. She suggests that in very young children (i.e., under 2 years) sensory integration therapy may help to develop new neural connections, and in the older children it may help to strengthen existing connections.

By using the conscious higher centre to force control over innate reflexes in this way and allowing the cerebellar neural connections to become strengthened (i.e., more heavily myelinised in the way that they originally should have done), this may feed back to higher brain levels and support future attempts at motor control. It may also help the cerebellum's inhibitor function to "kick in" or become stronger, preventing innate / irrelevant pathways from being automatically invoked.

McGlown (1990) reports a study conducted by Klosovskii (1963) who found 23-35% more myelinisation in the vestibular receptors of kittens whom he had forced to engage in a concentrated amount of motor vestibular stimulation on a daily basis for 10-20 days, compared to the vestibular receptors of control kittens. McGlown's neurophysiological theory is similar, stating that the reflex motor responses must be activated repetitively in order to facilitate development of the neural pathways; this must be done in a way which recapitulates normal development so that the brain learns the "feel" of performing a normal action. Repetition is something which is stressed by all advocates of neuro-developmental treatments.

7.3.1 Reflex treatments

Orton was convinced that language dysfunction, like language function, had neurological underpinnings. Delacato is famous for his part in the "Doman and Delacato" treatment methods of the 1950s, which attempted to use motor treatments to "rewire" the brain. As previously discussed, the criticism unleashed as a result of these practices is legendary and they have long now disappeared. In the 1970s Bender, from the Purdue Achievement Centre for Children, designed both tests and treatments based on persistent primitive reflexes (the "Bender-Purdue Reflex Test" was published in 1976). The reflex-exercise used in the current study was based upon the reflex-suppressing ideas of these different groups. Not only is the theory underlying these movements published widely in the literature (as we have seen), and the ideas practiced by movement therapists the world over, but as previously mentioned, the Primary Movement exercises are themselves based upon reflex-replication exercises practiced by INPP in Chester. This is stated publicly by both McPhillips himself (2000, p. 541) and the directors of INPP (Sally Goddard-Blythe and Peter Blythe) (Goddard-Blythe, 2000, p.155).

INPP / NDT:

INPP believe that retained primitive reflexes will not "release" the postural reactions (Blythe, 1992, p.12). According to INPP's spokeswoman, Goddard (1996), reflex inhibition is based upon "replicating specific stages of early development through the repetition of movement patterns based upon early development" (p.81). For example, to combat a retained ATNR, the child is advised to lie on the ground and practise slowly extending the limbs ipsilateral to head-turning, and progress from here to contralateral limb extension, followed by cross-lateral movements with head at the mid-line. These movements reflect the sequential development of normal walking. The similarity between this line of reasoning and that of Doman and Delacato before them is clear. Their approach is one which mixes vestibular stimulation with reflex inhibition in much the same way as deQuiros and Schrager did.

Primary Movement:

McPhillips et al. (2000) suggested movements which were designed to inhibit a retained ATNR reflex first, followed by the Moro reflex second. As these are the first

reflexes seen in prenatal development and some of the first to be inhibited, post-infant suppression should work following the same sequence. They describe part of the movement for inhibiting the ATNR which involves sitting in a chair, eyes closed, extending one arm slowly, followed by turning of the head in that direction. The child then returns the arm to the midline, followed by the head and repeats this process with the contralateral arm. This sounds similar to some of the movements described by Glaeser et al. (1966), when studying the Doman-Delacato technique, where the child was encouraged to sit up and follow the hand out to the left or right in an attempt to improve hand-eye coordination. For the retained Moro reflex, movement of the head, then each arm singly, then both arms together, is suggested. This is described in more detail in the methodology section and can be seen in detail in Appendix 1. The movements used by McPhillips, and Primary Movement in general, are based on movements initially devised by Peter Blythe at INPP, however, they are also influenced by the ideas of Raymond Dart, a South African professor of anatomy, who was interested in skill, poise and the Alexander Techniques (Dart, 1996). The essence of the latter, the Alexander Technique, is learning conscious control of the voluntary musculature, thereby enabling one to not only become aware of body misuse but improve that body-use and eliminate handicaps (see Alexander, 1923; 1932). Inhibiting useless reflexes (particularly those resulting in asymmetry) and integrating them into purposeful movement seems to be the underlying goals of the techniques of both Alexander and Dart. Dart (1934) was struck by the evolutionary development in skill that humans showed, arising from the skill of the simplest species (fish), through to amphibians, mammals and primates, and he considered the stage of fetal development to be the most important stage in human development. This view shows a striking similarity to the theories of Carl Delacato and it comes as no surprise to learn that Dart held a chair at the Institute for the Achievement of Human Potential in Philadelphia, where Doman and Delacato were prescribing "Neurological Rehabilitation" for children with learning difficulties. Dart considered human skill to involve muscular and mental collaboration, and as such, could be harnessed to benefit the educational system as well as every-day life. Despite this, he felt that most people did not progress beyond that level of skill that we are born to automatically achieve (i.e., rolling, turning, sitting up, crawling, standing, climbing, walking, running, leaping). Tireless practice and attention are needed to achieve these skills, but they are genetically programmed into us and once achieved, we are prone to abandon the pursuit of further skill. Mental skill

can come only once man had achieved a vertical stance, and development of this and further physical excellence can only be achieved with the suppression of what is not necessary (the idea of suppressing reflexes here is striking). Skilled performance, Dart claimed, could only occur once the unwanted muscles were perfectly relaxed. By concentrating on the bases of the skill and building from there, the memories of particular muscular sensations are formed so that only these muscles are invoked when the task is performed. Such sensations are achieved by repetitive practice and complete attention to those precise movements. The most important skills, according to Dart, were naturally those separating us from other primates; the movements afforded by erect posture such as dancing and balancing; co-ordinated eye-hand-movements; the skills of language and the skills of music, rhythm and time. His movements seem very much to reflect the human infant's development of muscular control. Studying oneself (i.e., one's imperfections and how to control them) begins by adopting a semi-supine position in which the person must reflect on their physical sensations. Then, in slow-motion for 15-20 minutes a day, over the course of weeks, months or years, the person graduates his positions from semi-supine, to supine, to prone, left / right rocking then rolling from prone to supine and back again. The person then progresses to support himself on the forehead / elbow in the prone position (i.e., the "primary crawling reflex") and gradually lift the chest. This becomes the forehead / elbow / knees position (i.e., the "primary creeping reflex") and so on (there are many more; see Dart, 1996). Some of these movements involve rotation of the head and contemplation of left and right arm movements for approximately 5 minutes each. All the while, the person is obliged to consider their sensations of equilibrium and support in a way that combats our innate fear of falling.

As far back as the 1930s and 1940s an English woman, and acquaintance of Dart's, called Irene Tasker (Dart, 1934), was applying the use of the Alexander technique to seriously handicapped children in South Africa, with the claim of huge success. The idea of such movements as a treatment, therefore, is not a new one. However, despite the professional, academic and scientific backgrounds of all those who have profited from its conception, it had never been subject to scientific scrutiny until the seminal paper of McPhillips et al. in 2000. The evolution of many of the current reflex-inhibiting ideas and their implementation in organisations is outlined in Figure 11 (see also chapter 4).

Peabody Developmental Motor Scales – 2nd edition (PDMS-2); Folio and Fewell, 2000):

Although they do not label the movements in the same way as INPP and Primary Movement, Folio and Fewell (2000) do suggest similar exercises. One is that in which the child is encouraged to extend both arms out in order to grasp something, first with both hands and then with one. The arms should be bent at an angle greater than 90 degrees. In addition, they should be encouraged to move at least one and possibly both hands toward the mid-line of the body, from an extended position, in order to develop eye-hand coordination, especially where direction is concerned. This would help them to cross parts of the body. Finally, in order to learn independent finger use, the child should move his fingers sequentially, with each finger meeting the thumb in turn.

There are many other groups making use of reflex-based therapies. Some of these are listed below (see Appendix 2 for further details):

- Neuro-Developmental Therapy (Ireland): NDT.
- Brain Gym
- The Handle Institute
- The Miriam Bender Achievement Centre
- Jean Ayres's Sensory Integration
- The Bobath Approach
- SMART (Lyelle Palmer)
- BIRD (McGlown)

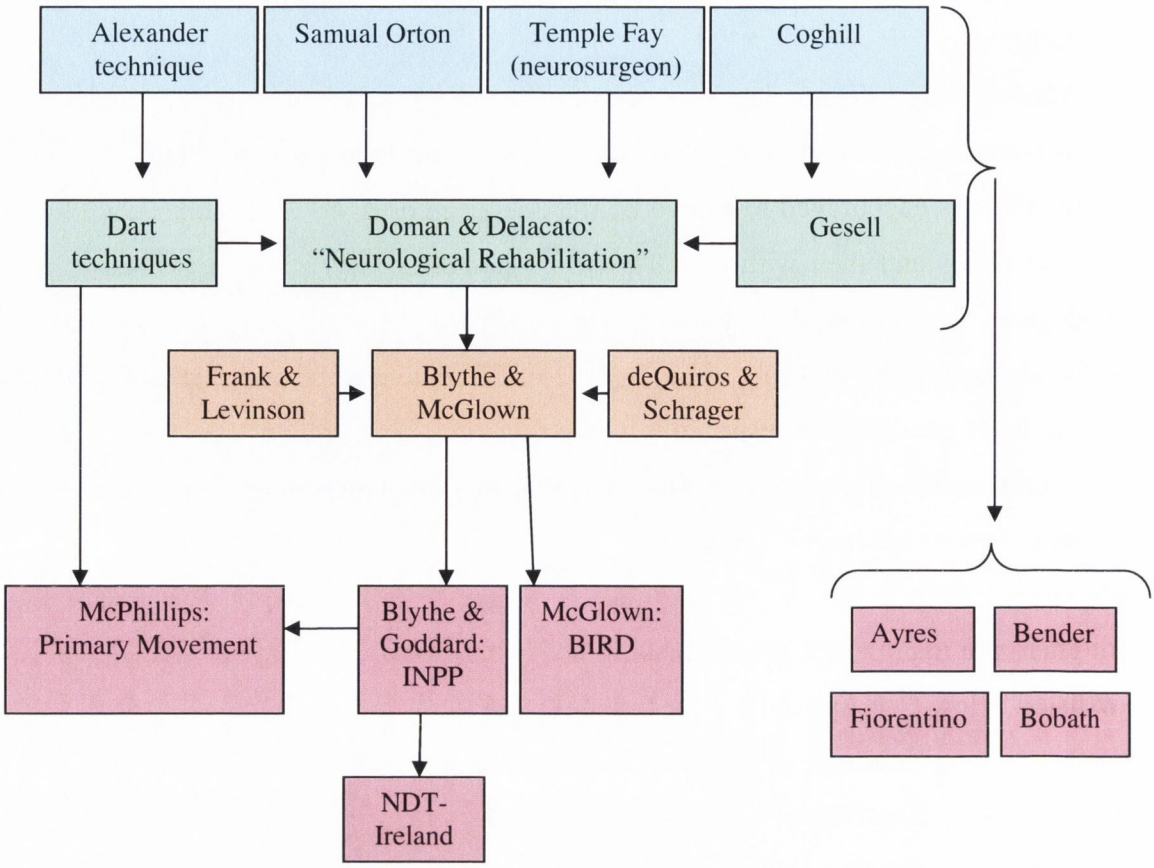

Figure 11: The evolution of current reflex-inhibiting organizations (chapters 4 and 7).

7.3.2 Balance

Kephart (1971) suggests that balance is the mechanism through which we learn laterality. From the need to innervate one side, detect which side must move and how it must move, the child selects the appropriate compensatory movements to maintain balance. In this way he learns to differentiate right from left. Thus, employing a balance programme is important, not just in order to try and replicate previous findings (i.e., Reynolds et al., 2003) but because its effects should promote those developments deemed so important by the early theorists (e.g., Orton, Delacato).

Kephart's training activity suggestions for the learning-disordered child include bilateral movements, unilateral movements and cross-lateral movements. The former of these involves moving the arms and legs at the same time and this has been incorporated into the balance training programme in the current study in the form of jumping jacks (for example). Unilateral movements refer to the movements of one side

of the body followed by the other and this is found in the form of movements such as making a driving motion with the arms (one arm up, then down followed by repetition with the other arm; galloping with one leg leading, then the other). He also suggests cross-lateral movements which are included in the balance programme in the form of marching, running, crawling and walking. Movements across the mid-line of the body are also encouraged, for example, sitting crossed-legged (while bouncing) and swaying (like a tree whilst feet remain static). He also suggests altering normal positions, in order to promote body-image and motor control, by using unusual positions. He recommends the use of "animal" type walks and this too has been used in the current study. The movements of the kangaroo, gorilla, horse, frog, crab, ostrich, eagle and giraffe are all mimicked by the child on this programme. He rationalised that regardless of the precise exercise, what was important was the ability of the child to generalise from one movement to another (p.101). The same rationalisation was used in the current study with regard to designing a balance programme in order to replicate Reynolds et al.'s (2003) study. Although it was impossible to know exactly what kind of movement the DDAT used in treating dyslexia[31], the underlying theory was the targeting of the vestibular system (and, thus, cerebellum). A range of balance-based movements were used in the current study which would, in principle, do the same job.

Kohen-Raz (1996) describes the rationale adopted by most of these programmes; one in which primitive, sub-cortically controlled reflex responses gives way to a pre-programmed sequence of developmental stages, each one necessary for the subsequent one to appear and develop properly. As we have seen here, where this does not happen the skills required for academic performance will be deficient, not allowing for quick and easy learning. His exercises, which were also designed to target the vestibular, proprioceptive and thus, cerebellar systems, included occluded vision and required the child to engage in gross motor tasks such as turning, walking, jumping, stepping (forwards and backwards), throwing, catching and pushing. Those tasks which employed symmetric and asymmetric movements did so in the attempt to stimulate the pyramidal and extra-pyramidal areas, which, in conjunction with the vestibular system and cerebellum, were designed to suppress synkinetic (i.e., reflexive) movements.

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[31] Reynolds et al. (2003) would not comment on the details of their study for "commercially sensitive" reasons.

Simple execution of synergetic movements involved tasks such as the movement of one limb whilst the contralateral limb either moved in the opposite direction or remained still (in tonic suspension). This aimed to suppress the (usually involuntary) synkinetic movements in the opposing limb not engaged in voluntary movement (full details of this programme are available in Kohen-Raz, 1996). No placebo-controlled or subsequent replications of his work have been reported.

Kohen-Raz compiled a list of what he deemed the "general principles" of perceptual-motor programmes. These include some of those already mentioned in his own study, as well as some others:

- Stimulation of body awareness
- Fostering of spatial awareness
- Execution and suppression of synkinetic movement (as described)
- Vestibular stimulation 1: Training static and dynamic balance:
    - Standing on tiptoe / narrow support / heel to toe
    - Standing eyes closed
    - Walking on beams / lines
- Vestibular stimulation 2:
    - Rocking
    - Whirling
    - "Animal" walks
- Rhythmical movements
- Stimulation of auditory perception
- Oculomotor training
- Combination of motor and mental activities
- Social interaction

There are five distinct gait forms in human locomotion, which consist of walking, running, galloping, hopping and skipping. Of these the average 4 year old can perform the first four and most of them can perform the last one (Whitall, 2004). There are three categories suggested by Portwood (2003) as suitable exercise programmes for dyslexic children.

1. Balance tasks requiring the ability to remain in place while stretching the body (e.g., standing on one leg);

2. Motor tasks which are repetitive (e.g., running, rolling / tumbling, slithering, jumping, skipping and crawling – hands and knees then hands and feet).
3. Coordination skills which require tasks to be put together (e.g., "hop, skip and a jump").

A circuit can be developed to include walking on tip-toes, running, stretching, creeping, turning and moving in rhythm. She recommends "Simon Says" because it helps children develop their listening skills and plan their movement. This, she claims, will improve automatisation.

Ayres (1979) suggests that running, jumping or anything which lets the body move a lot is good for vestibular stimulation (p165) such as running, turning, bending, touching, pushing, pulling, rolling, crawling, climbing and jumping.

Goddard (1996) advises that any attempt at physical remediation should take into account the fact that development in children is both cephaolo-caudal and proximo-distal, and that it follows a set sequence of vestibular, tactile, auditory, visual and proprioceptive emerging skills. She advises stimulating the vestibular system (particularly where there is a retained TLR) by rotating, rolling, rocking, balancing (eyes open and closed) and by stretching and flexing the limbs.

DeQuiros and Schrager (1978) suggest that vestibular therapy with 6 to 7 year old children should include exercises based on posture (including exercises to reduce primitive reflexes); on kinesthesis and proprioception, such as walking, kicking and hopping in different directions; on body-awareness, recognition, action, imitation and representation; on vibration; on integrating different modalities etc. Because the eventual acquisition of body division, or "functional independence" of the body, is the ultimate aim, exercises based on vestibular-proprioceptive stimulation are necessary as these are linked to such division. The inclusion of exercises without visual stimulation is important in order to maximise not just eye-control but head and body balance as well. Crawling, falling, "lumbering" ("bear-like" walking), elephant walking, standing on one leg, walking, hopping, kicking, etc are all suggested by them as aids to improve balance and postural integration. Simply attempting to remediate reading and writing alone is seen as ignoring the cause of the problem. Such exercises, they say, "will develop control of the complex neuromuscular systems on which reading, writing and speech depend".

Spinning, rotating, bouncing, rolling and rocking are traditionally those used for normalising vestibular sensitivity (Crutchfield and Barnes, 1993). They also suggest that exercises designed to improve postural stability should involve the following: a decrease in the base of support (from wide to narrow standing), an avoidance of arm-use when balancing, a decrease in sensory cues (e.g., visual cues), a change from static to dynamic tasks, and an increase in difficulty (any combination of the above is particularly good). They also suggest that progress is made from activities such as rocking and walking to more complex movements such as rolling, going from prone to supine, kneeling, creeping and walking a balance beam. Total body balance and coordination movements include skipping, jumping and hopping. Cratty (1968) also suggests that when using static tasks, for example, the area of performance should be made smaller and the base of support should be decreased by asking the child to lift his arms or stand on one foot. Movements should include arm-leg integration, such as Jumping Jacks; travelling tasks such as rolling; and loco-motor tasks such as skipping and galloping.

The PDMS-2 (Folio and Fewell, 2000) suggests the following activities:

- Standing on one foot: This is used as a functional skill and is necessary for more advanced movements such as hopping.
- Walking: this improves the child's mobility and encourages exploration and discovery of new ideas.
- Running: this develops coordination between the arms and the legs.
- Hopping: this develops upper and lower body integration.
- Rolling forward: this gives the child a feeling of the change of spatial orientation.
- Galloping: this develops rhythm and coordination of the upper and lower body parts.
- Jumping: this develops coordination and directionality.
- Skipping: this develops rhythm and arm / leg coordination.

By the time a child is 3-4 years of age, McGlown (1990) suggests that they should be able to engage in walking, running, tandem walking, standing on one leg, skip and jump.

O'Dell and Cook (1997) from the Bender Achievement Centre suggest the following gross motor exercises, for approximately 10 minutes a day, five days a week; toe-touching, foot touching, arm twirls, side-bends, side-twists, kicking and balancing.

In conjunction with age-appropriate motor development norms (see chapter 3), an attempt was made to integrate these ideas and practices into a full motor programme which focussed on balance in order to stimulate the vestibular system. By setting the exercises to child-friendly stories (e.g., taken from Evetts-Secker, 1998) and games, it was hoped to engage the children's excitement and commitment to the routine (see Appendix 3-5).

7.3.3 Placebo-Control

It is of course necessary to employ a placebo-control group in order to eliminate the possibility that a Hawthorne and or placebo effect may be at work amongst the experimental groups. Bluechart and Shepard (1995) showed that improvements are often found in control groups which makes it difficult to interpret findings properly.

The placebo-control programme was composed of two sets of movements, a "patterning" set and a "facial" set of movements.

(1) Patterning

Patterning is that Doman and Delacato-devised method of imposing relevant movements on to a child's central nervous system, by a team of adults, employed when the child is unable to engage in the task alone (i.e., usually in the case of Cerebral Palsy) (Doman et al., 1960). The reasons for using patterning as a placebo-control are four-fold:

1) As previously discussed, patterning in general simply doesn't work beyond chance levels.

2) Randomising the programme's order has been found to be even less effective than not randomising it (Kavale and Mattson, 1983).

3) The current programme has used ideas and components of the original movements, which Doman et al. (1960) dismissed as having any efficacy.

4) The programme has some face-validity, given that it "looks" like a movement programme and it is important that parents believe this is a treatment group.

With regard to the theory that such a treatment will "impose patterns on the central nervous system" (Cohen et al., 1970) and on the basis that motor information reaching the brain is generally of two sorts; either kinaesthetic from the muscles regarding their state of tension, or proprioceptive from the joints regarding the position of the limbs, Cummins (1988) points out that it is unlikely that this sort of passive and repetitive input will have any significant affect on brain maturation and development. He explains that the passive aspect of the exercises goes against one of the fundamental precepts of brain function, because, although environmental stimulation is necessary for brain development, it is most effective when that stimulation is interesting and challenging to the child because the attention which the child puts toward the stimulation leads to excitation of neurons hence increasing dendritic connectivity. Passive movements are about as low a level of motor excitation as can be constructed.

In institutional settings there may have been an entire team devoted to the manipulation of one child. The original patterns used by Doman and Delacato, for example, are far too difficult for one adult (especially a mother) to achieve alone, and to request two or more people per night to work with a participant makes the study too demanding. Following the patterning idea but not employing the exact movements, would allow the mother (or father) alone to manipulate the child. While the movements involve the manipulation of the child's head, arms and legs, they are not identical to those proposed by Doman and Delacato (they are more simplistic) and do not reflect human motor development in any real sense. They nevertheless reflect the idea of patterning in that someone else is responsible for making the movements of the child.

Furthermore, Doman et al. (1960) claimed that the use of components of the programme would not lead to success.

(2) Oro-facial exercises

The oro-facial exercises were compiled by Cogan[32] (2002) in 1995 and she granted permission for their use in the current study as part of the placebo-control treatment. They were originally designed for use with stroke victims who had lost the use of their oral muscles and were suffering from Dysarthria. No part of the body was engaged in the movements of these exercises except for the tongue, cheeks and lips. Furthermore, these movements did not involve any vocalisation, articulation or speech patterns and thus cannot be said to invoke speech and language therapy movements.

Neither of the placebo-control approaches required the children (or their parents) to cross the midline of the child's body.

7.4 Conclusion

The beauty of the suggested possible methods for detecting and treating learning disabilities is, as Kohen-Raz explains, straight-forward, non-invasive and suitable for an educational setting. Kohen-Raz concludes that not only can the study of posture lead to a reliable method of psycho-motor treatment but also that it can teach us about the neuropsychological underpinnings of learning disorders. As we have seen, theories claiming similar sorts of things have not stood the test of scrutiny or time, and it remains to be seen whether the modern ideas in the treatment of dyslexia are a misguided (albeit genuine and well-intentioned) continuation of these practices or if they offer something new to the field of motor therapies and learning, particularly with regard to dyslexia. Kavale and Mattson (1983) presented two considerations in the study of therapies; firstly and simply, does it work? Secondly, as previously stated, is it causing undue harm, financially, emotionally or functionally in that students are removed from a better environment?

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[32] Cogan, P. (2002): Personal communication, Dublin, 2003. Cogan designed these tests as an M.A. student in neurology and linguistics at the Brugmann Hospital (Salle 16) in Belgium.

8 Chapter 8: Methodology

Method

8.1 Design

The study was of a longitudinal, experimental, mixed factorial design (2x4x3). The three main factors were "dyslexia risk", "treatment type" and "time sample", with repeated measures on the last factor. The study employed the double-blind, randomly-assigned, placebo-controlled gold standard of treatment efficacy research.

Dyslexia Risk was composed of 2 levels. In the attempt to add further balance to group composition, each child, once assessed, was labelled either "high" or "low" risk with regard to developing dyslexia, a score dictated by their level of retained reflexes (a score of above 2 was considered "high risk", a score below 2 "low risk"). Level of retained reflexes was used to decide risk because this was the initial focus of the research. It is important to stress that the number "2" was a somewhat arbitrary cut-off point, decided at a time before testing began. Therefore, it is not relied upon in the stages of analysis as a valid indicator of high and low retained reflexes.

Treatment Type had 4 levels, consisting of 2 experimental conditions and 2 control conditions. The experimental conditions included both a reflex replication programme and a balance programme; the control conditions included a placebo control as well as a standard control group or, as was explained to the parents of the participating children, a "delayed treatment" group.

The repeated measures factor, time sample, required that each child was assessed on 3 occasions over the course of a year. The initial assessment, necessary for establishing a set of baseline data, was followed by 2 review sessions to measure change, one 6 months into the child's programme and a final one at the end of the year-long programme.

As stated above, the study ensured that each child was randomly assigned to one of the 4 groups. This was achieved by generating a list of random numbers using an on-line random number service (Haahr, 1998). Unless they were assigned to the delayed treatment control group, the parents of the children in the study, as well as the children themselves, were unaware of which treatment group they were in. In addition, the experimenter responsible for scoring the participants' movements from video-data,

was blind as to the grouping of the child in question. Thus, this was a double-blind study.

8.2 Participants

The study recruited a total of 178 children. Of these 42 withdrew at some point during the study either officially or by failing to make scheduled appointments and being uncontactable. This left us with a total of 136 children who completed the entire study. These consisted of 117 boys and 19 girls, with ages ranging from 3 years, 3 months to 7 years, 4 months, at time of initial testing. The breakdown of group assignment, gender and age is shown in table 5 in the results section.

The sampling bias in gender reflects the disproportionately larger incidence of dyslexia in boys than girls. It is currently estimated that there are 3-4 times as many dyslexic boys as there are girls (Stein and Walsh, 1997). Because the treatments in question are marketed for dyslexic children it made sense to recruit those more likely to develop dyslexia - hence an initial appeal for boys only. Later in the study, girls were accepted, but only if they showed signs of having a problem which resembled dyslexia. The children were recruited through primary schools, Montessori schools, newspaper advertisements, the Dyslexia Association of Ireland (DAI) and radio interviews. All children had an immediate family history of dyslexia or reading / spelling disabilities which affected either their older siblings or their parents. This step was taken to maximise the likelihood that any observed difficulties would be the result of genetic influences and not environmental ones because it is estimated that the resulting sample would contain at least 40% who were at risk for dyslexia (Scarborough, 1990; Lahey and Edwards,1995; Hallgren, 1950; Bishop and Edmundson, 1986; Gallagher et al., 2000; Gilger et al., 1991; Pennington, 1990). Each child was labelled as being of either high or low risk of developing dyslexia (on the basis of the reflex tests) and randomly assigned to one of 4 groups.

There was some transfer of participants from the treatment groups into the control group where parents found it too difficult to implement the treatment they were assigned. This usually equated to only a couple of weeks of irregularly performed treatment.

8.3 Apparatus

- A pencil with rubber top for easy focus, and a ruler (in centimetres) for measuring distance, were used for the tests of visual-motor functioning.
- A packet of letter cards was used for the test of letter knowledge. These consisted of 26 lower case letters of the alphabet, drawn in black ink on 8.5 x 12 cm white cards (Collins Educational Letter-land flashcards, stage 1).
- A TIM digital stop-watch was used for timing children on some balance tasks.
- A digital video camera was used for filming the children's movements (Canon 3CCD digital video camcorder XM2PAL, 20x fluorite L series lens).
- Palm-pilot (Tungstern / E) was used to record the beginning and end of each test on the video for ease of locating when scoring at a later date.
- SPSS 12 for Windows was used for statistical analysis.
- Retrospective questionnaire.
- Tape-Recorder.
- Conner's Rating Scale for ADHD.
- Certificates and T-shirts for the children.
- Room-Layout & Floor Set-Up (Appendix 6)

8.4 Materials

The Dyslexia Early Screening Test, 2nd edition (DEST-2): Designed by Nicolson and Fawcett (2004), this is a 12 item screening test for children between the ages of 4.6[33] and 6.5 years. It takes approximately 30 minutes to administer. All necessary items are included in the test battery with the exception of paper for the child to write on and a container for the beads of one of the tests. A tape-recorder is also necessary on which to play some of the sub-tests. The predictive validity for this test appears to be good, suggesting as it does, that a cut-off point of 0.9 will catch 90% of those who can be diagnosed later as dyslexic from a full psycho-educational assessment (Fawcett et al., 1998). To catch those at "mild risk" for dyslexia a cut-off point from 0.6 - 0.8 is suggested. Nicolson and Fawcett (2004) suggest it as a good measure of whether a teaching method is benefiting a child (p. 76). The test has strong test-retest reliability (p

[33] All children in the study had reached this age by the time they were tested using the DEST-2.

< 0.001) with individual tests ranging from 0.637 to 0.878 (tests administered 1 week apart), and strong inter-rater reliability, with two experienced testers showing similar ratings (0.98) and one experienced and one inexperienced tester showing almost as similar ratings (0.94) (Nicolson and Fawcett, 2004, p. 69 & 70).

The Dyslexia Screening Test (DST): Designed by Fawcett and Nicolson (1996), this is an 11 item screening test for children between the ages of 6.6 and 16.5 years. It takes approximately 25 minutes to administer. All necessary items are included in the test battery with the exception of paper for the child to write on and a container for the beads of one of the tests. A tape-recorder is also necessary on which to play some of the sub-tests. Both the DEST-2 and the DST were standardised on English children. The predictive validity of this test is not known, however its concurrent validity is represented by the fact that of a sample of diagnosed dyslexics (n=17), 90% score above 1.0. Of a sample of normal children (n=20), all scores were below 0.3 (Fawcett and Nicolson, 1996). Test-retest reliability is above 0.7 for all tests (ranging to 0.9 for some tests; tests administered 1 week apart). Inter-rater reliability was 0.98 for two experienced tester and 0.94 for one experienced and one inexperienced tester (Fawcett and Nicolson, 1996, p.11 & 12).

Conners' Parent Rating Scale – Revised (CPRS-R): The CPRS-R was designed by Conners (1997), standardised in the U.S and revised in 2003. There are three scales; teacher, parent and self-report. The one used in the current study was the parent rating scale (short version), suitable for children aged 3-17 years. It contains 27 items and covers 4 subscales (Oppositional; Cognitive Problems / Inattention; Hyperactivity; ADHD index). The parent scale yields information about the child's behaviour in the home and other parental environments (Conners, 1997). The ADHD index contains items for distinguishing between possible ADHD and normal children and, thus, is useful for identifying those who may benefit from further investigation. In the current study it was used to identify those who may bring confounding variables to the study. The internal consistency coefficients for the CPRS-R ranges from .857 to .938 (for the ADHD index specifically, it was 0.938 for males and 0.921 for females). The test-retest (6-8 weeks) reliability coefficient was .72 (Conners, 1997, p113 & 119).

8.5 Procedure

8.5.1 Initial Work

Recruitment appeals began at the Dyslexia Association of Ireland's (DAI's) AGM in April, 2003, with a talk about the intended research. Further recruitment resulted from regional radio-interviews (Lite FM, June 2003; East Coast Radio, the "Declan Meehan" show, October 2003; South East Radio, the "Alan Maguire" show, November 2003; RTE radio 1, "Future Tense" with Ella Mac Sweeney, March, 2004); 3 national newspaper appeals (The Irish Times - "Education Today", 18/11/03; The Sunday Independent, 11/05/0; The Evening Herald, 01 and 02/07/04 (see example, Appendix 7). Notices were also put in the Irish National Teacher's Organisation (INTO) magazine, "In Touch" and "Profexcel[34]", in the summer of 2004, as well as the National Parents' Council newsletter, "Newsbrief" (Summer, 2004) and posted on the parents' website www.rollercoaster.ie this same summer. Contact with primary schools / Montessori schools, the Irish Speech and Language Therapists' association and the DAI regional workshops also aided in our recruitment. All of these, including every primary school in the republic of Ireland, was sent a letter outlining the aims of the study and requesting the principal or resource teacher to alert relevant families to the nature of the research (see Appendix 8). Montessori schools and DAI workshops were sent similar letters.

Several sources of information were used in deciding upon the tests and treatments employed (in addition to the review of the literature). Firstly, I attended the European conference on Neuro Developmental Delay, held in Maynooth, Co. Kildare, in October, 2002; secondly, I attended a training course run by Mary O'Connor of Neuro Development Therapy - Ireland (NDT) in Maynooth, Co. Kildare, in September, 2002. Thirdly, I met and spoke with Sally Goddard and Peter Blythe from the Institute of Neuro-Physiological Psychology (INPP), in Chester, England (December, 2002), about the testing of young children and the reflex treatments that can be used for them. Finally, another member of the research team attended a training course run by Martin McPhillips of Primary Movement, which focused on the early years of development. This was held in Dublin, in November, 2002. Thus, we collected information from

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[34] This is an on-line news and training service which aims to promote psychology and education (http://www.profexcel.net/).

NDT, INPP and Primary Movement and, therefore, along with the review of the literature, felt equipped to prepare a set of tests and treatments from what we had learned.

Ethics had been sought and approved from the School of Psychology's ethics committee (Trinity College, Dublin) (Appendix 9). The participants were slowly recruited over the course of one year (April, 2003- November, 2004), meaning they were all first assessed and began their programme at different times. On making contact with the researcher, each parent was sent a questionnaire, to be returned by the time of the initial assessment (Appendix 10). In addition, they were also sent an information sheet, a consent form and an insurance disclaimer (Appendices 11, 12 and 13 respectively). The questionnaire consisted of 3 main parts; section 1, dealing with general details of the family including contact details; section 2, with the child's developmental, medical and general behaviour details and Section 3, with the family history of dyslexia. The questionnaire was designed to ensure 2 principal things; firstly, that there was a family history either of dyslexia, or of a reading/writing/spelling disorder resembling dyslexia. This latter allowance was made for the reason that most of those people suffering from dyslexia in older generations would not have had an official diagnosis of dyslexia, whilst nevertheless suffering from the problem. Secondly, the questionnaire also allowed us to screen out those children whose problems may have been due to other factors, such as hearing or visual difficulties (in a section on general medical problems), hence the need for Section 2. This section also gave some insight into whether the child showed any other symptoms which are known to be common in dyslexic pre-schoolers. This included factors such as meeting language and motor milestones, learning nursery-rhymes easily, ease of toilet-training, behavioural development and prenatal / peri-natal history, as all are known to pose difficulties for the dyslexic child. The inclusion of developmental milestones (i.e., motor, language, behavioural), past medical history and family history satisfies most of the criteria, as specified by Palmer (1996), on collecting an adequate developmental history.

The information sheet, designed for parents of participants, detailed the aims of the study, the basic procedure of the study – including the use of a delayed treatment group (i.e., the no-treatment control group), the things that would be required of the

parent and the scale of commitment to the study. It also informed them of their right to withdraw from the study at any point and of the fact that the study satisfied the ethical guidelines of the Psychological Society of Ireland (PSI).

The consent form reiterated the basic aims of the study as well as detailing some finer points regarding the assessments. In signing this form, parents were agreeing to their child being video-taped and were confirming that they had been made aware of their anonymity, confidentiality and their right to withdraw.

A disclaimer form was issued to, and signed by, each parent, stating their agreement to participate at their own risk and not to hold Trinity College liable should any injury associated with the study occur.

The first families satisfying the criteria of the study were invited to "parent information evenings", held in the Department of Psychology in May and June, 2003. Appointments for an initial assessment were made at those presentations. All subsequent parents who made contact had their initial appointments made directly over the telephone. Each child's second and third assessment appointments were made on the day of their first and second appointments respectively. At the end of each initial assessment the parent was asked to sign a form agreeing not to lend any treatment materials, and to return the material at the end of the study (Appendix 14).

8.5.2 Testing

Testing took place inside the Department of Psychology, Trinity College, Dublin, in a quiet laboratory room. Parents had the option of remaining in the room with the child during testing and most chose to do so, sitting quietly in one corner opposite the child ("F")[35]. Two other people were present in addition to the child; the principal researcher and an assistant. The room was set up in a permanent way, so that each child had exactly the same surroundings. Green sheets were used to cover the walls and floors so that the child could be easily discerned at later video-scoring. For the same reason, all testers wore black clothes to conceal their own skin, allowing for clear identification of the child's limbs. The child was requested to wear t-shirt and shorts throughout testing

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[35] For a description of the room lay-out, please see Appendix 6. This also details the locations at which the children had to position themselves for each tests.

so that their elbows and knees were clearly visible. A digital video camera stood in one corner, opposite the child, and this was operated by the research assistant. Duck-tape floor markings ensured that each child performed the series of tasks in approximately the same places. Each child was tested on 3 occasions over the course of a year with each test lasting approximately one hour except for the final assessment which lasted about an hour and a half due to the need to include an extra, standardised, screening test for dyslexia.

There were 5 parts to each assessment which included tests of; general balance, retained reflexes, visual-motor functioning, fine motor skills and phonological skills. As one of the reasons for the current study was replication of previous research, many of the tests used here were those used by McPhillips et al. (2000) in their study of reflex replication efficacy. Attendance at a "Primary Movement" training course, held in Dublin in the Autumn of 2002, was undertaken by one of the researchers involved in the current project, as mentioned earlier, where notes on testing were issued from Martin McPhillips himself. At the final appointment each child was also tested on the Dyslexia Early Screening Test (DEST) or Dyslexia Screening Test (DST) depending on their age, to get an idea of whether or not they were at risk for developing dyslexia and, if so, the severity of this risk of dyslexia. Each task, in accordance with the advice given by Kohen-Raz (1996), was both verbally explained to the child and personally demonstrated by the tester. In addition, children failing their first trial were given a couple of opportunities to practise the task before a formal measure was taken. A copy of the testing and score-sheets can be seen in Appendix 1.

8.5.2.1 Balance

There were 8 sub-items of general balance (see Appendix 1). The tester demonstrated each test for the child then gave the child a chance to practise the task before attempting the actual test. Only when the tester was happy that the child understood what he was doing did the test version proceed. The sub-tests are listed below:

1) One-Leg Balance

2) Mann Test (Advanced Romberg)
3) Tandem Walk
4) Walking on Tiptoe
5) Slalom Walk
6) Hopping on the Spot
7) March and About-Turn
8) Crawling on all Fours

1) One-Leg Balance: This test had to be performed 4 times by the child; on each leg, eyes open and on each leg, eyes closed. The child was instructed to stand on one leg, bend the knee of the other leg and lift the foot off the ground (Appendix 1, test 1). If the child was under the age of 5 they were permitted to place their arms out at shoulder height to balance themselves. Above the age of 5, however, they were asked to place the hands on their hips. Using a stop-watch, the tester then scored, in seconds, how long the child could maintain balance on each foot under the above conditions. A child of 4 years should be able to maintain balance for 5 seconds and a child of 6 years should be able to do so for 6 seconds. In addition to the number of seconds the child could maintain balance, they were also scored on the quality of their balance. One-Leg Balance which was perfectly rock-solid, was scored as a "1", providing that the number of seconds that this position was held matched the age norms of the child (above). A slight wobble or elevation of the hands equal to or greater than 20 degrees, was scored as "2"; a definite sway or visible hand movement resulted in a score of "3"; if the child put the foot down on the ground within 5 seconds they were scored as "4" and if they were completely unable to perform the task they were scored as "5".

2) Mann Test (Advanced Romberg): This test was performed twice by the child; once with eyes open and once with eyes closed (as before). The child was instructed to stand still, placing one foot in front of the other, in a heel to toe position (Appendix 1, test 2). Children below the age of 4 were permitted to hold their arms out at shoulder level to aid balance, but children above the age of 4 were requested to hold their arms down by their sides. Once again, the tester recorded the number of seconds the child could hold the position and scored the quality of the stance. Every child in our age range should have been able to hold the position for at least 8 seconds. If they could do this properly

they scored a "1"; if their feet wobbled they were scored a "2"; if they swayed or had to readjust their arms for balancing they were scored a "3" and if they stepped out of the position within the 8 second minimum then they were scored a "4". As before, if they were completely unable to perform the task they were scored a "5".

3) Tandem Walk: The task required the child to walk heel to toe in a straight line between 2 line markings placed on the ground (Appendix 1, test 3). They were instructed to take their time while walking, in order to give them the best possible chance of performing the task accurately. If under the age of 5 the child was permitted to hold the arms out at shoulder level and to look down at their feet. An approximation of heel to toe was also acceptable from them. For those over 6 years old, the hands were placed on the hips and, in addition to walking forwards, they were also required to walk backwards. Each child should have been able to walk 10 steps in this manner. They were scored on the number of steps they took; counting of steps ceased immediately a foot came off the line. A perfect 10 steps scored "1". Inability to walk heel to toe over the age of 5 (i.e., taking large steps) was scored as "2". A major wobble was scored as "3"; the inability to keep the feet on the line or taking fewer than 10 steps was scored as "4" and completely unable or putting the foot down on the first step was scored as "5".

4) Walking on Tiptoe: This test was very similar to the previous one. It required the children to walk, on tiptoe, in a straight line (Appendix 1, test 4). The same rule applied to the arm positions and once again the child was only required to also walk backwards if they were above the age of 5. A score of "1" was given to a perfect 10 steps; a score of "2" if the child was only able to take large steps or if they shuffled backwards; a score of "3" if there was sway of 20 degrees from the vertical; a score of "4" if they were unable to stay on tiptoe or if they took fewer than 10 steps and finally a score of "5" if they were completely unable to perform the task.

5) Slalom Walk: Here the child was asked to walk along a single line marking on the ground (Appendix 1, test 5), alternately lifting each leg and crossing it over the line. The child stood initially to the right of the line with hands on hips, regardless of age,

lifted the leg at the knee and placed the foot on the other side of the line. They were allowed to look down at the feet. Again they were scored on their ability to take 10 of these steps and counting stopped if the foot did not cross the line or if the child overbalanced and put the foot down. As before, if the task was completed to requirement it was scored as "1"; if there was slight wobble or hesitance placing the foot over the line then this was a "2"; if there was body sway this was a "3"; if there are fewer than 10 steps then this was a "4" and if the child stepped on the line or put the foot down immediately (i.e., cannot perform task) then this was scored a "5".

6) Hopping on the Spot: The child was asked to hop (Appendix 1, test 6), on the spot, for at least 10 hops on each foot. He was instructed to hop for as long as possible[36]; first on one foot then on the other. If the child could do 10 or more hops he was scored as "1"; if there was some wobble on landing or some moving around whilst hopping he was scored as "2"; if the hops were performed very quickly without gaining any height or if the other foot was resting on the hopping leg, he was scored as "3", and if there were fewer than 10 hops he was scored as "4". Once again, if he was unable to hop at all he was scored as "5".

7) March and About Turn: In this test the child had to march around the testing area, swinging his arms and lifting his knees high (Appendix 1, test 7). The pattern of his marching was observed as either homolateral or cross-lateral. A score of "1" was administered if the marching was perfectly cross-lateral; a score of "2" if it began as cross-lateral but reverted to homolateral marching; a score of "3" if the marching was of mixed cross and homolateral gait; a score of "4" if it was fully homolateral and a score of "5" if the child was completely unable to march at all.

8) Crawling on all Fours: The child was requested to crawl along a line marking on the ground (Appendix 1, test 8). Once again the tester noted the laterality of the movement and scoring was similar to that of the marching. A score of "1" was administered if the

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[36] The masculine form of the personal pronoun will be used to denote both sexes to avoid stylistic awkwardness.

marching was perfectly cross-lateral; a score of "2" if it began as cross-lateral but reverted to homolateral marching; a score of "3" if the marching was of mixed cross and homolateral gait; a score of "4" if it was fully homolateral and a score of "5" if the child was completely unable to march at all.

8.5.2.2 Retained reflexes

There were 6 sub-items on this test (Appendix 1, tests 9-14). The tester explained each test to the child before doing the actual test. Each test was repeated a second time to allow for consistency in scoring after which the entire test was repeated with the child's eyes closed. All tests were scored for eyes open and closed and for all tests the child was instructed to keep as still as possible. Of the tests included here, numbers 1, 3 and 5 resemble the scoring method from the primitive reflex profile designed by Capute et al. (1978). These have been slightly modified by McPhillips (training course notes, 2002) and this was the scoring-system employed by the current study. Number 2 was suggested by Ayres (1972; 1978; 1979) and number 4 by Schilder[37], and the scoring method constructed by McPhillips[38]. Number 6 was suggested by Blythe and Goddard-Blythe[39] (2002) as more suitable for our age group. The sub-tests are listed below:

1) ATNR - Supine Version
2) ATNR - Ayres Version
3) STNR
4) ATNR - Schilder Version
5) TLR
6) Moro

1) ATNR – Supine Version: The child lay supine on the floor with the tester kneeling behind the head (Appendix 6, point "C"). The child's arms were placed at 45 degrees to

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[37] McPhillips, personal communication, December, 2002.
[38] McPhillips's Primary Movement training course, Dublin , December, 2002.
[39] Blythe and Goddard: Personal communication, at INPP, Chester, December, 2002.

the body with the elbows bent and the hands facing palm down with the fingers spread, their tips just touching the floor. The tester then positioned herself behind the head of the child and placed her hands of the child's head, just behind the ears (Appendix 1, test 9). The head was turned firstly to the left to approximately 90 degrees, with a pause of 5 seconds, then returned to the midline for 1 second, then turned to the right for 5 seconds. Only the side of the body to which the head was turned was scored. Scoring for the arms was as follows. If there was no movement at all in the arm then a score of "1" was recorded; if there was some tremor in the arm then a score of "2" applied; if the fingers moved a score of "3" was necessary and if the hand and arm both moved then a score of "4" was recorded. Scoring for the feet differed slightly, with no movement being given a "1" and any movement at all being given a "4". There were no 2s or 3s for foot movement.

2) ATNR – Ayres Version: The child was put in a table position (Appendix 1, test 10, facing the camera), resting on all fours with palms facing down and head looking at the floor. The tester kneeled to the right of the child, placing her hands on his head, just behind the ears. The head was turned firstly to the left by approximately 90 degrees, with a pause of 5 seconds, then returned to the midline for 1 second, then turned to the right for 5 seconds. In accordance with McPhillips's instructions, the side of the body to which the head was turned was scored, however, this is not consistent with the advice given by Morrison (1985) who cautions that there may be varying degrees of flexion in the occiput elbow and that this is the most reliable measure of the ATNR. Thus, both elbows were scored in case one yielded more sensitive measures than the other. Scoring for the arms was as follows. If there was no movement at all in the arm then a score of "1" was recorded; if there was some tremor in the arm then a score of "2" applied; if the elbow flicked or the child was attempting to keep the arm straight then a "4" applied and if there was a definite bend in the arm a score of "4" was recorded. There was no scoring for the feet. Twisting of the body or sinking of the shoulder were also recorded if they occurred.

3) STNR: Here the child was instructed to maintain the table position, but face the left hand wall ("G", Appendix 1, test 11), and perform the action without the tester guiding

the head. The child moved his head slowly downwards so that he could see between his knees, held that position for 5 seconds, then moved the head slowly upwards so that he could see the ceiling, which he again held for 5 seconds. Scoring was made for the arms and the trunk/legs separately. On the downwards motion the arms were scored as; "1" indicating no movement; "2" indicating tremor in the arms; "3" a flick in the arms and a "4" a definite bend. For the trunk/legs a "1" indicated no movement; a "2" indicated either 1 lurch backwards with correction of position or crossing of the legs; a "3" indicated 2 lurches backwards with correction of position and a "4" indicated either lurching backwards but with no correction of position or sitting on the heels.

4) ATNR – Schilder Version: The child stood with arms outstretched, at shoulder height, in front of the body and hands hanging loosely downwards (Appendix 1, test 12). The tester stood behind the child with hands placed on the head, behind the ears. The tester then turned the child's head to the left, approximately 80 degrees from the mid-line or as far as it would go, paused for 5 seconds, returned the head to the midline for 1 second and then moved the head to the right for 5 seconds. A short rest was given between testing with eyes and open and eyes closed as this test can be quite tiring on the arms. Once again only the side of the body to which the head was turned was scored. Arms were scored for degrees of movement from the horizontal or the vertical. If there was no movement at all or only up to 10 degrees, either downwards or in the direction in which the head was being turned, then the child scored a "1"; movement of 10-20 degrees scored a "2"; movement of 20-45 degrees scored a "3" and movement of more than 45 (i.e., 45 to 90) degrees scored a "4".

5) TLR: The child stood with feet together and arms down by the sides. The tester stood to the side, ready to catch the child should they lose balance. The child was then told to move the head slowly downwards such that they could see their toes, to hold this for 5 seconds, then to move the head slowly upwards such that they could see the ceiling (Appendix 1, test 13). Both knee movement and balance were monitored in this case. On moving the head downwards the child was scored for the movement that occurred in the knees, such that "1" represented no movement; "2" tremor in the knees; "3" little flicks in the knees and "4" a definite bend in the knees. On moving the head

upwards the child was scored for loss of balance such that "1" represented no movement; "2" represented definite sway in the body; "3" represented sway with arms involved and "4" a loss of balance.

6) Moro: The child stood, as before, looking straight ahead with feet together and arms down by the sides. The tester kneeled behind the child holding him gently by the waist. The tester then gently rocked the child forwards 2-3 inches, then back to the neutral position followed by rocking them gently backwards 2-3 inches (Appendix 1, test 14). If the child had no problem with backward or forward movement they were scored a "1". If their arms came upwards on the backwards movement they were scored a "2"; if they went forwards or backwards but stepped out of position, they scored a "3" and if they opted totally out of performing the movement (by shuffling or resisting movement) they scored a "4".

8.5.2.3 Visual-motor functioning

Four tests of visual-motor functioning were performed. For each test the child stood opposite the tester with feet together and arms by the sides. The tester explained what the child was expected to do and also demonstrated the task. These are listed below:

1) Visual Tracking
2) Hand-Eye Tracking
3) Fixation with Eyes
4) Near Point Convergence and Re-establishment of Binocular Vision

1) Visual Tracking: The child stood upright with feet together and arms by his side. The tester held out a pencil with a coloured top, vertically, about 3 inches below the child's eye level, approximately the same distance from the child as that length from the child's finger to elbow. The tester then instructed the child to keep the body and head completely still but to follow the pencil top with his eyes as it moved if from left to right for 10 movements. Eye movements were carefully observed for evidence of

anticipation, jumping or head movement. Children who could follow the movement of the pen smoothly, without jumping ahead of it, scored a "1"; if their eyes did jump ahead of the pen they scored a "2"; if there was head movement in following the head then they scored a "3" and if they were unable to focus on and follow the pen they scored a "4".

2) Hand-Eye Tracking: This process is similar to that of the previous test except that the child was responsible for holding and moving the pen. He was encouraged to reach for the pen with whichever hand he felt most comfortable, and to move it from left to right, following it only with eye-movement. This continued for 10 movements. Scoring was identical to that of the previous test with those able to follow the movement of the pen smoothly, without jumping ahead, scoring a "1"; those whose eyes jumped ahead of the pen scoring a "2"; those whose head movement in following the head then they scored a "3" and if they were unable to focus on and follow the pen they scored a "4".

3) Fixation with Eyes: Standing position was as before. Again the tester manipulated the pen holding it in the same position in front of the child. The child was instructed to move only the head, slowly from left to right, but to keep the eyes fixated upon the pen top. This continued for 10 movements. The tester waited until the movement was stable then observed the eyes for deviation from the fixation point which would indicate an ocular-reflex. Scoring was based simply on either the ability to maintain fixation ("1") or inability to maintain fixation ("2").

4) Near Point Convergence and Re-establishment of Binocular Vision: The child stood as before in the soldier position but this time with the left shoulder against a wall. The tester held the pencil, now in a horizontal position, with the coloured top pointing towards the child, at the child's eye level and about an arm's distance from their face. The child was instructed to fixate upon the end of the pencil as the tester brought it nearer towards the child's face and to shout "stop" when it no longer seemed clear or began to blur. At that point the tester then slowly withdrew the pencil away from the child and asked them again to indicate when they could see it properly. The 2 points were marked and the distance between them measured. If the distance exceeded 8cm

then convergence was considered to be slow and scored as a "4"; if it was any less than this it was considered to be adequate and was scored as a "1".

8.5.2.4 Fine motor skills

For the following tests the child remained seated for all but the first test. These were all explained and demonstrated to the child by the tester. There are 5 of them, as listed below:

1) Motor Control - Finger to Nose
2) Motor Control - Heel on Shin
3) Adiadochokinesia (Rapid Alternate Movement) - Fingers
4) Adiadochokinesia - Rapid Reversal of Hands
5) Adiadochokinesia - Finger and Thumb

1) Finger to Nose: The child stood opposite the tester with feet together and arms out to the sides at shoulder height. They were instructed to bring the tip of each forefinger alternately to the tip of the nose 10 consecutive times. This was done first with the eyes open then repeated with the eyes closed. The child scored "1" if they could perfectly perform the task; "2" if they were bringing the head to the nose or the side of the finger to the nose; "3" if they were hitting the side of the nose; "4" if they were hitting the face and a"5" if they were completely unable to perform the task.

2) Heel on Shin: The child and tester sat in chairs opposite one another. The child was instructed to place the heel of one foot just below the knee of the other and to slide the heel slowly down the front of that leg. At the bottom of the leg they were required to push the foot outwards. This was then repeated with the other leg. If they could perform this task correctly they scored a "1"; if the heel went to the side of the leg they scored a "2"; if the inside of the foot went down the leg they scored a "3" and if they were unable to do it they scored a "4".

3) Adiadochokinesia (Rapid Alternate Movement) – Fingers: The child held out one hand and brought the index finger over to tap the thumb followed by the middle finger, 4th finger, little finger then the same in reverse order. They continued this rhythmic sequence for 10 consecutive taps then repeated the sequence with the other hand. If the tapping had a perfect rhythmic ripple it was scored a "1"; if the child could do it but was consciously performing the task then they scored a "2"; if they could do it but it was slow and laboured they scored a "3"; if they couldn't do the movement with one or more fingers they scored a "4" and if unable to do it all then they scored a "5".

4) Adiadochokinesia - Rapid Reversal of Hands: The child was told to put both hands on the knees with one facing palm down and the other facing palm up. They then had to lift both hands simultaneously and slowly reverse their orientation such that the hand that had been facing palm up was now facing palm down and vice-versa. They then returned the hands to the knees. They were to attempt 10 rotations. A fast and automatic speed was scored as "1"; if the action was correct but slightly slower then it scored a "2"; if they had to really had to think about it but could still perform the movement then they scored a "3"; if they could not turn their opposite hands they scored a "4" and if they could not perform the movement at all they scored a "5".

5) Adiadochokinesia - Finger and Thumb: The child was asked to stretch out the hands, stick out the index finger and thumb of each hand, fold back the others and to place the opposing index finger and thumb together. They were then told to separate the digits pointing towards the floor, to twist them upwards so that they were pointing up at the ceiling and to join them together again. The child was timed until they had completed 10 of these rotations. The number of seconds was recorded if they were able to perform the movement and if they could not then they were scored as "unable".

8.5.2.5 Phonological skills

A record of whether or not the child was in school was taken along with the class they were in and whether or not they were familiar with "Letter-Land". There then followed 3 short tests, listed below;

1) Letter Name Knowledge
2) Phoneme Deletion
3) Non-Word Repetition

1) Letter Name Knowledge: The tester held up a plain card with a lower-case letter[40] printed on it and the child was tested on his knowledge of the alphabet by correctly identifying what it was, either by giving its name or by giving the sound that it makes. The child was permitted to either name the latter or to make the sound associated with it. All 26 letters were held up, in random order, and a record taken of the ones the child got wrong or did not know.

2) Phoneme Deletion: In this test the child was required to remove the initial phoneme of a one-syllable word and say the rest of the word. The tester told the child the word loudly and slowly and asked him to repeat it. The tester then explained to the child that they had to try to say that word without the beginning part. There were 4 example words which the tester went through with the child before proceeding to the 8 test words. The child was scored on the number he could do.

3) Non-Word Repetition: This was a relatively simple test in which the child was required to repeat the non-words spoken by the tester. Devised by Gathercole et al. (1994) "The Children's Test of Nonword Repetition" consisted of 10 simple words of one syllable (e.g.,"sa", "fu") followed by 10 slightly more complex one-syllable words (e.g., "smip", "clird") which progressed on to 30 more complex 2-4 syllable words (e.g., "hampent", "skiticult" "woogalamic"). The words of 5 syllables were not used here. The child was scored on the number they could repeat properly.

The Dyslexia Early Screening Test (DEST): The DEST-2 had 12 different sub-tests. The child was requested to sit down on a chair close to the tape-recorder and told

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[40] The scoring-sheet shows upper case letters but lower case was printed on the cards that the children viewed.

that they were to engage in a few more games. The instructions for each sub-tests were given and a practice session, as per the standardised instructions was given before each sub-test. The DEST-2 included the following set of sub-tests:

1) Rapid Naming
2) Bead Threading
3) Phonological Discrimination
4) Postural Stability
5) Rhyme / First Letter
6) Forwards Digit Span
7) Digit Naming
8) Letter Naming
9) Sound Order
10) Shape Copying
11) Corsi Frog
12) Vocabulary

The child's score for each of these was determined on the basis of normed ranges. Their overall score was then calculated. If this fell between 0.5 - 0.9 the child was considered to be at "mild risk" for developing dyslexia. If the score was above 0.9 the child was considered to be at "high risk" for developing dyslexia (see Appendix 15 for score-sheet).

The Dyslexia Screening Test (DST): Instructions were as with the DEST-2. This test included the following sub-tests:

1) Rapid Naming
2) Bead Threading
3) One Minute Reading
4) Postural Stability
5) Phonemic Segmentation
6) Two Minute Spelling
7) Backwards Digit Span
8) Nonsense Passage Reading

9) One Minute Writing

10) Verbal Fluency

11) Semantic Fluency

The child's score for each of these was determined on the basis of normed ranges. Their overall score was then calculated and if it fell above 1.0, he was considered to be at risk of developing dyslexia (see Appendix 16 for score-sheet).

The reader is referred to the test manuals for information on the standard test administration used here (Nicolson and Fawcett, 1996; Fawcett and Nicolson, 2004). Each parent was sent a report of their child's DEST / DST results, detailing their child's performance and informing them of further steps they should take should they be concerned (see Appendices, 17 and 18 for samples).

On the third testing occasion, the attending parent was requested to fill out 2 further questionnaires. One of these was the "Retrospective Questionnaire", the other was the Conner's Rating Scale - Revised (CRS-R) for ADHD..

Final Questionnaires

1) Questionnaire 2

This was designed in order to investigate the quantity and quality with which each child performed their respective programme. This questionnaire also enquired about other extra-curricular or academic programmes the child might have undertaken throughout the previous year, and whether any formal diagnoses (of dyslexia or otherwise) had been made during that time. A copy of this questionnaire may be seen in Appendix 19.

2) Conners' Parent Rating Scale – Revised (CPRS-R): Short-form

As mentioned, the parent was also asked to complete the "Conner's Parent Rating Scale – Revised" (CPRS-R) for Attention Deficit Hyperactivity Disorder (ADHD)

(Appendix 20). It was explained to them that ADHD and dyslexia are often co-morbid and it was necessary to determine whether their child's performance might be influenced by one factor or another (or indeed both). The parents were given the time of the screening test (i.e., approximately 30 minutes to complete both this and the retrospective questionnaire and asked to complete it with respect to the child's behaviour in the last month. Consideration was given to the possibility that parents may also have a reading disorder and as such, the researcher offered to go through the forms with the parent if that was preferable. This also maintained the validity of the responses by eliminating reading error / misinterpretation. In the event of an unanswered item, an average was determined for that scale, as instructed in the rating scale manual. The ADHD index is the best initial indicator of whether a child is likely to have an attention problem (Conners, 1997). A t-score of 65 or above on this index was taken to indicate a clinically significant problem (again as recommended by the manual). This corresponds to a percentile point of 93.3.

Token of thanks

Before leaving on the final occasion, each child was presented with a "Dyslexia Early Years" emblazoned certificate and t-shirt (Appendix 21). They were thanked for helping us with our work and for their cooperation. It was explained to each parent that they would shortly receive a copy of their child's "Dyslexia Screening Test" report (e.g., samples in Appendices 17 and 18), through the post, indicating their child's degree of risk for dyslexia and their individual tests results, and that, should they have any questions, they should contact us immediately. Furthermore, care was taken to explain the following two points: (1) that this report was not meant as a substitute for a full psycho-educational assessment and as such was not of use in the application for special waivers and resource hours from the Department of Education and Science and (2) that regardless of the risk level, there was always the chance of a "false-negative" or "false-positive" and they should take academic performance and / or family history into consideration in monitoring their child's progress in school.

Each family was also informed that they would receive a short report of the findings when the study was completed.

8.5.3 Treatments

There were 4 groups in the study and each child was randomly assigned to one of these. They consisted of a control group, in which there were no particular exercises, 2 experimental conditions (the "reflex replication" group and the "vestibular stimulation" or "balance" group) and the placebo-control group. All treatment groups required the child to perform their relevant treatment exercises for approximately 10 minutes on a daily basis. The parents of the children were sent both a video and a manual (see DVDs and Appendices 3-5). The video showed how the exercises should be performed and the manual incorporated stories and games in a scripted fashion which matched those exercises shown on the video. The manuals consisted of a contents page, an introductory letter and an instruction page after which the exercises were presented in an orderly manner. At the back of each manual was a log book for each day of the year; this allowed the parent to see at a glance which exercise to do on which particular day and which page the instructions were on. It also provided a column the parents were to tick to confirm that the exercises had been performed that day. Following this there was a comments page where the parents were encouraged to write about any problem they had encountered on any day of the programme. The different treatments are discussed in more detail below.

8.5.3.1 Group 1 – Reflex replication

These exercises were based on the same ideas behind Primary Movement and Neuro-Developmental Therapy from INPP, Chester. There were 4 different exercises in total, the first of which corresponded to inhibition of the ATNR, with the other three corresponding to inhibition of the Moro reflex. The child's parents were instructed to help the child perform the relevant movement on a daily basis. Precise instructions were given as to the start position and subsequent movements of the exercise in question followed by suggested stories to accompany them (Appendix 3). All exercises, once mastered with the eyes open, which would eventually take approximately 4 minutes, were to be then attempted with the eyes closed, bringing the total time per night to approximately 8 minutes. These exercises lasted for a period of 24 weeks, after which the child repeated the entire programme to bring the total period up to 48 weeks. The important thing about the exercises in this treatment was that they were performed

very slowly and carefully. They were designed to get the child to imagine a slow-moving scenario and to pretend to watch and follow it as they did the exercises. All movements took place with the child sitting in a low chair so that their feet were placed flat on the ground. Parents were permitted to incorporate their own stories if they so wished. Details of these exercises are as follows (see also Appendices 3-5).

1) Exercise 1: 12 weeks (ATNR)
2) Exercise 2: 4 weeks (Moro)
3) Exercise 3: 4 weeks (Moro)
4) Exercise 4: 4 weeks (Moro)

1) Exercise 1 - ATNR: Although only one exercise relates to the ATNR, the number of weeks spent on it is equal to the number of weeks spent on the Moro. This exercise lasted for 12 weeks. Each performance involved movement on the right side, followed by movement on the left side. The child sat on a chair, feet crossed with the appropriate foot in front (i.e., right over left when movement was to the right and vice-versa when movement was to the left). The arms were crossed at the mid-line of the body, with each hand resting on the chest, pointing towards the shoulder. The exercise involved slowly moving in the following pattern:

- Head turn to the right
- Right arm lifts up and out, bent 90 degrees at the elbow so that hand is at face level.
- Each finger touches in the thumb in successive directions.
- Arms returns to mid-line.
- Head returns to the mid-line.

This is then repeated on the left. Each of these movements must be slowed over the first week of performance, until it lasts for 30 seconds every time it is performed.

The suggested stories for this exercise consisted of watching "A dragon-fly in flight", "A spaceship" and "A little bird flying".

2) Exercise 2 - Moro: This exercise lasted for 4 weeks and focused on slow movements of the head. The child sat in the same position described in the first exercise but with the chin resting on the chest. It too, was performed twice, once with the feet crossed in one direction and once with them crossed in the opposite direction.

- Head slowly rises to the ceiling.
- Head remains towards ceiling.
- Head returns to chest.
- Head rests on chest.

Over the first two weeks the child worked on extending these movements so that each one lasted one minute. The suggested stories for the exercise consisted of watching "The little daisy", "The spaceship landing" and "The bird asleep".

3) Exercise 3 - Moro: This exercise also lasted 4 weeks and involved both the head and one of the arms simultaneously. It was performed twice, first on the right side and then on the left side, with each side consisting of 3 movements.

- Head moves up whilst right arm extends outwards.
- Each finger touches the thumb in turn in both directions.
- The arm and head return down / to the mid-line.

This was then repeated on the left side and as before, each movement had to last one minute. The suggested stories were "The little daisy begins to grow", "The little spaceman is not too sure" and "The little bird wet in the rain".

4) Exercise 4: Again the exercise lasted 4 weeks. It involved the head and both arms simultaneously and again had 3 movement parts to it, which, as before, were to be performed once with the feet crossed in one direction and once with them crossed in the opposite direction.

- Both arms extend from the mid-line, outwards, whilst the head is raised upwards.
- Fingers touch the thumbs in turn, in both directions.
- Head and arms return to the mid-line and chin.

This too must last one minute in each direction. The suggested stories were "The little daisy", "The little spaceship gets ready to fly home" and "The little bird sunning herself".

8.5.3.2 Group 2 – Balance programme (vestibular stimulation)

These exercises were all based on movements which involved dynamic or stable balance skills. They were designed in such a way that they were appropriate for our age group, there was a variety of activities, the activities required no special materials and they could be performed anywhere with a ten foot space (i.e., most houses). There were 7 of them in total; the first 4 were presented under the heading "John's big adventure" and were primarily made up of games. The next 3 were all based on traditional stories adapted from "The barefoot book of mother and son tales" (Evetts-Secker, 1998). It was felt that these were appropriate stories as it would probably be the child's mother who was helping him perform the exercises (Appendix 4). As there were 7 stories, one was performed each day of the week, in a serial fashion, for a period of 48 weeks. Each exercise was preceded by a short introductory page and the exercises themselves were presented in a line by line format, making them easy to follow. The parent was to read the top line and have the child perform the actions in bold, presented underneath. In order to ensure that the exercise lasted approximately 10 minutes, each static movement was to be performed for a period of 10 seconds and each dynamic movement to be repeated 10 times. The exercises are listed below and then described in further detail.

John's Big Adventure:

1) John in the Jungle
2) John and the Giant
3) John and the Animals
4) Simon Says

Traditional Stories:

5) The Goddess of Luck
6) Lucky Hans

7) The Magic Doll

1) John in the Jungle: This exercise identified a fictitious boy called John who found himself in a jungle and had to make his way out in order to get home in time for dinner. However, there were certain things he had to do in order to accomplish this task, such as row a boat across a river, fly an airplane etc. The child had to perform a series of running, jumping and balancing tasks to enact getting out of the jungle. At the end of this session John decided to have a rest which led on to exercise 2 the next day.

2) John and the Giant: John now met a giant who would aid him in his journey out of the jungle only if John imitated various modes of transport. The child engaged in spinning, bouncing and galloping tasks whilst imitating helicopters, pogo sticks etc. This again resulted in a rest before the giant would help John by bringing him to the animals, whom he thought would know how John could get home.

3) John and the Animals: Here John met many different animals who insisted upon him acting like them before they would help him. Thus, he had to gallop like the horse, march like the ants, leap like the frog, crawl like the crab, fly like the bird and so on. This again led to a rest before day 4 ended the story.

4) Simon Says: This exercise in based on the children's game "Simon says". The animals brought John to Simon, whom they say will help him get home. Simon requested that John do all the tasks that he says, before he would help him. These included such things as jumping jacks, stretching, tumbling and other general balance specific tasks. It ended with Simon helping John to get home for his dinner.

5) The Goddess of Luck: Day 5 began the movements based on traditional stories. This story was based on a traditional Nepalese story in which a young boy attempts to help his mother earn money by looking for the Goddess of luck. Again it incorporated plenty of running, hopping, jumping and swaying tasks as the child enacted the story.

6) Lucky Hans: This story is based on a traditional German story. The young boy has finished working for his employer and wants to go home to his mother. On the way home he encounters many animals, and once again, must copy their movements to achieve some gain. This incorporated many of the previous balance tasks as well as such things as clapping, waddling and rolling.

7) The Magic Doll: This story is loosely based on a traditional Wabanaki story in which a boy is given a magic doll which grants wishes. In this version of the story, however, the doll will only do as the boy wishes if he performs certain exercises. Bowing, marching and skipping are all features of this exercise.

8.5.3.3 Group 3 – Placebo control

The placebo-control exercises were all carefully selected and prepared so that they allowed for neither vestibular stimulation nor reflex replication nor, indeed, any movements that crossed the mid-line of the body or involved gross muscular activity on the child's part. However, they still allowed for parental involvement with the child for a period of 10 minutes each night and, the procedures, as described in the manuals, were as precise as the previous manuals, in order that they had sufficient face validity.

There were 8 exercises comprising the placebo-control programme which were, again, divided into 2 sets of games (Appendix 5); the first termed "Parental help games" and the second termed "Face games." "Parental help games" required the parent to take *full* responsibility for manipulating the limbs of the child in accordance with a particular story-line whilst preventing the child from generating *any* self-made movements. The child lay on the floor or bed or sat propped up by pillows on a chair so that they did not have to support any of their own weight. They were instructed to let their body "go floppy" whilst the parents held their arms or legs in particular positions for approximately 10 seconds. As for the "Face games" these included a series of story-lines and games in which the parent and child made movements with the tongue, nose and mouth but moved no other muscles. The parent was instructed to ensure that the child held the expression for approximately 5 seconds. After a month, in which the child got 3 consecutive days of each game, in order to get used to it, all the games were

then randomised in order for the remaining 44 weeks. The games are described in further detail below:

Parental Help Games

1) The Teddy Game
2) Mr. Jelly
3) Finding Santa
4) Walking in the Jungle

Face Games

5) Simon Says
6) Party Food
7) Snap
8) The Slow-Fast Game

1) The Teddy Game: This game involved teddies, dolls or any 2 items to which the children may have been attached, and was performed by firstly lying comfortably on the ground and then repeated with the child propped up in a chair. The 2 different items were placed, one on either side of the child, alternating between the legs and the arms. The child's task was to guess which item was where after the parent's prompt of pointing the relevant limb at the relevant item. This continued in mixed sequence for approximately 10 minutes.

2) Mr. Jelly: This was a story involving 2 characters called Mr. Jelly and Mrs. Nelly who take a trip to the sea-side. On reaching the sea-side Mr. Jelly (the child) becomes all floppy and cannot move so the Mrs. Nelly (the parent) must help him to do things to enjoy the day, such as getting to the sea, swimming in the sea, eating his lunch, etc. Thus, the parent moves the relevant limbs responsible for the various actions. The parent is requested to repeat the story a second time if it does not last 10 minutes.

3) Finding Santa: This was yet another story which required the parent to move the child's limbs to match a series of actions. A toy soldier falls out of Santa's bag at

Christmas time and the story tells of the soldier's attempts to find Santa in order to be delivered to his Christmas destination. The parent does cycling and running motions with the child's legs, waving and pointing motions with the child's arms and so on.

4) Walking in the Jungle: Similar to one of the previous stories about the jungle, this story is about a little boy who is walking in the jungle and meets lots of animals whose characteristic motions he copies. However, in this case it is the parent who is manipulating the child's limbs in accordance with the flapping motion of a bird's wing or the up and down movement of an elephant's trunk (for example).

5) Simon Says: This was the first of the Face Games and was similar to the well-known child's version of the game except that there was no "catching out" of the child. The parent and child were instructed to sit opposite one another, with the child sitting in a high backed chair so that the head was supported and the parent ensuring that they keep the head and body still. The parent demonstrated one of 15 different movements and the child must copy it accurately (or persist until is has been accurately copied). These 15 movements are repeated 4 times, in random order each time, to lengthen the time they take.

6) Party Food: Here the parent narrates a story about a little girl whose mother is making her a birthday tea. There are lots of different, delicious foods which the child smells, tastes and eats. These provide the bases for the facial movements the child must make in conjunction with the story.

7) Snap: This is essentially a guessing game, similar to the popular and well-known child's game of "Paper, rock, knife", in which 2 people simultaneously make a movement. Here the child can make any of the facial movements they wish, at the same moment that their parent makes any one they wish, to see if they make the same one. If they do, it is a "snap". Because it is necessary for the child to be familiar with the facial movements before being able to play this game, they do not get to play it until they have had a few weeks' practice with the other games.

8) Slow-Fast: The parent gives a prompt to the child as to which face they are about to do and whether they are to do it slowly or quickly, but the object of the game is for the child to try to do it more slowly or more quickly than the parent. Each of the movements is worked through and the game is repeated, if necessary, to bring it up to 10 minutes.

9 Chapter 9: Results

The results are split into two sections: Descriptive Statistics and Inferential Statistics. Descriptive Statistics covers proportion of dyslexics, group assignment, break-down in age and gender, programme compliance, issues of power and inter-rater reliability as well as the different samples analysed and the aims of the study. Inferential Statistics covers treatment efficacy using chi-square and MANOVA, and predictive efficacy using multiple and logistic regression.

9.1 Descriptive Statistics

9.1.1 Proportion of dyslexics

The final sample (discussed below) numbered 136 children. The number scoring at risk for dyslexia, when tested at the end of the study, was expected to be approximately 50% (see section 1.2.11.2). Where dyslexia was classified on the more stringent DEST / DST basis, only 41 children were classed as dyslexic, rising to 53 when the milder criterion was used. These numbers represent approximately 30% and 39% of the full sample respectively, which supports the findings of Gilger et al. (1991) and Pennington (1990) who put the familial risk rate for dyslexia at the lower percentage of 35 - 45% from the Colorado Family Reading Study. The low percentage suggests that means analyses may be swamped by the larger numbers not classified as dyslexic and results should be interpreted in the light of this. Chi-square analyses were employed to try to address this problem.

It has been suggested that half of those diagnosed with either dyslexia or ADHD show symptoms of the other condition (Stein, 2004). The current study showed that of those classed as dyslexic, 39% also showed evidence ADHD (36% where the milder form of dyslexia was considered). Of those classed as ADHD, 51% also scored at risk for dyslexia, which rose to 61% when the milder form was considered.

9.1.2 Group assignment

The initial number of children recruited and assessed at time 1 numbered 178. These were randomly assigned to the four various groups, with 59 in the reflex programme,

50 in the balance programme, 31 in the placebo-control programme and 38 in the control group[41]. Over the year these groups were subject to withdrawals and some unavoidable "reassignment" of participants. The 42 withdrawn children left a sample of 48 in the reflex group, 46 in the balance group, 23 in the placebo-control group and 19 in the control group. Problems that occurred subsequently, such as deaths in the family or the child suffering a medical illness (e.g., haemophilia) meant that 10 children who had been assigned to a programme initially (i.e., groups 1-3) never actually started their programme and had to be transferred to the control group (group 4). Similarly, 11 children who had been in a programme but could or would not continue it, and completed very little of it, were also counted as group 4[42]. This meant that the final sample comprised 39 children in the reflex group, 38 in the balance group, 19 in the placebo group and 40 in the control group. This distribution is illustrated in Figure 12.

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[41] For consistency throughout the remaining chapters the groups will be referred to by their programme names. Where group numbers appear in diagrams or charts, the following should be remembered:
Group 1 – Reflex replication programme (referred to as the "reflex group")
Group 2 – Balance programme (referred to as the "balance group")
Group 3 – Placebo control programme (referred to as the "placebo group")
Group 4 – Control group.

[42] A t-test was run to compare the mean DEST / DST scores of those who had done some of the programme and moved groups with those who remained in the initial group. This showed no significant difference between the two groups ($t = -1.7$, $df = 14$, p .107, 2-tailed). None of those who moved groups had done any more than a few weeks of their programme.

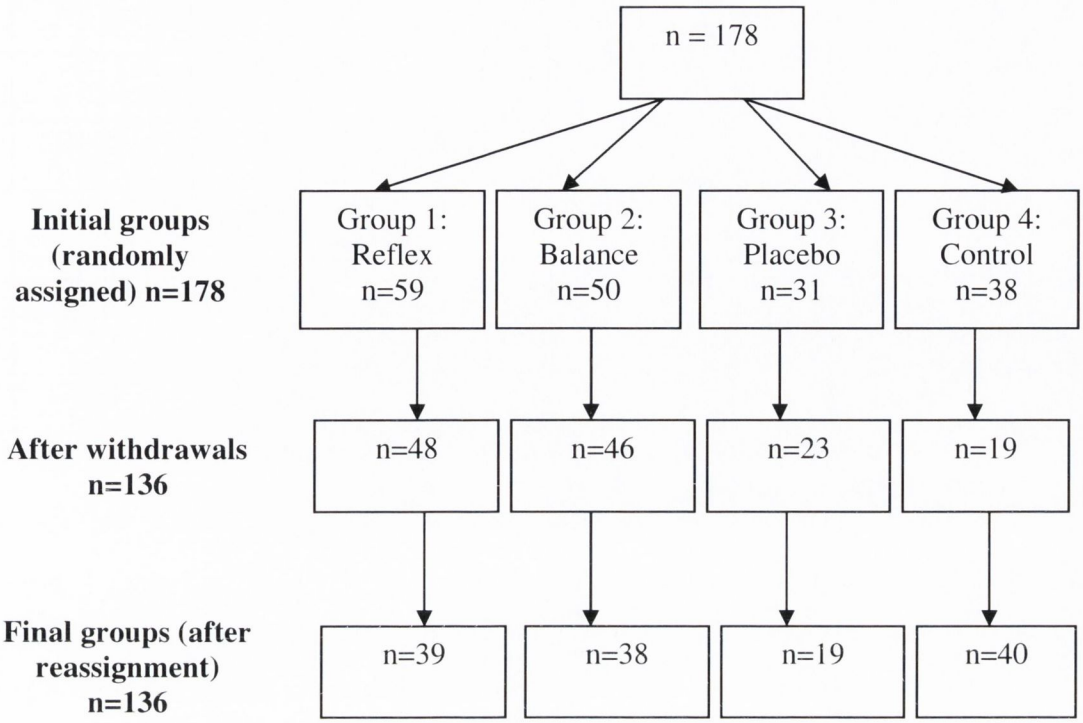

Figure 12: The formation of groups engaging in programme participation.

9.1.3 Age[43] and gender

Thus, the final sample consisted of 136 children. Of these 19 were girls and 117 were boys. Ninety-six of these were involved in a movement programme, be it experimental or placebo. The age range was from 3 years, 3 months to 7 years, 4 months, with a mean age of 5 years, 5 months and a standard deviation of 12.5 months. The breakdown of ages and groups is summarised in table 5.

	GROUP								
	Reflex		Balance		Placebo		Control		
Gender	F	M	F	M	F	M	F	M	**Total**
Age(years)									
3	0	1	0	7	0	1	1	1	**11**

[43] All ages refer to age of children at the start of the study (unless otherwise stated).

4		3	6	1	8	0	6	1	13	**38**
5		3	9	0	11	0	4	2	9	**38**
6		5	7	0	11	2	6	1	10	**42**
7		0	5	0	0	0	0	0	2	**7**
Total		**11**	**28**	**1**	**37**	**2**	**17**	**5**	**35**	**136**
Mean age*		5,7		5,5		5,8		5,4		5,5
S.D. **		13		13		11		12		12.5

Table 5: Totals of children, broken down by group, age and gender (n = 136).
* Years, months; **months

Of those 39 children in the reflex group, 5 of them (1 girl and 4 boys) were participating in privately run versions of this therapy. However, this sample could be selected as a distinct group and, where necessary, relevant analyses were run both with and without them. It was not possible to equate the ratio of girls to boys across the groups given that there were so few girls involved in the study. This is not of concern however because although girls may be less likely to be at risk of dyslexia in the general population, this does not necessarily seem to be the case within families with a history of dyslexia. As previously discussed, one of the reports from the Colorado Family Reading Study looked at the predictive importance of affected parents for the two genders. Here the risk for males was increased by a factor of 4 - 10% and the risk for females was increased from 8 - 13%. Thus, while the incidence of reading disabilities may be lower for females than males in the general population (2.2% and 7.8% respectively, where the overall incidence is 5%), the presence of a parental history may increase the *relative* risk more so for females than for males (Vogler et al., 1985) and yet the groups containing more females (i.e., the Reflex and Control groups) were not significantly greater than the other group in terms of their DEST / DST scores. In addition, the difference in spread of ages across groups was not significant. Not only was there no difference in the mean ages, measured in months, between the groups ($df = 3$, $F = 3.01$, $p > .05$) but Appendix 24 shows that whether or not age is controlled for is the treatment efficacy analyses, the findings are the same.

9.1.4 Programme Compliance: Quality and Quantity

9.1.4.1 Quality

The retrospective questionnaire (Appendix 19), administered to parents at the final testing session, was analysed to determine whether there were any uncontrolled differences between the programme groups (i.e., groups 1-3). There were 78 people who had engaged in their programme for the minimum desirable length of time (for obvious reasons there were no children from the control group). The group breakdown was: Reflex, n = 35; Balance, n = 27; Placebo, n = 16. The qualitative differences explored included:

(1) Estimated average length of time spent doing the programme per day
(2) Estimated general quality of programme performance
(3) Participation in sports independent of the programme
(4) Participation in academic help, either inside or outside of school
(5) Participation in other specialist help (e.g., speech and language therapy)
(6) Participation in any other extra-curricular programmes (e.g., drama; art & crafts).

Chi-square analysis was employed to examine the data. This revealed that there were no significant group differences on any of the above variables. Thus, it is unlikely that any differences found in later analyses might be due to the confounding effects of alternative interventions, extra help or any inadvertent motor-improving activities. The results of the chi-square analyses are presented below and percentage break-downs can be seen in Appendix 22.

Time (minutes): $\chi^2(6) = 11.2,\ p > .05$

Quality : $\chi^2(6) = 1.4,\ p > .05$

Sport: $\chi^2(2) = 4.08,\ p > .05$

Academic: $\chi^2(2) = 1.64,\ p > .05$

Specialist: $\chi^2(2) = .36,\ p > .05$

Other: $\chi^2(2) = 5.5,\ p > .05$

Other questions featured on this questionnaire concerned the child's attendance at school, where parents were requested to tick approximately how many days the child had missed during the year, and the possibility of comorbid disorders. Here, parents were asked whether the child had been diagnosed with any other developmental disorders since joining the study, such as ADHD, Asperger's, dyspraxia, etc.

These data was not exposed to any formal statistical analyses because a review of the material suggested little variability. No child missed more than a couple of weeks of school (with several days being the norm). There were few formal "other" diagnoses as the children were still quite young.

Chi-squares and Pearson correlations[44] were also run to determine if there were any differences between the proportions of dyslexics v non-dyslexics[45] (and ADHD children v non-ADHD children) in terms of the number of minutes spent doing their programme each night and the degree of control they exerted when engaged in the programmes (i.e., "quality"). There were no differences between dyslexics and non-dyslexics either in terms of the time per night they spent on their programme or on the degree of quality they exerted in doing the programmes. There were, however, differences between those at risk of ADHD and those who were not, on both of these measures. The results of the chi-square analyses are presented below and percentage break-downs can be seen in Appendix 23.

- Time (minutes) - dyslexia: upper risk level : $\chi^2(3) = 2.5, \quad p > .05$
- Time (minutes) - dyslexia: lower risk level : $\chi^2(3) = 1.76, \quad p > .05$
- Time (minutes) - ADHD: $\chi^2(3) = 8.5, \quad p < .05$
- Quality - dyslexia: upper risk level: $\chi^2(3) = 1.37, \quad p > .05$
- Quality - dyslexia: lower risk level: $\chi^2(3) = 3.14, \quad p > .05$
- Quality - ADHD: $\chi^2(3) = 16.6, \quad p < .01$

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[44] Dyslexia and ADHD data were all normally distributed and variances were equal.
[45] Both the upper risk level (i.e., >= 0.9 on the DEST) and the lower risk level (i.e., >= 0.6 on the DEST) were used. The findings were the same for both definitions of dyslexia risk.

The correlations showed similar results; as above, the only significant finding was between quality of programme performed and ADHD, which showed a negative relationship between the two. The results from these correlations are presented below.

- Minutes & dyslexia: r = .061 , df = 76 , p = .6
- Quality & dyslexia: r = .022, df = 76, p = .85
- Minutes & ADHD: r = -.034, df = 76, p = .775
- Quality & ADHD: r = -.404, df = 76, p = .000

9.1.4.2 Quantity

As in the McPhillips et al. (2000) study, the children were requested to perform their treatments on a daily basis for a period of 12 months (i.e., a maximum of 365 days performance). There were the expected breaks from this in the way of summer holidays, sicknesses, occasional broken limbs or family emergencies (e.g., moving house, deaths in the family). Daily treatment compliance was seldom 300+ as McPhillips et al. suggest it was in their study. Table 6 shows the mean number of days each group carried out their programme.

	Groups		
No. of days spent doing the programme	**Reflex**	**Balance**	**Placebo**
Mean	224	203	231
Standard deviation	81	96	83
Minimum	85	52	65
Maximum	364	336	350
No. / group	**39**	**38**	**19**

Table 6: The mean number of days completed per treatment group over the course of the year (n = 136 less control group).

However, classroom-based Primary Movement interventions and studies engage in 3 sets of 12 week movements over the course of a year. These are only performed on school days (i.e., 5 days of the week) and the first set of 12 weeks consist of song and

dance routines containing little in the way of reflex replication. This leaves 24 weeks of reflex replication, with compliance at 5 days a week (24 weeks x 5 days = 120 days). Given the reported academic success of programmes on the basis of this number of days (i.e., Jordan-Black, 2005), the minimum number of days compliance for the current study was set at 120 days (see Table 7). For consistency, the same criterion was set for the Balance and Placebo groups.

	Groups		
No. of days spent doing the programme	**Reflex**	**Balance**	**Placebo**
Mean	239	255	258
Standard deviation	71	58	59
Minimum	120	163	156
Maximum	364	336	350
No. >= 120 days	**35**	**27**	**16**

Table 7: The mean number of days completed per treatment group over the course of the year where the criterion was set at a minimum of 120 days (n = 118 less control group).

As a result of imposing this condition the number engaging in an acceptable level of programme compliance reduced by 4 in the reflex group, 11 in the balance group and 3 in the placebo group. The changes in gender and age per group are summarised in Table 8.

	GROUP								
	Reflex		Balance		Placebo		Control		
Gender	F	M	F	M	F	M	F	M	**Total**
Age(years)									
3	0	1	0	5	0	0	(1)	(1)	**6 (8)**
4	3	6	0	5	0	5	(1)	(13)	**19 (33)**
5	2	8	0	7	0	4	(2)	(9)	**21 (32)**
6	4	6	0	10	2	5	(1)	(10)	**27 (38)**
7	0	5	0	0	0	0	(0)	(2)	**5 (7)**
Total	**9**	**26**	**0**	**27**	**2**	**14**	**(5)**	**(35)**	**78(118)**
Mean age*	5,7		5,5		5,8		5,4		5,7
S.D. **	13		13		11		12		12.5

Table 8: Totals of children, broken down by group, age and gender when the criterion for programme compliance was set at a minimum of 120 days participation (brackets denote or include those in the control group).
* Years, months; **months

Thus, 78 children from groups 1-3 performed at least the minimum number of days of their programme. The remaining 40 were from the control group and so the full sample size was n = 118. Of the 35 children in the reflex group, the same 5 as previously mentioned (1 girl and 4 boys) participated in privately run versions of the therapy. In total, 16 of this sample were girls and 102 were boys. The age range was from 3 years, 3 months to 7 years, 4 months with a mean age of 5 years, 7 months and a standard deviation of 12.5 months. This reduction of numbers was added to the previous break-down of number loss (Figure 12) and presented in Figure 13.

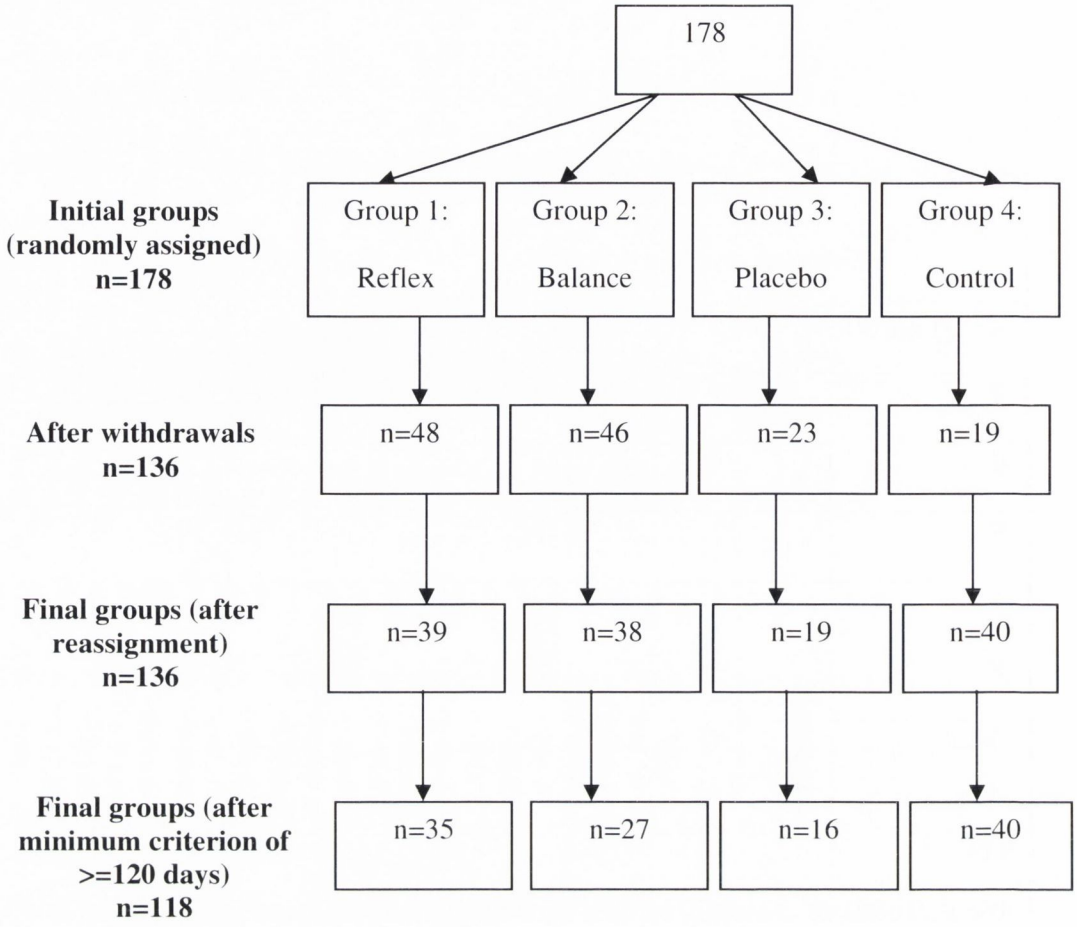

Figure 13: The formation of groups engaging in minimum programme participation criterion (i.e., a minimum of 120 days participation).

9.2 Relevant points

Two points of consideration are necessary before further analysis. These include the power of the final samples and the reliability of the scoring method.

9.2.1 Power

A strong effect size had been previously assumed by McPhillips et al. (2000) for the study on which this one is based. Although the effect size was assumed by them in advance, their outcome measures across groups justify the use of this strong effect size. Therefore, a strong effect size was also adopted here. As such, detecting a significant difference (α = .05) between groups on our outcome measures (i.e., evaluating treatment efficacy) using a one-way ANOVA (with 4 levels) required 18 children per

group (Cohen, 1992). The reflex and balance groups met this requirement easily (n = 35 & 27 respectively). The placebo group contained just under this number (n = 16 children).

In terms of predictive validity where Pearson correlations were used and the same assumptions were made, 28 children were required to detect, with 80% power, significant correlations between variables. The number of children ultimately used for predictive purposes was 98.

9.2.2 Inter-rater reliability

Observation methodology is not ideal for measuring human behaviour as there can be a large degree of subjective interpretation when scoring. There is also the possibility of missing subsequent behaviour whilst recording a specific behaviour. Such issues could render the findings unreliable. The use of video-recorded data probably helped to combat the latter of these problems because it allowed for repeat observation and scoring as well as the checking of scores. The issue of subjective interpretation was examined by using a second scorer. Another benefit of video-data is that it anchors the location of the observer to one spot thereby ensuring that scorers are measuring the same angle, limb or movement.

The second scorer was trained in the administration and scoring of the test battery and instructed to select a sample of 24 children to score. This scorer was blind to the group to which the child belonged and did not have access to the original scores.

Scattergrams of the relationship between the initial and the second scorer were examined for six comparisons. Three of these were for balance scores[46] (times 1, 2, and 3) and three for reflex scores (times 1, 2 and 3). None of the scattergrams showed any evidence of a curvilinear relationship or the undue influence of outliers and so Pearson correlations were calculated for each relationship. There was a statistically significant positive correlation in the case of all pairs of measures ($p < .01$) with those scoring poorly on tests of balance and reflex retention according to one scorer, also scoring poorly according to the second scorer (poor performance resulted in a high score and

[46] "Balance tests" and "reflex tests" should not be confused with "balance group" and "reflex group". All children performed all tests, but only some children were randomly allocated into either the balance or reflex groups.

good performance in a low score). The correlations for each comparison are set out below:

- Balance tests (time 1): $r = .914, df = 23, p < .01$
- Balance tests (time2): $r = .907, df = 23, p < .01$
- Balance tests (time 3): $r = .846, df = 23, p < .01$
- Reflex tests (time 1): $r = .917, df = 23, p < .01$
- Reflex tests (time 2): $r = .893, df = 23, p < .01$
- Reflex tests (time 3): $r = .698, df = 23, p < .01$

This represents a strong and acceptable level of inter-rater reliability and, thus, it may be assumed that the scoring method is reasonably reliable.

9.3 Inferential Statistics:

As mentioned, inferential statistics was run using MANOVA, chi-square, logistic and multiple regression. The main findings, based on difference scores, are found in Appendix 25. If interested, the reader is directed to Appendix 26 to view the mean pre-, mid- and post-scores (i.e., times 1, 2 and 3) across groups, as well as the DEST / DST and ADHD scores taken at time 3.

There were several possible confounds within the full sample although care was taken to identify these so that their effect could be taken into consideration when interpreting findings. Questions were explored using varieties of samples as necessary. These different samples included those listed below.

9.3.1 Samples[47]

- The full sample (n = 136, where 96 participants were involved in a motor programme).

- The "minimum number of days" sample where participants did at least 120 days (n = 118, where 78 were involved in a motor programme).

- Those 5 participants involved in the private versions of our therapies plus the rest of the "full sample" (n = 102, where 62 were involved in a motor programme).

- Those 5 participants involved in the private versions of our therapies plus the rest of the "minimum number of days" sample (n = 93, where 53 were involved in a motor programme).

- The full sample without the private 5 participants (n = 131, where 91 were involved in a motor programme).

- The "minimum number of days" sample without the private 5 participants (n = 113, where 73 participated in a motor programme).

Given the confounding relationship between ADHD and dyslexia when referring to motor theory (e.g., Denckla et al., 1985; Wimmer et al., 1999), it was necessary to separate this group from other samples. Therefore, the following samples were also used:

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[47] The samples are presented in this order here for easy appreciation of the difference between the samples. Clear presentation of findings, however, does not necessarily allow for samples to be presented in this linear way throughout the Results section. For example, where the "minimum number of days" sample is explored, it was thought more meaningful to follow this analysis without the "5 private" sample, then without the "ADHD" sample and then without either of these samples.

- The full sample minus those scoring at risk of ADHD (n = 105, where 76 participants were involved in a motor programme).

- The "minimum number of days" sample minus the ADHD sample where participants did at least 120 days (n = 89, where 60 were involved in a motor programme).

- The full sample without the private 5 participants and without the ADHD sample (n = 100, where 71 were involved in a motor programme).

- The "minimum number of days" sample without the private 5 participants and without the ADHD sample (n = 84, where 55 participated in a motor programme).

- Only those from the full sample who showed evidence of being at risk of ADHD (n = 31).

- Only those from the "minimum number of days sample" who showed evidence of being at risk of ADHD (n = 29).

9.3.2 Aims

The aims of the study, previously stated in chapter 6, are set out below and subsequently addressed as appropriate. The first two refer to the treatment efficacy of the programmes, the next five refer to the predictive efficacy of the tests, and the final three to other relevant issues of the study (e.g., the cerebellar deficit hypothesis).

9.3.2.1 Treatment efficacy

Aim 1

To investigate whether a programme designed to integrate retained reflexes into higher order movements reduces the subsequent probability of risk for dyslexia, as measured by the DEST / DST[48].

Aim 2

To investigate whether stimulating the vestibular system through a programme of gross motor co-ordination reduces the subsequent probability of risk for dyslexia, as measured by the DEST / DST.

9.3.2.2 Predictive efficacy

Aim 3

To investigate whether retained reflexes can discriminate between dyslexic and non-dyslexic children by predicting which children of a pre- or early-school sample will subsequently score "at risk" for dyslexia on the basis of the DEST / DST.

Aim 4

To investigate whether vestibular functioning, as measured by a battery of gross motor co-ordination tests (i.e., balance), can discriminate between dyslexic and non-dyslexic children by predicting which children of a pre- or early-school sample will subsequently score at risk for dyslexia on the basis of the DEST / DST.

Aim 5

To test the cerebellar deficit hypothesis (Nicolson and Fawcett, 1990) of dyslexia by investigating whether fine-motor skills can discriminate between dyslexic and non-dyslexic children by predicting which children of a pre- or early-school sample will subsequently score at risk for dyslexia on the basis of the DEST / DST.

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[48] Dyslexia Early Screening Test – Second Edition / Dyslexia Screening Test (Nicolson and Fawcett, 2004; Fawcett and Nicolson, 1996).

Aim 6

To investigate whether visuo-motor skills can discriminate between dyslexic and non-dyslexic children by predicting which children of a pre- or early-school sample will subsequently score at risk for dyslexia on the basis of the DEST / DST.

Aim 7

To investigate whether phonological skills can discriminate between dyslexic and non-dyslexic children by predicting which children of a pre- or early-school sample will subsequently score at risk for dyslexia on the basis of the DEST / DST.

9.3.2.3 Final aim

Aim 8

To investigate whether tests of phonological skills are related to performance on cerebellar tests in order to test this prediction from Liberman and Mattingly's (1985) motor theory of speech.

9.3.3 Treatment Efficacy (Aims 1 & 2): Statistical Analysis

Eight dependent variables were used in total. It was seen in chapter 6 that there is one major dependent variable, and this is the DEST / DST score (i.e., the measure of dyslexia risk). There were 5 minor dependent variables which consisted of: retained reflexes, balance skills, visuo-motor skills, fine-motor skills and phonological skills. The scores on these minor dependent variables were actually difference scores, calculated by summing the tests of each measure at time 1 and time 3 and subtracting those summed scores of time 3 from time 1. The uncontrolled variable from the "Conners' Parent Rating Scale – Revised" was a measure for ADHD. Thus, in total there are seven dependent variables. The design of the study is reproduced in figure 14 from chapter 6 (i.e., Figure 10).

In exploring the relationship between treatment group and outcome variable (i.e., dependent variable), data were analysed by means of chi-squares and MANOVAs (including the relevant ANOVAs and post-hoc tests). It was explained in Chapter 8 that

the cut-off point for (strong) "dyslexia risk" was 0.9 on the DEST (with 0.6 - 0.8 representing "mild risk") and at 1.00 on the DST[49]. Chi-square tests, where the independent variable was "group" and the dependent variable was "risk" (i.e., for dyslexia) were run twice. The first included "risk" where the separation between "at risk" and "not at risk" was the upper level of 0.9 on the DEST and, thus, represented "strong risk". The second chi-square used the milder separation between "at risk" and "not at risk" where 0.6 represented the "mild risk" on the DEST. The separation was always 1.00 on the DST.

For the MANOVAs, the assumptions of normal-distribution and homogeneity of variance were checked for all samples' dependent variables. For the most part, the assumption of variance homogeneity was not violated and Bonferonni tests were checked. However, where homogeneity was questionable (i.e., Levene's test was significant at the $p < .05$ level) Games-Howell post-hoc tests were used when considering significant differences at a fine level (it is indicated when this is the case). The assumption of normal-distribution was met for many, though not all, of the dependent variables. Because there is no non-parametric alternative to a MANOVA in SPSS-12 it was not possible to adopt an alternative method of analysis and so a MANOVA was employed despite these cases.

These procedures were performed for the main sample, with relevant confounds from this sample removed to eliminate their effects and the analysis then repeated on subsequent samples as necessary. The reader is alerted to the repetitive nature of this linear presentation and is made aware from the outset that few significant results were revealed. In this case, "group" was used as the independent variable and the eight dependent variables were those described above.

The extent of further investigative analyses depended upon what was revealed from the MANOVA. Neither the DEST / DST measure nor the Conners' ADHD measure were taken at testing session 2 (i.e., after 6 months) and so the effect of Group x Time cannot be explored for these variables. The remaining dependent variables were all measured at time 2 although they did not vary as a function of group when subject to a MANOVA (Time 3 – Time 1) and so were not considered worth exploring over the initial 6 months (i.e., Time 2 – Time 1).

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[49] The 2nd edition of the Dyslexia Screening Test (DST-2) also defines 0.6 as a cut-off for "mild risk", however, this was not specified in the first edition of the test which was the edition used in the current study.

To explore the predictive efficacy of these tests with regard to reading outcome only (as opposed to the full DEST / DST score), a non-parametric Kruskall-Wallis, with associated Mann-Whitney-U tests, was performed.

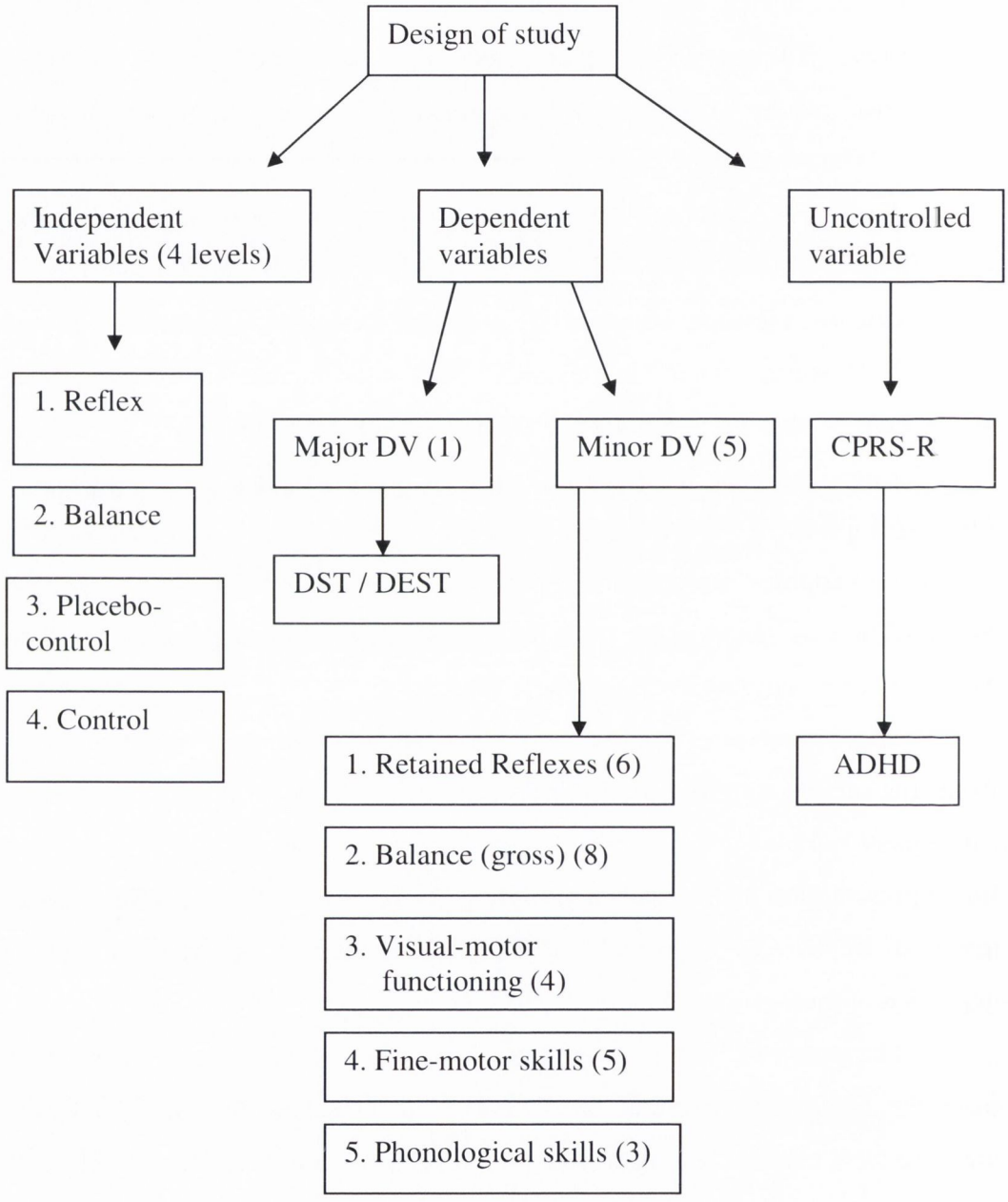

Figure 14: Design of the study.

9.3.3.1 Minimum days samples

9.3.3.1.1 Select sample (n = 118)

Based on application of minimum number of days criterion.

Chi-square analysis revealed that there was a significant association between "group" and "risk of dyslexia" (when the "high risk" criterion of 0.9 was used for the DEST score) ($\chi^2(3) = 10.07$, $p = .018$). Further analysis showed that those in the balance group were 2.4 times as likely to be at risk for dyslexia as those in the reflex group ($\chi^2(1) = 6.98$, $p = .008$) and 2.5 times as likely to be at risk as the control group ($\chi^2(1) = 7.66$, $p = .006$). When the "mild risk" category of the DEST was taken into consideration (i.e., when the "mild risk" criterion of 0.6-0.8 was used for the DEST score) this significant association was lost, although the trend remained ($\chi^2(3) = 7.39$, $p = 0.06$). A chi-square matrix depicting the proportions of those at risk / not at risk of dyslexia, per group is shown in Table 9.

	At risk (n)	*Not at risk (n)*	**Total**
Group			
Reflex	8	27	**35**
Balance	15	12	**27**
Placebo	6	10	**16**
Control	9	31	**40**
Total	**38**	**80**	**118**

Table 9: Breakdown of numbers at risk / not at risk for dyslexia, per group (n = 118).

One MANOVA test statistic (Roy's Largest Root) was significant ($F(10, 107) = 2.29$, $p = .018$). This suggested that the outcome measures varied as a function of group, however, it did not indicate between which groups such difference(s) might lie, or on which DVs. Further analysis, using univariate ANOVAs were conducted on each dependent variable, to reveal where these differences lay (this practice was adopted throughout the analyses). This showed only an effect of group on the dependent variable of "dyslexia risk", as measured by the DEST / DST ($F(3, 114) = 3.50$, $p = .018$).

Post-hoc t-tests, with Bonferroni correction, reflected the chi-square results. These showed differences between the reflex group and balance group, and between the balance group and control group. The mean DEST / DST score for the balance

group ($\bar{x}$ = 0.98, SD = 0.56) was significantly higher (t = 2.9, df = 60 , two-tailed, p = .027) than the mean for the reflex group ($\bar{x}$ = 0.62, SD = 0.39) and significantly higher (t = 2.7 , df = 65 , two-tailed, p = .044) than the mean of the control group ($\bar{x}$ = 0.65, SD = 0.44). In fact, this mean represented a score which, for the DEST-aged children (i.e., 4.5 years – 6.5 years), was over the threshold for dyslexia risk, and for the DST-aged children (>6.5 years) was as good as at the threshold level (i.e., 0.98 versus 1.00). The means and standard deviations of the four groups are presented in Table 10.

Group	DEST / DST	
	Mean	SD
Reflex	0.62	0.39
Balance	0.98*	0.56
Placebo	0.82	0.63
Control	0.65	0.44

Table 10: The DEST / DST means and standard deviations of each group at the end of the 12 month intervention (n = 118).
*Balance group is significantly higher than both Reflex and Control groups (p < .05)

An independent samples t-test was run to explore whether the DEST / DST means of those who had moved from the balance group to the control group were significantly lower than those who remained in the balance group. If this was so it would suggest that a sample of particularly high DEST / DST scorers might have made up this group. Because the variances for the two groups were significantly unequal (F = 4.44, p < .05), a t-test for unequal variances was used. This shows that, although the means were lower for those who left the Balance group (M = 0.61, SD = 0.38) than those who remained in it (M = 0.89, SD = 0.55), they were not significantly lower (t = -1.7, df = 14, p = 0.107, 2-tailed).

9.3.3.1.2 Select sample (n = 113)

Based on application of minimum number of days criterion & removal of "private" children.

To explore whether these findings had been influenced unduly by the inclusion of those who were engaged in privately run versions of the experimental therapies, those 5 children were excluded from the sample and the analyses run again. This meant that the only group number to change was the Reflex Group which was reduced from n = 35 to n = 30. The other groups remained Balance 27, Placebo 16 and Control 40. Thus, 73 children from groups 1-3 performed at least the minimum number of days of their programme as instructed by our study. The remaining 40 were from the control group and so in terms of treatment efficacy, this sample size was n = 113. In total, 15 of this sample were girls and 98 were boys. The age range was from 3 years, 3 months to 7 years, 4 months with a mean age of 5 years, 5 months and a standard deviation of 12.4 months. The number breakdown is presented in Table 11.

	GROUP								
	Reflex		Balance		Placebo		Control		
Gender	F	M	F	M	F	M	F	M	**Total**
Age(years)									
3	0	1	0	5	0	0	(1)	(1)	**6 (8)**
4	3	5	0	5	0	5	(1)	(13)	**18 (32)**
5	1	8	0	7	0	4	(2)	(9)	**20 (31)**
6	4	3	0	10	2	5	(1)	(10)	**24 (35)**
7	0	5	0	0	0	0	(0)	(2)	**5 (7)**
Total	**8**	**22**	**0**	**27**	**2**	**14**	**(5)**	**(35)**	**73 (113)**
Mean age*	5,7		5,5		5,8		5,4		5,5
S.D. **	13		13		11		12		12.4

Table 11: Totals of children, broken down by group, age and gender where the criterion for programme compliance was set at a minimum of 120 days and "private" children were removed (brackets denote or include those in the Control Group) (n = 113).
* Years, months; **months

A chi-square showed a significant association between "group" and "risk of dyslexia" (i.e., "strong risk" as measured by DEST / DST) ($\chi^2(3) = 8.79$, $p = 0.032$, 2-tailed). The children who participated in the balance programme were 2.1 times more likely to score at high risk of dyslexia as children in the reflex programme ($\chi^2(1) = 4.93$, $p = .026$, 2-tailed) and 2.5 times as likely to score at risk as those in the control group ($\chi^2(1) = 4.34$, $p = .037$, 2-tailed). When the "mild risk" category of the DEST was taken into consideration this significant association was lost, although the trend remained ($\chi^2(3) = 7.39$, $p = .06$). A chi-square matrix depicting the proportions of those at risk / not at risk of dyslexia, per group is shown in Table 12.

	At risk (n)	*Not at risk (n)*	**Total**
Group			
Reflex	8	22	**30**
Balance	15	12	**27**
Placebo	6	10	**16**
Control	9	31	**40**
Total	**38**	**75**	**113**

Table 12: Breakdown of numbers at risk / not at risk for dyslexia, per group (n = 113).

Roy's largest root was found to be significant ($F(10, 102) = 2.28$, $p = .019$) and consequent univariate ANOVAs on each dependent variable showed an effect of group on the dependent variable of "dyslexia risk", as measured by the DEST / DST ($F(3, 109) = 3.009$, $p = .033$).

Post-hoc t-tests, with Bonferroni correction, showed that these differences lay between the balance group and control group. The mean DEST / DST score for the balance group ($\bar{x} = 0.98$, $SD = 0.56$) was significantly higher ($t = 2.7$, $df = 65$, two-tailed $p = .047$) than the mean for the control group ($\bar{x} = 0.65$, $SD = 0.44$). The severity of this mean for the balance group remained as it did when these 5 children were included in analysis (i.e., very high risk for dyslexia). Significant differences between the balance group and the reflex group on DEST / DST performance were lost with the removal of these children, when the reflex group increased by 0.4 points from .62 to .66 (see Table 13 for means and standard deviations).

Group	DEST / DST	
	Mean	SD
Reflex	0.66	0.39
Balance	0.98*	0.56
Placebo	0.82	0.64
Control	0.65	0.44

Table 13: The DEST / DST means and standard deviations of each group at the end of the 12 month intervention where the private 5 individuals were removed from the reflex group (n = 113).
*Balance group is significantly higher than the control group ($p < .05$)

9.3.3.1.3 Select sample (n = 88)

Based on application of minimum number of days criterion where the reflex group were composed only of those in the "private" therapy.

The disappearance of the significant difference between the reflex and balance group on the DEST / DST score when these 5 children were removed warranted investigation because of its suggestion that these 5 children contributed to a lowering of mean DEST / DST scores when included in the sample. Thus, a MANOVA was rerun with the reflex group composed of just these children (i.e., n = 5 for reflex group; n = 88 for full sample). Although initially it appeared that there was an effect on DEST / DST scores (Roy's largest root, $F_{(10, 77)} = 2.04$, $p = .04$) and this was supported by the ANOVA ($F_{(3, 84)} = 3.153$, $p = .029$), further post-hoc t-tests did not show any significant differences between groups despite the apparent DEST / DST mean differences initially ($p > .05$ for all Bonferroni adjusted comparisons) although the balance and control group difference approached significance ($p = .07$). Given the striking reduction of the mean on the DEST / DST for this modified reflex group ($\bar{x} = 0.392$ which represents a "low risk" score) it would seem that the sample size is just too small to detect any difference that might be there.

A chi-square could not be performed on this sample because the expected counts of more than 2 cells were less than 5.

Group	DEST / DST	
	Mean	SD
Reflex	0.39	0.37
Balance	0.98	0.56
Placebo	0.82	0.63
Control	0.65	0.44

Table 14: The DEST / DST means and standard deviations of each group where the reflex group contained only the 5 private individuals.

9.3.3.1.4 Select sample (n = 89)

Based on application of minimum number of days criterion & removal of ADHD children.

The next analysis was composed of those who had conformed to the 120 days criterion minus those who had scored above the "risk" threshold for ADHD. For this sample n = 89. There were 28 in the Reflex group, 22 in the Balance group, 10 in the Placebo group and 29 in the Control group. Thus, the distribution of participants who scored above the "risk" threshold for ADHD amongst each of the four conditions is as presented in table 15.

Group			
Reflex	Balance	Placebo	Control
7	5	6	11

Table 15: The numbers of ADHD children per group (from the sample of n = 118).

Sixty children from groups 1-3 performed at least the minimum number of days of their programme as instructed by our study and did not score at risk for ADHD. The

remaining 29 were from the control group, and so in terms of treatment efficacy, this sample size was n = 89. In total, 10 of this sample were girls and 79 were boys. The age range was from 3 years, 3 months to 7 years, 4 months with a mean age of 5 years, 5 months and a standard deviation of 12.5 months. The number breakdown is presented in Table 16.

		GROUP								
		Reflex		Balance		Placebo		Control		
Gender		F	M	F	M	F	M	F	M	**Total**
Age(years)										
3		0	1	0	5	0	0	(1)	(1)	**6 (8)**
4		2	5	0	4	0	3	(0)	(11)	**14 (25)**
5		1	7	0	6	0	3	(1)	(6)	**17 (24)**
6		3	5	0	7	1	3	(1)	(7)	**19 (27)**
7		0	4	0	0	0	0	(0)	(1)	**4 (5)**
Total		**6**	**22**	**0**	**22**	**1**	**9**	**(3)**	**(26)**	**60 (89)**
Mean age*		5,7		5,3		5,8		5,3		5,5
S.D. **		13		13.5		11		12		12.7

Table 16: Totals of children, broken down by group, age and gender where the criterion for programme compliance was set at a minimum of 120 days and ADHD children have been removed (brackets denote or include those in the control group).
* Years, months; **months

A chi-square analysis showed a significant association between "group" and "risk of dyslexia" (i.e., "strong risk") ($\chi^2(3) = 9.207$, $p = .027$). The children who participated in the balance group were 4.25 times more likely to score at high risk of dyslexia as children in the reflex group ($\chi^2(1) = 7.73$, $p = .005$). When the "mild risk" category of the DEST was taken into consideration this significant association was lost, although the trend remained ($\chi^2(3) = 7.44$, $p = .06$). A chi-square matrix depicting the proportions of those at risk / not at risk of dyslexia, per group is shown in Table 17.

	At risk (n)	*Not at risk (n)*	**Total**
Group			
Reflex	3	25	**28**
Balance	10	12	**22**
Placebo	4	6	**10**
Control	6	23	**29**
Total	**23**	**66**	**89**

Table 17: Breakdown of numbers at risk / not at risk for dyslexia, per group.

Roy's largest root showed a significant difference amongst the dependent variables ($F_{(10, 76)} = 2.043$, $p = .040$) and the univariate ANOVA supported this finding suggesting that differences were amongst the DEST / DST scores ($F_{(3, 85)} = 3.119$, $p = .03$). Levene's test of homogeneity of variance for this D.V. showed $p < .05$[50] and so the Games-Howell t-statistic was used to locate this difference. This did not reveal statistically significant differences between any of the groups except the balance and reflex group which just met significance ($t = 2.6$, $df = 48$, two-tailed $p = .05$). The means of this sample are presented in Table 18.

[50] Transformations of the data made no difference. This was also the case for subsequent cases where Levene's $p < .05$.

Group	DEST / DST	
	Mean	SD
Reflex	0.53	0.36
Balance	0.92	0.60
Placebo	0.75	0.53
Control	0.61	0.43

Table 18: The DEST / DST means and standard deviations of each group where those with ADHD have been removed from the number who performed the minimum number of expected days (i.e., >=120 days).

9.3.3.1.5 Select sample (n = 29)

Based on application of minimum number of days criterion & ADHD children only.

The removal of the ADHD sample from the larger sample reduced the DEST / DST mean of each group, resulting in a loss of the significant difference between the balance and control groups. Although it represented a small sample size (n = 29) the ADHD "at risk" sample was selected for special analysis to determine if this group showed any evidence of group differences. Initial significant DEST / DST effects from the MANOVA suggested this might be the case (Roy's largest root, $F_{(10, 18)} = 3.420$, $p = .01$), however, this was not supported by the ANOVA ($F_{(3, 25)} = 1.057$, $p = .385$).

A chi-square could not be performed on this sample because the expected counts of more than 2 cells were less than 5.

9.3.3.1.6 Select sample (n = 84)

Based on application of minimum number of days criterion & removal of both "private" and ADHD children.

A further sample was examined of those who had conformed to the 120 days criterion minus those who had scored above the "risk" threshold for ADHD and minus those who had engaged in private versions of the our therapy. Those now in the Reflex group numbered 23, in the Balance group numbered 22, in the Placebo group 10 and in the Control group 29. Thus, 55 children from groups 1-3 performed at least the minimum number of days of their programme as instructed by our study, did not score at risk for ADHD and were not involved in external programmes. The remaining 29 were from the control group and so in terms of treatment efficacy, this sample size was n = 84 (9 girls, 75 boys). Their ages range from 3 years, 3 months to 7 years, 4 months with a mean age of 5 years, 5 months and a standard deviation of 12.5 months. The number breakdown is presented in Table 19.

	GROUP								
	Reflex		*Balance*		*Placebo*		*Control*		
Gender	*F*	*M*	*F*	*M*	*F*	*M*	*F*	*M*	**Total**
Age(years)									
3	0	1	0	5	0	0	(1)	(1)	**6 (8)**
4	2	4	0	4	0	3	(0)	(11)	**13 (24)**
5	0	7	0	6	0	3	(1)	(6)	**16 (23)**
6	3	2	0	7	1	3	(1)	(7)	**16 (24)**
7	0	4	0	0	0	0	(0)	(1)	**4 (5)**
Total	**5**	**18**	**0**	**22**	**1**	**9**	**(3)**	**(26)**	**55 (84)**
Mean age*	5,7		5,3		5,8		5,3		5,5
S.D. **	13		13.5		11		12		12.6

Table 19: Totals of children, broken down by group, age and gender, where the criterion for programme compliance was set at a minimum of 120 days, ADHD children were removed and private children were removed (brackets denote or include those in the control group).
* Years, months; **months

A chi-square failed to show a significant association between "group" and "risk of dyslexia" (i.e., "strong risk") ($\chi^2(3) = 7.446$, $p = .059$). This finding remained when the "mild risk" category of the DEST was taken into consideration ($\chi^2(3) = 5.326$, $p =$

.149). A chi-square matrix depicting the proportions of those at risk / not at risk of dyslexia, per group is shown in Table 20.

	At risk (n)	*Not at risk (n)*	**Total**
Group			
Reflex	3	20	**23**
Balance	10	12	**22**
Placebo	4	6	**10**
Control	6	23	**29**
Total	**23**	**61**	**84**

Table 20: Breakdown of numbers at risk / not at risk for dyslexia, per group (n = 84).

Roy's largest root showed a significant difference amongst the dependent variables ($F_{(10, 76)} = 2.043$, $p = .04$) and the univariate ANOVA supported this finding suggesting that differences were amongst the DEST / DST scores ($F_{(3, 73)} = 1.976$, $p = .048$). Levene's test of homogeneity of variance for this DV showed $p < .05$ and so the Games-Howell t-statistic was used to locate this difference. This did not reveal statistically significant differences between any of the groups. The means and standard deviations of the groups DEST / DST scores are shown in Table 21.

Group	DEST / DST	
	Mean	SD
Reflex	0.56	0.36
Balance	0.92	0.60
Placebo	0.75	0.53
Control	0.61	0.43

Table 21: The DEST / DST means and standard deviations of each group where those who engaged in the private therapies and those with ADHD were removed from those who performed the minimum number of expected days (i.e., >=120 days) (n = 84).

9.3.3.2 Full Samples

9.3.3.2.1 Select sample (n = 136)

Based on full number of participants regardless of number of programme days done

The next set of analyses consists of the same set of considerations as above except the *entire* original sample (i.e., n = 136) were included for initial analysis. The diagram representing the formation of this group and the table conveying the gender and age details have already been presented and shall not be reproduced (Figure 12 and Table 5 respectively).

There was no significant association between "group" and "risk of dyslexia" (i.e., "strong risk") in the chi-square analysis ($\chi^2(3) = 7.075$, $p = .07$) though the levels appeared to be approaching significance. This finding remained when the "mild risk" category of the DEST was taken into consideration ($\chi^2(3) = 5.226$, $p = .156$). A chi-square matrix depicting the proportions of those at risk / not at risk of dyslexia, per group is shown in Table 22.

	At risk (n)	***Not at risk (n)***	**Total**
Group			
Reflex	8	31	**39**
Balance	17	21	**38**
Placebo	7	12	**19**
Control	9	31	**40**
Total	**41**	**95**	**136**

Table 22: Breakdown of numbers at risk / not at risk for dyslexia, per group (n = 136).

From the MANOVA, Roy's largest root was significant ($F(10, 125) = 2.48$, $p = .01$). As before, univariate ANOVAs indicated that this difference lay in the dependent variable of DEST / DST ($F(3, 132) = 2.992$, $p = .033$).

Post-hoc t-tests, using Games-Howell (Levene's test was $p < .05$), showed that these differences lay between the reflex group and balance group. The mean DEST / DST score for the balance group ($\bar{x} = 0.88$, $SD = 0.55$) was significantly higher ($t =$

2.7, df =75 , two-tailed p = .034) than the mean for the reflex group ($\bar{x}$ = 0.58, SD = 0.38). These means and standard deviations are presented in Table 23.

Group	DEST / DST	
	Mean	SD
Reflex	0.58	0.38
Balance	0.88*	0.55
Placebo	0.81	0.61
Control	0.65	0.44

Table 23: The DEST / DST means and standard deviations of each group where the full set of participants were included in analysis (i.e., n = 136).
*Balance group differs significantly from the reflex group mean ($p < .05$).

9.3.3.2.2 Select sample (n = 131)

Based on full sample & removal of "private" children.

As before, those 5 children who had participated in private versions of the therapy were excluded and the analysis was re-run. This meant that the only group number to change was the reflex group which was reduced from n = 39 to n = 34. The other groups remained 38 in the Balance group, 19 in the Placebo group and 40 in the Control group. Of this sample, 18 of this sample were girls and 113 were boys (n = 131). The age range was from 3 years, 3 months to 7 years, 4 months with a mean age of 5 years, 4 months and a standard deviation of 12.4 months. The number breakdown is presented in Table 24.

	GROUP								
	Reflex		Balance		Placebo		Control		
Gender	F	M	F	M	F	M	F	M	**Total**
Age(years)									
3	0	1	0	7	0	1	(1)	(1)	**9 (11)**
4	3	5	1	8	0	6	(1)	(13)	**23 (37)**
5	2	9	0	11	0	4	(2)	(9)	**26 (37)**
6	5	4	0	11	2	6	(1)	(10)	**28 (39)**
7	0	5	0	0	0	0	(0)	(2)	**5 (7)**
Total	**10**	**24**	**1**	**37**	**2**	**17**	**(5)**	**(35)**	**91 (131)**
Mean age*	5,7		5,3		5,7		5,4		5,5
S.D. **	12.5		13		12		12		12.4

Table 24: Totals of children, broken down by group, age and gender where all children were included in the analysis except those who engaged in private versions of the therapy (n = 131).
* Years, months; **months

A chi-square showed no evidence of a significant association between "group" and "risk of dyslexia" (i.e., "strong risk") ($\chi^2(3) = 5.86$, $p = .119$). This finding also applied when the "dyslexia risk" was lowered to include those at mild risk of dyslexia ($\chi^2(3) = 3.78$, $p = .286$). The means and standard deviations are presented in Table 26. A chi-square matrix depicting the proportions of those at risk / not at risk of dyslexia, per group is shown in Table 25.

	At risk (n)	*Not at risk (n)*	**Total**
Group			
Reflex	8	26	**34**
Balance	17	21	**38**
Placebo	7	12	**19**
Control	9	31	**40**
Total	**41**	**90**	**131**

Table 25: Breakdown of numbers at risk / not at risk for dyslexia, per group (n = 131).

Roy's largest root was found to be significant ($F_{(10, 120)} = 2.395$, $p = .013$) but subsequent univariate ANOVAs failed to identify differences in any of the dependent variables ($F_{(3, 127)} = 2.472$, $p = .065$) (balance and reflex group means on DEST / DST measures were $\bar{x} = 0.88$ and $\bar{x} = 0.61$ consecutively).

Group	DEST / DST	
	Mean	SD
Reflex	0.61	0.38
Balance	0.88	0.55
Placebo	0.81	0.61
Control	0.65	0.44

Table 26: The DEST / DST means and standard deviations of each group where the full set of participants, minus those in private therapies, were included in the analysis (i.e., n = 131).

9.3.3.2.3 Select sample (n = 102)

Based on full sample where the reflex group were composed only of those in the "private" therapy.

As before, the disappearance of the significant difference between the reflex and balance group on the DEST / DST score when these 5 children were removed was investigated because of its suggestion that these 5 children contributed to a lowering of mean DEST / DST scores. Thus, a MANOVA was rerun with the reflex group composed of just these children (i.e., n = 5 for reflex Group; n = 102 for full sample).

Again, it appeared initially that there were differences between the groups (Roy's largest root, $F_{(9, 92)} = 2.15$, $p = .033$), however, this was not supported by the ANOVAs. The DEST / DST scores were, again, the only dependent variable which approached significance ($F_{(3, 98)} = 2.27$, $p = .086$).

Given the striking reduction of the mean on the DEST / DST for this modified reflex group ($\bar{x} = 0.39$ which represents a "low risk" score, compared to $\bar{x} = 0.88$ for the balance group which is approaching "high risk") it would seem that, as before, the sample size is just too small to detect any difference that might be there.

A chi-square could not be performed on this sample because the expected counts of more than 2 cells were less than 5.

Group	DEST / DST	
	Mean	SD
Reflex	0.39	0.38
Balance	0.88	0.55
Placebo	0.81	0.61
Control	0.65	0.44

Table 27: The DEST / DST means and standard deviations of each group where the reflex group consisted of only those in the privately-run therapies (n = 102).

9.3.3.2.4 Select sample (n = 105)

Based on application of minimum number of days criterion & removal of ADHD children.

Those who had scored above the "risk" threshold for ADHD were then removed from the sample. Here the final groups consisted of 32 in the Reflex group, 31 in the Balance group, 13 in the Placebo group and 29 in the Control group. Thus, the distribution of participants who scored above the "risk" threshold for ADHD amongst each of the four conditions is as presented in table 28.

Group			
Reflex	Balance	Placebo	Control
7	7	6	11

Table 28: The numbers of ADHD children per group (from the sample of n = 136).

Of this sample, 13 were girls and 92 were boys (n = 105). The age range was from 3 years, 3 months to 7 years, 4 months with a mean age of 5 years, 5 months and a standard deviation of 12.7 months. The breakdown in numbers are presented in Table 29.

	GROUP								
	Reflex		Balance		Placebo		Control		
Gender	F	M	F	M	F	M	F	M	**Total**
Age(years)									
3	0	1	0	7	0	1	(1)	(1)	**9 (11)**
4	2	5	1	6	0	4	(0)	(11)	**18 (29)**
5	2	8	0	10	0	3	(1)	(6)	**23 (30)**
6	4	6	0	7	1	4	(1)	(7)	**22 (30)**
7	0	4	0	0	0	0	(0)	(1)	**4 (5)**
Total	**8**	**24**	**1**	**30**	**1**	**12**	**(3)**	**(26)**	**76 (105)**
Mean age*	5,7		5,1		5,6		5,3		5,4
S.D. **	12.6		13		12		12		12.7

Table 29: Totals of children, broken down by group, age and gender where all participants, except those who scored at risk for ADHD, were considered (n = 105).
* Years, months; **months

A chi-square showed evidence of a significant association between "group" and "risk of dyslexia" (i.e., "strong risk") ($\chi^2(3) = 7.7$, $p = .05$). Further analysis showed a significant difference between the reflex and balance group ($\chi^2(1) = 6.2$, $p = .013$) such that those in the balance group 3.8 times more likely to be at risk of dyslexia than those in the reflex group. This finding was lost when the "dyslexia risk" was lowered to include those at mild risk of dyslexia ($\chi^2(3) = 6.76$ $p = .087$). The means and standard deviations of the DEST / DST scores are presented in Table 31, where the balance group $\bar{x} = 0.81$ and the reflex $\bar{x} = 0.50$. A chi-square matrix depicting the proportions of those at risk / not at risk of dyslexia, per group is shown in Table 30.

	At risk (n)	*Not at risk (n)*	**Total**
Group			
Reflex	3	29	**32**
Balance	11	20	**31**
Placebo	5	8	**13**
Control	6	23	**29**
Total	**25**	**80**	**105**

Table 30: Breakdown of numbers at risk / not at risk for dyslexia, per group (n = 105).

Roy's largest root showed a significant difference amongst the dependent variables ($F(9, 95) = 2.57$, $p = .01$). This was supported by the univariate ANOVA showing a significant different in the DEST / DST measure ($F(3, 101) = 2.62$, $p = .05$). Post-hoc tests showed the reflex and balance groups to be approaching a significant difference ($t = 2.6$, $df = 61$, two-tailed $p = .058$).

Group	DEST / DST	
	Mean	SD
Reflex	0.5	0.36
Balance	0.81	0.57
Placebo	0.76	0.52
Control	0.61	0.43

Table 31: The DEST / DST means and standard deviations of each group where the ADHD individuals were removed from the sample (n = 105).

9.3.3.2.5 Select sample (n = 31)

Based on the full sample's ADHD children only.

The removal of the ADHD sample from the larger sample reduced the DEST / DST mean of each group and resulted in a loss of the significant difference between the balance / reflex groups. Although it represented a small sample size (n = 31) the ADHD "at risk" sample was selected for special analysis to determine if this group showed any evidence of group differences. The DEST / DST scores of this group were much higher for each group than when the entire sample means were considered (balance group $\bar{x}$ = 1.22 and reflex group $\bar{x}$ = 0.96, compared to placebo control group mean = 0.93 and control group mean = 0.75; see Table 32). The MANOVA (Roy's largest root, $F_{(9, 21)} = 2.82$, p = .024) suggested initial group differences, however, this was not supported by the ANOVA ($F_{(3, 27)} = 1.2$, p = .329).

A chi-square could not be performed on this sample because the expected counts of more than 2 cells were less than 5.

Group	DEST / DST	
	Mean	SD
Reflex	0.96	0.28
Balance	1.22	0.34
Placebo	0.93	0.82
Control	0.75	0.51

Table 32: The DEST / DST means and standard deviations of each group where only the ADHD individuals were included (n = 31).

9.3.3.2.6 Select sample (n = 100)

Based on the full sample of participants minus those doing "private" therapies and minus those scoring at risk of ADHD.

The final sample was composed of all those who had participated in the study, minus those who scored above the "risk" threshold for ADHD and minus those who had engaged in private versions of the therapy. There were 27 in the Reflex group, 31 in the Balance group, 13 in the Placebo group and 29 in the Control group. Within this sample, 12 were girls and 88 were boys (n = 100). Their ages range from 3 years, 3 months to 7 years, 4 months with a mean age of 5 years, 4 months and a standard deviation of 12.6 months. This breakdown of numbers is presented in Table 33.

	GROUP								
	Reflex		*Balance*		*Placebo*		*Control*		
Gender	*F*	*M*	*F*	*M*	*F*	*M*	*F*	*M*	**Total**
Age(years)									
3	0	1	0	7	0	1	(1)	(1)	**9 (11)**
4	2	4	1	6	0	4	(0)	(11)	**17 (28)**
5	1	8	0	10	0	3	(1)	(6)	**22 (29)**
6	4	3	0	7	1	4	(1)	(7)	**19 (27)**
7	0	4	0	0	0	0	(0)	(1)	**4 (5)**
Total	**7**	**20**	**1**	**30**	**1**	**12**	**(3)**	**(26)**	**71 (100)**
Mean age*	5,7		5,1		5,6		5,3		5,4
S.D. **	12.6		13		12		12		12.6

Table 33: Totals of children, broken down by group, age and gender where those with ADHD and those engaging in private therapies were taken from the full, initial sample (n = 100).
* Years, months; **months

A chi-square failed to find any evidence of a significant association between "group" and "risk of dyslexia" (i.e., "strong risk" a ($\chi^2(3) = 6.14$, $p = .105$). This finding also applied when the "dyslexia risk" was lowered to include those at mild risk of dyslexia ($\chi^2(3) = 4.61$ $p = .202$). A chi-square matrix depicting the proportions of those at risk / not at risk of dyslexia, per group is shown in Table 34.

	At risk (n)	*Not at risk (n)*	**Total**
Group			
Reflex	3	24	**27**
Balance	11	20	**31**
Placebo	5	8	**13**
Control	6	23	**29**
Total	**25**	**75**	**100**

Table 34: Breakdown of numbers at risk / not at risk for dyslexia, per group (n = 100).

Roy's largest root showed evidence of a significant difference amongst the groups on the dependent variables ($F_{(9, 90)} = 2.48$, $p = .014$). Although there still appears to be a large difference between the balance and reflex groups on the measure of DEST / DST (balance $\bar{x} = 0.81$ and reflex $\bar{x} = 0.52$; see Table 35) it was not detected by the univariate ANOVAs when subject to further investigation.

Group	DEST / DST	
	Mean	SD
Reflex	0.52	0.36
Balance	0.81	0.57
Placebo	0.76	0.52
Control	0.61	0.43

Table 35: The DEST / DST means and standard deviations of each group where the ADHD individuals and those engaged in private versions of the reflex therapy were removed from the initial sample (n = 100).

9.3.3.3 Reading as outcome measure

Kruskall-Wallis / Mann-Whitney-U

In their study assessing the efficacy of the Dores Balance programme (i.e., DDAT), Reynolds et al. (2005) found that those with high initial DST scores benefited most from the therapy in terms of reading; they also reported a significant group by time interaction, in favour of the DDAT group, on the reading score of the DST ($p < .05$). Therefore, reading scores (taken from the DST) were also used as the outcome measure to explore whether either experimental group performed any better on tests of reading[51] than those children in the control groups.

Only one analysis was run using reading score as the outcome measure. Because only 75 children, of whom 65 had completed at least 120 days of their programme, were old enough to be tested on the DST (and, thus, the reading test) the sample was already quite small (see Table 36 for breakdown of age and genders). For this reason the analysis was not repeated after removing private children and ADHD children but was only performed once using the maximum number of candidates possible (i.e., n = 65 where at least 120 days of the intervention programme had been completed).

	Gender		
	Male	**Female**	
Age			**Total**
6	19	3	**22**
7	29	7	**36**
8	7	0	**7**
Total	**55**	**10**	**65**

Table 36: Breakdown of age and gender for n = 65 exploring motor programme effect on reading skill (as measured by the DST). (Children's ages are based on ages at end of study in order to avoid confusion regarding the use of DST for those over 6.5 years).

[51] This was the only reading test used in the current study due to time limitations. This was the test used because Reynolds et al. (2003) made use of it in their study. However, this is a test of both reading speed and accuracy so future studies might like to differentiate between the two. It should also be bourne in mind that generally speaking the subtests of the DEST or DST are not meant to be used in isolation as they contribute to a cumulative score.

The children's reading ability was classified into ranges based on percentiles; 1-4th percentile; 5-11th percentile; 12-24th percentile; 25-74th percentile and >75th percentile. Because of this, and because the data were not sufficiently normally-distributed, the non-parametric Kruskal-Wallis test was used instead of an ANOVA. This showed initial evidence for significant differences amongst the groups on reading ($H_{(3)} = 13.6$, $p = .003$) which appeared to lie with the balance group (see Table 37).

Group	**n**	**Mean Rank**
Reflex	19	38.84
Balance	16	23.53
Placebo	11	26.77
Control	19	38.74
Total	65	

Table 37: Mean rank scores per treatment group (n = 65).

Mann-Whitney-U tests were then conducted to discover where the difference(s) might lie. Six comparisons using a Bonferonni adjustment would require $p < .0083$ (exact tests were used given the small sample sizes). This was not the case for any of the comparisons (see below) although if the less stringent level of $p < .05$ (see below) is accepted, then the previous DEST / DST finding, in which the balance group was different from both the reflex and the control group, (see equivalent MANOVAs) is repeated. The results, including observed effect sizes (r) are listed below, where it can be seen that all except the first comparison represent small effects (the first represents a medium effect).

1. Reflex V Balance: U = 67.5, p = .002, r = -.32
2. Reflex V Placebo: U = 62, p = .064, r = -.22
3. Reflex V Control: U = 170, p = .88, r = -.0043
4. Balance V Placebo: U = 74.5, p = .591, r = -.54
5. Balance V Control: U = 77, p = .016, r = -.30
6. Placebo V Control: U = 67, p = .124, r = -.22

9.3.3.4 Summary of treatment efficacy findings

A summary of these results is also presented in Appendix 24. No effects on the reduction of at-risk status for dyslexia were found for either of the experimental treatments and similarly no effects were found on any of the minor dependent variables in terms of change between time 1 and time 2. The one reliable difference between treatment and control conditions was in the direction opposite to that suggested by the literature (assuming treatments would decrease, rather than increase dyslexia at risk status), with the balance group achieving higher at-risk scores and proportionately more of the balance group being of at-risk status than the control group. Post-hoc tests revealed a significant difference between the balance and control group when the full sample of programme-compliant participants was considered (i.e., n = 118). This finding was repeated when the 5 private individuals were removed from this sample (i.e., n = 113) and the trend was also evident in the sample containing only these private 5 as the reflex group (n = 88), but the effect was not statistically reliable, almost certainly because the sample size was too small to detect the effect. The balance and reflex groups were significantly different in both the full 120 day compliant sample and when the ADHD participants were removed.

Where the entire sample (i.e., n = 136) was considered the only findings to emerge were a significant difference between the reflex and balance group which was repeated when the ADHD sample was removed. This, plus the significant difference between the balance and control group, was also seen when the outcome was reading-only (n = 65) (as opposed to the full DEST / DST score)[52].

9.3.4 Predictive Efficacy (Aims 3-7): Statistical Analysis

Predictive efficacy was explored using multiple regression and logistic regression. Each was performed four times to explore the predictive efficacy of slightly different predictor variables (i.e., using either the full reflex score or just the Schilder ATNR reflex score) on varied outcome variables (i.e., either the full DEST / DST score or just the reading score from the DST).

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[52] These analyses were repeated using ANCOVA rather than MANOVA which controlled for age, given the different spread of ages across groups. The changes in findings were marginal and did not affect interpretation of the results. These can be also be seen in Appendix 24.

The sample used for this exploration contained 98 children (breakdown of age and gender is shown in Table 38). Because the intervention could be interpreted as having had some effect on those in the Balance Group (albeit in *raising* their DEST / DST scores) the balance group (n = 38) were subtracted from the full, initial sample of n = 136. There was no significant difference between the other three groups on this outcome measure and so they were collapsed into one overall group. Where the reading score was used as the outcome variable, only those children who had been involved in the DST test were included (n = 54). The breakdown for the sample, minus the balance group, is shown in Table 46.

	Gender		
	Male	**Female**	
Age			**Total**
3	3	1	**4**
4	25	4	**29**
5	22	5	**27**
6	23	8	**31**
7	7	0	**7**
Total	**80**	**18**	**98**

Table 38: Breakdown of age and gender for n = 98 exploring the predictive efficacy of the motor tests.

Before looking at the results in more detail, some issues need to be addressed:

9.3.4.1 Sample size

Field (2005) quotes Green (1991) when considering sample size for conducting a multiple regression. Green recommends calculating two minimum sample sizes; one to test the overall fit of the regression model and one to test the individual predictors. In doing the former, he suggests 50 + 8k, where k equals the number of predictors. In this case, the current study would require 50 + 8(5) = 90 participants. In doing the latter, he suggests 104 + k, in which case the current study would require 104 + 5 = 109 participants. Where the full DEST / DST score was the outcome variable the number included in this sample was 98 which satisfied the former of these. However, graphs

produced by Miles and Shevlin (2001) (in Field, 2005) found that where the sample size is required to achieve a high level of power (i.e., 0.08) then n = 80 is sufficient. In the case of using the reading score from the DST, the sample size was reduced to 54 and failure to detect significance due to lack of power should be considered if no effect is observed.

9.3.4.2 Predictors

There were 5 main predictors in the predictive analyses. These were the combined scores, from time 1, on:

1. Reflex tests
2. Balance tests
3. Fine motor tests
4. Visuo-motor tests
5. Phonological tests

As we have seen, each of these scores is composed of sub-scores (numbering 28 subtests in all). The possibility of running analyses using each sub-score as a predictor was considered but this was not possible given the sample size of 98. Reconsidering Green's calculation of 50 + 8(k), the sample would need 274 participants; if using the 104 + k, then 132 participants would be necessary. Even if the variables were good predictors there might not be enough power in the sample to detect this.

9.3.4.3 Multiple regression

9.3.4.3.1 Multiple regression using the full reflex score as a predictor variable and the DEST / DST score as the outcome variable

Multiple regression allows for the prediction of an outcome variable (in this case the DEST / DST score) using two or more predictor variables. The first multiple regression made use of the full reflex score as the first predictor variable and of the DEST / DST score as the outcome variable. Its use depended upon the non-violation of certain assumptions which are discussed below.

Assumptions

- Variable types: Predictor variables can be continuous or categorical and in this case they were continuous. The outcome variable, however, must be continuous, and in the case of the DEST / DST score, this was also the case.
- Multicollinearity: Dependency between predictor variables could bias the regression model. The Pearson correlation matrix showed no correlations higher than 0.55. Field (2005) suggests that any correlation over 0.8 represents the possibility of multicollinearity and so at first sight this appears safe. A more reliable method of assessing multicollinearity is the Variance Inflation Factor (VIF). If the largest VIF is greater than 10; if the average VIF is substantially greater than 1; if tolerance is below 0.1 (serious problem) or 0.2 (potential problem) then regression may be biased (p. 196). In the current sample of n = 98, no VIF was larger than 10, the average VIF was 1.6 and the tolerance was well above 0.2 (lowest was 0.45). Thus, it appears that this assumption was met.
- Homoscedasticity: The constancy of the residuals' variance was checked for each predictor and these were judged to show a random display of dots around the 0 point.
- Assumption of independent errors: The Durban-Watson statistic tests the correlation between the residuals of observations (which should not be correlated in order to satisfy this assumption). The closer the value is to 2 the better, with values less than 1 or greater than 3 being cause for concern. In this case, Durban-Watson was 2.112 so the assumption was not violated.
- Normal distribution of errors: A histogram and normal probability plot of the DEST / DST residuals was checked and these were judged to appear normal.
- Independence: Each value of the DEST / DST score came from a different participant.

Factors influencing the regression model

- No more than 5% of the standardised residuals should be outside of +/- 2, and no more than 1% should be outside of +/- 2.5. In the current study, only one case lay outside of the 2.5 boundary (as would be expected given the sample of 98); 3 others lay between the boundaries of 2 - 2.5. Casewise diagnostics of

Cook's distance, Mahalanobis distance and centered leverage value were employed to explore this case further to see whether it was having an undue influence on the regression parameters. Cook's distance suggests undue influence if it is above 1, however, here the value was 0.3. Mahalanobis distance has undue influence if it lies above 15 in sample sizes of approximately 100. In this case it was 10.6. The average leverage is calculated by adding 1 to the number of predictors and dividing by the sample size. If values exist which are 2 or 3 times this value then they may be influencing the regression model. In this case the average leverage value was 0.122 (if x 2) and 0.184 (if x 3). The value for this case here was 0.109.

Thus, it can be ascertained that the assumptions for multiple regression were not violated on any level.

The predictor "reflex" was entered first in the following analyses for two reasons. Firstly, because of McPhillips et al.'s (2000) finding which claimed that those with high retained ATNR reflexes (specifically the Schilder) were more likely to show reading difficulties, and secondly, because of Nicolson and Fawcett's theory linking cerebellar abnormality[53] to literacy (including reading) via motor and motor-based phonological difficulties. Thus, a hierarchical regression was employed in which the measure of reflex score was entered into the model first, followed by the remaining predictor variables. Their means and standard deviations are presented in Table 39 and the model summary shows how successful the tests were in predicting the outcome of dyslexia risk and is presented in Table 40.

[53] Cerebellar abnormality is here represented by the reflex scores which then affect balance, visual-motor and fine-motor skills.

		Means	Standard Deviation
Outcome Variable	DEST / DST	0.66	.46
Predictor Variables	Reflex	1.84	.40
	Balance	2.54	.71
	Visuo-motor	9.13	3.7
	Fine-motor	15.7	5.02
	Phonological	57.8	15.9

Table 39: The means and standard deviations of the outcome and predictor variables for n = 98 participants.

Model	**R**	**R^2**	**Adjusted R^2**	**R^2 Chg.**	**F Chg.**	**Df 1**	**Df 2**	**Sig F Chg.**	**Durban-Watson**
1	0.041	0.002	-0.009	0.002	0.163	1	96	0.687	
2	0.284	0.08	-.03	0.078	1.969	4	92	0.106	2.112

Table 40: The model summary table (n = 98) where the full reflex score is a predictor variable and the DEST / DST score is the outcome variable.

R^2 shows how much of the variability of the DEST / DST score is due to the different levels entered into the model. From this we can see that the reflex tests accounted for 0.2% of the variability of the DEST / DST score ($R^2 = 0.002$, $F_{(1, 96)} = 0.163$, $p > .05$). Adding the remaining 4 predictors all together accounted for another 7.8% of the variability ($R^2 = 0.078$, $F_{(4, 92)} = 1.969$, $p > .05$). This amounts to an explained 8% of the variance overall. It is apparent, therefore, that not much of the variation of the DEST / DST score is explained by these predictors.

The *b*-values in Table 41 denote the individual contribution of each variable to the regression model. In theory positive values reflect a positive relationship and negative

ones a negative relationship[54]. There were no individual significant variables which contributed to this model. The values suggest that dyslexia risk increased as balance and fine-motor scores increased and as reflex, visuo-motor and phonological scores decreased. It has previously been explained that an increase in score reflects an improvement in ability on only the phonological measure. Therefore, Table 41 tells us that dyslexia risk increased as balance, fine-motor and phonological skills worsened and that dyslexia risk increased as reflex and visuo-motor skills improved. The standardised beta (β) shows the change in standard deviations for each predictor variable as the outcome variable increased by one standard deviation.

	B	SE B	β
Step 1			
Constant	0.57	0.22	
Reflex	0.05	0.12	.04
Step 2			
Constant	0.69	0.38	
Reflex	-0.10	0.14	-.09
Balance	0.15	0.08	.22
Visuo-motor	-0.00	0.01	-.02
Fine-motor	0.00	0.01	.02
Phonological	-0.00	0.00	-.13

Table 41: Multiple regression using the full reflex score as a predictor variable and the DEST / DST score as the outcome variable.
Note R^2 for Step 1 = 0.002; ΔR^2 for Step 2 = 0.08*(p > .05). *p < .05 **p < .01, ***p < .001*

Despite the direction of relationship between outcome and predictor variables suggested by this table, it is worth looking at the confidence intervals to examine the reliability of these findings. It can be seen in Table 42 that 95% of cases had relationships between the outcome and predictor variables whose lower and upper

[54] The direction of relationship between variables must be interpreted in light of the scoring method employed. For example, most, but not all, tests reflected worsening of ability as scores increased.

confidence boundaries crossed 0, meaning the relationship may be positive or negative. This provides further evidence that the model is not good for predicting the outcome variable and that the findings in Table 41 are unlikely to represent the true population.

	95% Confidence Interval for β	
	Lower bound	**Upper bound**
Model		
1 Constant	0.132	1.009
Reflex	-0.185	0.280
2 Constant	-0.063	1.445
Reflex	-0.377	0.174
Balance	-0.018	0.309
Visuo-motor	-0.030	0.025
Fine-motor	-0.026	0.029
Phonological	-0.011	0.003

Table 42: The 95% Confidence intervals for each predictor where the full reflex score is a predictor variable and the DEST / DST score is the outcome variable.

By comparing it to the value of R^2, the adjusted R^2 can be used to assess how well the model generalises to other samples. The difference between R^2 and the adjusted R^2 (i.e., 0.08 - 0.03) was 0.05 or about 5%. Given that only 8% of the variance was explained by the predictors, this was a large difference, suggesting that only 3% of the variance might be explained by these predictors in the true population. Stein's formula[55] (Field, 2005, p. 172, 188) yielded the likely value of adjusted R^2 in other samples, as – 0.033 which was not similar to the R^2 of 0.08 presented here. The difference corresponded to 0.113, or approximately 11% and suggested that the cross-validity of the model was not good.

An ANOVA was also performed to compare the model's ability to predict the outcome variable with the ability of the mean to do so. In the first instance it looked at whether

[55] $$Stein'sFormulaforAdjustedR^2 = 1-\left[\left(\frac{n-1}{n-k-1}\right)\left(\frac{n-2}{n-k-2}\right)\left(\frac{n+1}{n}\right)\right](1-R^2)$$

the reflex score could improve the ability to predict the outcome score, and in the second instance it looked at whether the other factors improved prediction upon this. Neither the reflex tests ($F_{(1, 98)} = 0.163$, $p > .05$), nor the next set of predictor variables ($F_{(1, 92)} = 1.609$, $p > .05$), was shown to improve prediction of the outcome variable greater than would expected by using the mean as a "best guess".

9.3.4.3.2 Multiple regression using the Schilder ATNR reflex test as a predictor variable and the DEST / DST score as the outcome variable.

Of the reflexes suggested as being important in identifying those at risk of reading failure, one set of tests stands out. This is the ANTR (Schilder) test, in which the child stands upright with arms extended outwards. It is this test in particular that McPhillips et al. reported as being a good predictor of reading failure in their sample of children. Therefore, the tests using this method (both eyes closed and open) were combined to give a total Schilder score, and this alone was used as the predictor variable to explore whether it might fare any better in predicting those at risk of dyslexia than the full reflex score. The 4 other predictor variables remained as before.

As before, all assumptions were satisfied and no factors were found to unduly influence the regression model. The means and standard deviations remain as before. The only change was for the Schilder test, for which the mean was 9.53 and the standard deviation was 3.96.

Again, the model summary allows us to see how successful the tests are in predicting the outcome of dyslexia risk and is presented in Table 43.

Model	R	R^2	Adjusted R^2	R^2 Chg	F Chg	Df 1	Df 2	Sig F Chg	Durban-Watson
1	0.043	0.002	-0.009	0.002	0.174	1	96	0.677	
2	0.289	0.083	-.034	0.081	2.047	4	92	0.094	2.117

Table 43: The model summary table (n = 98) where the Schilder score is a predictor variable and the DEST / DST score is the outcome variable.

The Schilder tests accounted for 0.2% of the variability of the DEST / DST score (R^2 = 0.002, $F(1, 96) = 0.174$, $p > .05$). Adding the remaining 4 predictors all together accounted for another 8.1% of the variability (R^2 = 0.081, $F(4, 92) = 2.047$, $p > .05$). This amounted to an explained 8.3% of the variance overall. As before, the predictors accounted for very little of the DEST / DST variation.

The *b*-values are presented in Table 44. Exactly as before, we can see that there were no individual significant contributors to the model and dyslexia risk increased as balance, fine-motor and phonological skills got worse and increased as Schilder and visuo-motor skills got better. The standardised beta (β) shows the change in standard deviations for each predictor variable as the outcome variable increases by one standard deviation.

	B	SE B	β
Step 1			
Constant	0.61	0.12	
Schilder	0.01	0.01	.04
Step 2			
Constant	0.61	0.36	
Schilder	-0.01	0.01	-.10
Balance	0.17	0.09	.26
Visuo-motor	-0.01	0.01	-.01
Fine-motor	0.00	0.01	.02
Phonological	-0.00	0.00	-.13

Table 44: Multiple regression using the full Schilder score as a predictor variable and the DEST / DST score as the outcome variable.
Note R^2 for Step 1 = 0.002; ΔR^2 for Step 2 = 0.083*(ps > .05). *p < .05, **p < .01, ***p < .001*

Table 45 shows that 95% of cases have relationships between the outcome and predictor variables whose lower and upper confidence boundaries cross 0. This again suggests that the model was not good for predicting the outcome variable and that the findings in Table 44 (above) were unlikely to represent the true population.

	95% Confidence Interval for β	
	Lower bound	**Upper bound**
Model		
1 Constant	0.365	0.855
Schilder	-0.19	0.29
2 Constant	-0.100	1.31
Schilder	-0.4	0.015
Balance	-0.06	0.345
Visuo-motor	-0.029	0.027
Fine-motor	-0.027	0.024
Phonological	-0.011	0.003

Table 45: 95% Confidence intervals for each predictor.

Comparing R^2 and adjusted R^2 (i.e., 0.083 - 0.034) shows that the difference was 0.049 or still about 5%. As 8.3% of the variance is explained by the predictors, this means that only about 3.3% of the variance would be explained by these predictors in the true population. Stein's formula to determine the likely value of adjusted R^2 in other samples was 0.03, which is not similar to the R^2 of 0.083 presented here. The difference corresponds to 0.113, or approximately 11%. The difference suggests that the cross-validity of the model is not good.

From the ANOVA, it was found that, once again, neither the Schilder tests ($F(1, 98) = 0.174$, $p > .05$), nor the next set of predictor variables ($F(5, 92) = 1.67$, $p > .05$) improved prediction of the outcome variable greater than would expected by using the mean as a "best guess".

9.3.4.3.3 Multiple regression using the full reflex score as a predictor variable and the reading test from the DST as the outcome variable (i.e., Reynolds et al., 2005)

Given the previously mentioned effects of the Dores (i.e., DDAT) programme on reading scores (Reynolds et al., 2005) a multiple regression was performed using only

the reading score on the DST as the outcome measure. The DEST is designed for children who may not yet have learned to read and as such does not have a reading test. Therefore, it was only possible to use the DST sample (n = 54[56]). Although this sample size was less than ideal, it was nevertheless performed to explore whether any variables might predict reading. The breakdown for this group is shown in Table 46.

	Gender		
	Male	**Female**	
Age			**Total**
6	12	4	**16**
7	23	8	**31**
8	7	0	**7**
Total	**42**	**12**	**54**

Table 46: Breakdown of age and gender for n = 54 exploring the predictive efficacy of the motor tests. (Children's ages are based on ages at end of study in order to avoid confusion regarding the use of DST for those over 6.5 years).

This was done using both the full reflex score as one of the predictor variables and using the Schilder ATNR score only (see next section). The 4 other predictor variables remained as before. Again, all assumptions were satisfied for both the following Regression models and no factors were found to unduly influence the regression model. The means and standard deviations of the outcome and predictive variables are shown in Table 47 and the model summary is presented to show how successful the tests are in predicting the outcome of dyslexia risk.

[56] This number is smaller than the previously used sample size of 65 for children tested on the DST because for the predictive efficacy analysis those in the balance group were not included.

		Means	Standard Deviation
Outcome Variable	DST Reading score	35.07	22.44
Predictor Variables	Reflex	1.73	0.33
	Balance	2.35	0.63
	Visuo-motor	8.2	3.37
	Fine-motor	13.65	3.96
	Phonological	67.81	9.668

Table 47: The means and standard deviations of the outcome and predictor variables for n = 54 participants.

Model	R	R^2	Adjusted R^2	R^2 Chg	F Chg	Df 1	Df 2	Sig F Chg	Durban-Watson
1	0.095	0.009	-0.01	0.009	0.472	1	52	0.495	
2	0.506	0.257	0.179	0.248	3.995	4	48	0.007	1.69

Table 48: The model summary table (n = 54) where the full reflex score is a predictor variable and reading in the outcome variable.

The reflex tests accounted for 0.9% of the variability of the DST reading score (R^2 = 0.009, $F_{(1, 52)} = 0.472$, $p > .05$). Adding the remaining 4 predictors all together accounted for another 24.8% of the variability ($R^2 = 0.248$, $F_{(4, 48)} = 3.995$, $p < .01$). This amounted to an explained 25.7% of the variance overall. The inclusion of the four new predictors explained a significant amount of the reading outcome, whereas the reflex score did not.

Examining the individual contribution of the predictor variables to the reading outcome (Table 48) it was evident that three variables in step 2 made a significant contribution to the outcome variable; reflex, fine-motor and phonological scores.

Reflex retention (standardised β = -.35): The reflex score appeared to have a negative relationship with the outcome suggesting that as reflex score increased by one standard deviation (.33 units) (i.e., higher level of retained reflexes), the reading score would drop by approximately one third of a standard deviation. The standard deviation for reading scores was 22.44%, so this constituted a change of -7.86% (22.44% x -.35)

in reading scores for every .33 units increase in reflex retention scores. This is true only if the other factors in the model are held constant.

Phonological ability (standardised β =.5): These scores were positively related to reading. As the former goes up, reading score improved by half a standard deviation. So for every one standard deviation increase of .5 units in phonological ability, reading improved by 11.22% (22.44% x .5).

Fine-motor ability (standardised β = .4): The direction of scoring on the fine-motor tasks is such that an increase suggests poorer performance. Thus, as fine-motor ability got worse, reading scores improved (by 1/3-1/2 a standard deviation). A change of one standard deviation in fine-motor skills (i.e., .4 units) was reflected in a change of approximately 9% in reading skills (i.e., doesn't make sense).

	B	SE B	β
Step 1			
Constant	46.17	16.45	
Reflex	-6.41	9.34	-.01
Step 2			
Constant	-33.10	27.05	
Reflex	-23.44	10.78	-.35*
Balance	2.86	5.56	.08
Visuo-motor	-0.86	0.87	-.13
Fine-motor	2.28	0.94	.40*
Phonological	1.15	0.32	.5**

Table 49: Multiple regression using the full reflex score as a predictor variable and the reading test from the DST as the outcome variable.
Note R^2 for Step 1 = 0.009 ($p > .05$); ΔR^2 for Step 2 = 0.248*(s < .01). * p < .05, ** p < .01*

Table 50 shows the boundaries for 95% of cases. Two of the predictor variables have boundaries which cross 0 suggesting they are not good for predicting the outcome variable. The three variables, of reflex skills, fine-motor skills and phonological skills, have tighter boundaries, either above or below 0.

	95% Confidence Interval for β	
	Lower bound	**Upper bound**
Model		
1 Constant	13.163	79.191
Reflex	-25.145	12.32
2 Constant	-87.45	21.281
Reflex	-45.115	-1.77
Balance	-8.321	14.044
Visuo-motor	-2.6	0.883
Fine-motor	0.4	4.16
Phonological	0.515	1.78

Table 50: The 95% confidence intervals for each predictor where the full reflex score is a predictor variable and reading score is the outcome variable.

Comparing R^2 and adjusted R^2 (i.e., 0.257 - 0.179) to assess the generalisability of the model, the difference was 0.078 or about 7.8%. As 25.7% of the variance was explained by the predictors, there remains approximately 18% which is explained by these predictors. Stein's formula showed that the likely value of adjusted R^2 in other samples was – 0.005 which is not similar to the R^2 of 0.257 presented here. The difference suggests that the cross-validity of the model is not good.

From the ANOVA, it was found that the Schilder test ($F_{(1, 52)} = 0.472$, $p > .05$) did not significantly predict the outcome measure. The added variables in step 2 however did ($F_{(5, 48)} = 3.312$, $p < .05$) significantly improve prediction of the outcome variable better than would be expected by using the mean as a "best guess".

9.3.4.3.4 Multiple regression using the Schilder ATNR as a predictor variable and the reading test from the DST as the outcome variable (i.e., Reynolds et al.)

Multiple regression was conducted as before using the Schilder ATNR score only as the Reflex measure and the using DST reading-only for the outcome measure. The

other 4 predictor variables remained as before and all assumptions were satisfied. No factors were found to unduly influence the regression model.

The means and standard deviations remain as before. The only change was for the Schilder test, for which the mean was 8.41 and the standard deviation 3.45.

The model summary allows us to see how successful the tests are in predicting the outcome of dyslexia risk and is presented in Table 51.

Model	R	R^2	Adjusted R^2	R^2 Change	F Change	Df 1	Df 2	Sig F Change	Durban-Watson
1	0.196	0.038	0.02	0.038	2.076	1	52	0.156	
2	0.472	0.223	0.142	0.185	2.85	4	48	0.034	1.729

Table 51: The model summary table (n = 54) where the Schilder score is a predictor variable and reading in the outcome variable.

The Schilder tests accounted for 3.8% of the variability of the DST reading score ($R^2 = 0.038$, $F_{(1, 52)} = 0.156$, $p > .05$). Adding the remaining 4 predictors all together accounted for another 18.5% of the variability ($R^2 = 0.185$ $F_{(4, 48)} = 2.85$, $p < .05$). This amounts to an explained 22.3% of the variance overall. The inclusion of the four new predictors explained a significant amount of the reading outcome, whereas the Schilder score did not.

The *b*-values are presented in Table 52. The only significantly meaningful finding here is that as phonological score increased by one standard deviation (i.e., 9.7 units), DST reading score increased by just over a third of a standard deviation. The standard deviation for reading is 22.44%, therefore an 8.3% (i.e., 22.44% x .37) increase is seen in this skill with 9.7 unit change in phonological scores.

		B	SE B	β
Step 1				
	Constant	24.38	8.02	
	Schilder	1.27	0.88	.20
Step 2				
	Constant	-36.58	27.55	
	Schilder	1.53	0.98	.24
	Balance	-6.02	6.22	-.17
	Visuo-motor	-0.99	0.89	-.15
	Fine-motor	1.65	0.89	.30
	Phonological	0.86	0.32	.37**

Table 52: Multiple regression using the Schilder as a predictor variable and reading as the outcome variable.
Note R^2 for Step 1 = 0.038 ($p > .05$); ΔR^2 for Step 2 = 0.142 *($p < .05$). *$p < .05$, **$p < .01$, ***$p < .001$*

Table 53 shows that for all predictors except phonological skill, 95% of cases had relationships between the outcome and predictor variables whose lower and upper confidence boundaries cross 0.

	95% Confidence Interval for β	
	Lower bound	**Upper bound**
Model		
1 Constant	8.30	41.46
Schilder	-0.50	3.04
2 Constant	-91.98	18.82
Schilder	-0.43	3.49
Balance	-18.53	6.49
Visuo-motor	-2.77	0.80
Fine-motor	-0.14	3.44
Phonological	0.23	1.50

Table 53: The 95% confidence intervals for each predictor where the Schilder score is a predictor variable and the reading score is the outcome variable.

Comparing R^2 and adjusted R^2 (i.e., 0.223 - 0.142), the difference yielded was 0.081 or about 8%. Given that 22.3% of the variance was explained by the predictors, this meant that 8% less (or 14.3%) might be explained in the true population. Stein's formula gave the likely value of adjusted R^2 in other samples– 0.05 which was not similar to the R^2 of 0.223 presented here. The difference suggested that the cross-validity of the model was not good.

From the ANOVA, it was found that the Schilder tests ($F_{(1, 52)} = 2.076$, $p > .05$) did not improve prediction of the outcome variable better than the mean would be expected to, but that the next set of predictor variables ($F_{(5, 48)} = 2.754$, $p < .05$) improved prediction of the outcome variable greater than would expected by using the mean as a "best guess".

9.3.4.4 Logistic regression

The only change made between multiple and logistic regression is that the outcome variable is categorical rather than continuous. Thus, DST score is labelled as either "at risk" or as "not at risk" according to the scoring procedure. Similarly, where the

reading-only score was used children were labelled according to whether they fell within the average range of reading performance (or above) (i.e., >= 25th percentile) or below the average range of performance (i.e., < 25th percentile)[57].

As with the multiple regression, the logistic regression made use of the forced entry method because of the existence of previous research which allows for the testing of a theory. Reflex score was entered in the first block with the other predictor variables (see next paragraph) being added in the second block.

The same predictor variables were used here as were used in the multiple regression (i.e., reflex scores, balance scores, visual-motor scores, fine-motor scores and phonological scores). A second analysis was conducted using only the Schilder ATNR test in place of the Reflex scores, in accordance with McPhillips et al.'s (2000) findings. The outcome variable was again DST scores, with DST reading-only score being used in a further analysis.

Assumptions

- Multicollinearity: Logistic regression is as prone to the biasing effects of multicollinearity as is multiple regression. As before, the Variance Inflation Factor (VIF) was examined to ensure that no values were greater than 10. Also, tolerance was well above 0.1. This was the case for all of the following logistic regression analyses.
- Logistic regression makes no assumption about the form taken by the predictor variables (i.e., they do not have to be normally distributed). Neither does it make any assumptions about homoscedasticity.

Factors influencing the regression model

- As before, no more than 5% of the standardised residuals should be outside of +/- 2 standard deviations, and no more than 1% should be outside of +/- 2.5. Using the initial samples (i.e., n = 98 and n = 54) there was evidence that for the first analysis, 7 of the 98 participants' scores lay outside of these boundaries (i.e., n = 91). For the second sample, 3 of the participants' scores lay outside of

[57] The 25th percentile corresponds to one standard deviation below the mean, and marks the point at which performance is "below average".

these boundaries (i.e., n = 51). For the third analysis, 8 of the scores lay outside of these boundaries (i.e., n = 90). For the fourth analysis, the same three participant scores as in analysis two lay outside the boundaries (i.e., n = 51). These participants were removed from the samples.

- Casewise diagnostics of Cook's distance and centered leverage value were also examined for undue influence on the regression parameters. All values satisfied the criteria (i.e., < 1 in the case of Cook's Distance and not greater than 3 x [(k+1)/n] for centered leverage.

Thus, it is unlikely that the assumptions for logistic regression were violated.

9.3.4.4.1 Logistic regression using the full reflex score as a predictor variable and the DEST / DST as the outcome variable (n = 91)

The Hosmer and Lemeshow statistic showed that the first block of this model predicted real-world data well, because the predicted and observed values were not significantly different from each other ($p > .05$)[58]. With only the constant included in the model, 81.3% of cases were correctly classified. With the addition of reflex as a predictor, the same number of cases were correctly classified (an increase of 0%). With the addition of this predictor variable to the model, the ability of the model to predict dyslexia status did not improve significantly ($p > .05$).

The significance value of the Wald statistic for this predictor further indicated that it did not significantly predict dyslexia risk ($p > .05$). The value of Exp *b* is greater than 1 here (see Table 54) so the chance of being at risk of dyslexia increased as reflex score increased by one point (which given the direction of scoring suggested that as reflex scores got worse, risk of dyslexia increased). However, the confidence interval crosses 1 and so the generalisability of this finding is limited because it suggests that this value could indicate either a positive or a negative relationship in the population. Calculating the R^2 showed that only 1.4% of the variance in dyslexia risk status was accounted for by the reflex predictor.

With the addition of the second set of predictor variables the Hosmer and Lemeshow statistic remained $p > .05$. The percentage of cases correctly classified now

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[58] This was the case for all subsequent logistic regression analyses.

rose from 81.3% to 83.5% - an increase of 2.2%. The effect of adding these predictor variables was a significant increase in the model's ability to predict dyslexia status ($p < .001$) however the Wald statistic did not identify any specific variables as significant predictors themselves. R^2 shows that 22.4% of the variance in dyslexia risk was now accounted for by the entire model.

			95% CI for Exp *b*		
		B(SE)	Lower	Exp *b*	Upper
Step 1					
	Constant	-2.82 (1.26)			
	Reflex	0.72 (0.64)	0.58	2.04	7.15
Step 2					
	Constant	-1.53 (2.4)			
	Reflex	-0.64 (0.87)	0.1	0.53	2.00
	Balance	0.95 (0.52)	0.94	2.6	8.60
	Visuo-motor	-0.15 (0.1)	0.71	0.86	0.95
	Fine-motor	0.12 (0.09)	0.94	1.13	1.46
	Phonological	-0.04 (0.87)	0.92	0.96	1.00

Table 54: Logistic regression using forced entry with Reflex score as first predictor and DEST / DST as outcome variable.

Note: Step 1: R^2 = 3.87 (Hosmer & Lemeshow), .013 (Cox & Snell), .02 (Nagelkerke). Model $\chi^2(1) = 1.23$
Step 2: R^2 = 4.9 (Hosmer & Lemeshow), .19 (Cox & Snell), .31 (Nagelkerke). Model $\chi^2(5) = 19.63$

Reflex as predictor and mild dyslexia risk as outcome (n=91)

The same analysis was run where the DEST criteria for "dyslexia risk" was dropped to a "mild" level. Findings for the first block were similar to that of the previous analysis (i.e., "strong risk" of dyslexia). With the addition of reflex to the model in block 1 the model showed no significant ability to predict dyslexia status ($p > .05$) and there was no change in the percentage of people correctly classified (it remained at 70.3%). The Wald statistic again showed this predictor to have no significant predictive efficacy in status prediction ($p > .05$) and the confidence intervals again crossed 1, limiting the

generalisability of the finding. Calculating the R^2 showed that only 1.4% of the variance in dyslexia risk status was accounted for by the Reflex predictor.

In block 2, percentage of classification rose to 84.6% (an improvement of 14.3%) and the addition of the next five predictors significantly improved the model's ability to predict dyslexia status ($p < .001$). Three predictors demonstrated significant abilities to predict such status: visual-motor skills, fine-motor skills and phonological skills. With visual-motor skills ($p < .05$), Exp *b* was less than 1 (.77) suggesting that dyslexia risk increased as visual-motor scores reduced (i.e., as this ability improved, dyslexia risk increased). With fine-motor skills ($p < .05$), it appeared that dyslexia risk increased as these scores increased, suggesting that dyslexia was more probable if fine-motor coordination worsened. Finally, with phonological skills ($p < .05$), it was evident that dyslexia risk increased as such scores reduced which meant that dyslexia was more probable if phonological ability was poor. None of the confidence intervals corresponding to these variables crossed 1 (see Table 55). R^2 showed that, with the addition of the second block, 40% of the variance in dyslexia risk status is now explained.

		95% CI for Exp *b*		
	B(SE)	Lower	Exp *b*	Upper
Step 1				
Constant	-2.12 (1.08)			
Reflex	0.72 (0.56)	0.69	2.04	6.1
Step 2				
Constant	2.3 (2.4)			
Reflex	-1.07 (0.9)	0.06	0.34	2.00
Balance	1.04 (0.57)	0.94	0.94	8.60
Visuo-motor	-0.27* (0.1)	0.62	0.62	0.95
Fine-motor	0.19* (0.1)	1.00	1.2	1.45
Phonological	-0.09* (0.03)	0.87	0.92	0.96

Table55: Logistic regression using forced entry with Reflex score as first predictor and DEST / DST (mild) as the outcome variable.

Note: Step 1: R^2 = 5.83 (Hosmer & Lemeshow), .018 (Cox & Snell), .03 (Nagelkerke). Model $\chi^2(1) = 1.65$

Step 2: R^2 = 10.28 (Hosmer & Lemeshow), .39 (Cox & Snell), .55 (Nagelkerke). Model $\chi^2(5) = 44.23$

*p < .05, **p < .01, ***p < .001

9.3.4.4.2 Logistic regression using the Schilder ATNR reflex test as a predictor variable and DEST / DST as the outcome variable (n = 91)

With only the constant included in the model, 81.3% of cases were correctly classified and this did not change with additions to the predictive model, either in the first block where combined Schilder scores were used instead of combined reflex scores or where the other five variables were introduced. The addition of Schilder to the model did not significantly improve the model's ability to predict dyslexia status (p > .05) and the Wald statistic for this variable showed it to have no significant predictive ability (p > .05). Only .05% of the dyslexia status variance was explained by this part of the model.

The effect of adding the remaining five predictor variables significantly improved the model's ability to predict dyslexia status (p < .001). The Wald statistic

identified balance scores as a variable which significantly predicted dyslexia status ($p < .05$). The confidence intervals did not cross 1 (see table 56) and Exp *b* was above 1, suggesting that dyslexia risk increased as such scores increased (i.e., as balance skills got worse, dyslexia risk increased). R^2 shows that 22.5% of the variance in dyslexia risk was now accounted for by the entire model.

		95% CI for Exp *b*		
	B(SE)	Lower	Exp *b*	Upper
Step 1				
Constant	-1.61 (0.71)			
Schilder	0.01 (0.07)	0.89	1.01	1.16
Step 2				
Constant	-1.62 (2.17)		0.2	
Schilder	-0.14 (0.09)	0.72	0.87	1.04
Balance	1.27* (0.57)	1.16	3.56	10.93
Visuo-motor	-0.12 (0.1)	0.73	0.89	1.08
Fine-motor	0.08 (0.08)	0.92	1.08	1.27
Phonological	-.04 (0.02)	0.92	0.96	1.00

Table 56: Logistic regression using forced entry with Schilder score as first predictor and DEST / DST as the outcome variable.

Note: Step 1: R^2 = 7.75 (Hosmer & Lemeshow), .00 (Cox & Snell), .00 (Nagelkerke).
Model $\chi^2(1) = .045$
Step 2: R^2 = 4.87 (Hosmer & Lemeshow), .2 (Cox & Snell), .32 (Nagelkerke).
Model $\chi^2(5) = 19.8$ (only one interaction has not been reported here).

* $p < .05$, ** $p < .01$, *** $p < .001$

Schilder as predictor and mild dyslexia risk as outcome (n=90)

As before, the analysis was run where the DEST criteria for "dyslexia risk" was dropped to a "mild" level. Findings for the first block were again similar to that of the previous analysis (i.e., "strong risk" of dyslexia). With the addition of Schilder to the model in block 1 the model showed no significant ability to predict dyslexia status ($p > .05$) and the percentage of people correctly classified into dyslexia status remained at 71.1%. The Wald statistic showed this predictor to have no significant predictive

efficacy in status prediction ($p > .05$) and the confidence intervals crossed 1. Calculating the R^2 showed that only 0.8% of the variance in dyslexia risk status was accounted for by the Schilder predictor.

In block 2, percentage of classification rose to 86.7% (an improvement of 15.6%) and the addition of the next five predictors significantly improved the model's ability to predict dyslexia status ($p < .001$). Two predictors demonstrated significant abilities to predict such status; visual-motor skills and phonological skills. With visual-motor skills ($p < .05$), Exp *b* was less than 1 (.77) suggesting that dyslexia risk increased as visual-motor scores reduced (i.e., as this ability improved, dyslexia risk increased). With phonological skills ($p < .001$), it appeared that dyslexia risk increased as the scores decreased, suggesting that dyslexia was more probable as this skill worsened. None of the confidence intervals corresponding to these variables crossed 1 (see Table 57) and R^2 showed that, with the addition of the second block, 42.7% of the variance in dyslexia risk status was now explained.

		95% CI for Exp *b*		
	B(SE)	Lower	Exp *b*	Upper
Step 1				
Constant	-1.42 (0.62)			
Schilder	0.05 (0.06)	0.94	1.06	1.18
Step 2				
Constant	1.84 (2.21)			
Schilder	-1.3 (0.9)	0.73	0.88	1.06
Balance	1.26 (0.65)	0.99	3.5	12.46
Visuo-motor	-0.27* (0.11)	0.62	0.77	0.96
Fine-motor	0.16 (0.09)	1.00	1.2	1.4
Phonological	-0.1*** (0.03)	0.86	0.91	0.96

Table 57: Logistic regression using forced entry with reflex score as first predictor and DEST / DST (mild) as the outcome variable.

Note: Step 1: R^2 = 4.82 (Hosmer & Lemeshow), .009 (Cox & Snell), .01 (Nagelkerke). Model $\chi^2(1) = 0.85$

Step 2: R^2 = 4.16 (Hosmer & Lemeshow), .4 (Cox & Snell), .58 (Nagelkerke). Model $\chi^2(5) = 46.6$

*p < .05, **p < .01, ***p < .001

9.3.4.4.3 Logistic regression using Reflexes as a predictor variable and the reading tes from the DST as the outcome variable (i.e., Reynolds et al.) (n = 51)

With only the constant included in the model, 66.7% of cases were correctly classified. With the addition of reflex as a predictor, this remained the same. There was no significant change in the model's ability to predict dyslexia status (p > .05). The significance value of the Wald statistic for this predictor further indicated that it did not significantly predict dyslexia risk (p > .05). R^2 showed that only 0.53% of the variance in dyslexia risk status was accounted for by the reflex predictor.

With the addition of the second set of predictor variables the percentage of cases correctly classified rose from 66.7% to 92.2% - an increase of 25.5%. The effect of adding these predictor variables was a significant increase in predictive ability of the model ($p < .001$). The Wald statistic identified all predictor variables except visuo-motor scores as significant predictors ($p < .05$) of dyslexia status. For reflex scores ($p < .01$), Exp *b* is above 1 which means that dyslexia risk increases as reflex scores go up (i.e., ability gets worse), however, given the size of the confidence interval here, this variable should be considered unreliable and care should be taken in deriving any meaning from it[59]. For balance ($p < .05$), Exp *b* was less than 1 suggesting that dyslexia risk increased as these scores went down (i.e., as the ability improved). For fine-motor and phonological scores ($p < .01$), Exp *b* was less than 1 which meant that dyslexia risk increased as these scores reduced. In the case of fine-motor skills this meant as this ability improved but in the case of phonological scores it meant that as this ability worsened. The confidence intervals for these predictor variables did not cross 1. R^2 shows that 61.5% of the variance in dyslexia risk was now accounted for by the entire model. The values for these predictor variables are shown in Table 58.

[59] Confidence intervals in logistic regression are determined by using log odd ratios to calculate the standard error. This is the square root of the reciprocals of the frequencies. For the confidence interval to be this large, it would suggest that the frequency of one of the cells is extremely small (possibly 0) which would give rise to a tiny / infinite fraction. In this case the standard error would go up causing large confidence intervals. In such cases it is probably best to leave out the predictor variable or to exercise extreme caution when deriving meaning from it.

		95% CI for Exp *b*		
	B(SE)	Lower	Exp *b*	Upper
Step 1				
Constant	-1.6 (1.6)			
Reflex	0.52 (0.88)	0.3	1.68	9.36
Step 2				
Constant	33.05 (12.33)			
Reflex	16.62** (6.4)	60.89	1.7E+07	4.5E+12
Balance	-2.9* (1.37)	0.00	0.06	0.8
Visuo-motor	0.30 (0.25)	0.84	1.35	2.2
Fine-motor	-1.6** (0.61)	0.06	0.2	0.66
Phonological	-0.57** (0.21)	0.37	0.57	0.86

Table 58: Logistic regression using forced entry with reflex score as first predictor and Reading as the outcome variable.
Note: Step 1: R^2 = 2.46 (Hosmer & Lemeshow), .007 (Cox & Snell), .009 (Nagelkerke). Model $\chi^2(1) = .35$
Step 2: R^2 = 1.72 (Hosmer & Lemeshow), .54 (Cox & Snell), .75 (Nagelkerke). Model $\chi^2(5) = 39.95$
* $p < .05$, **$p < .01$, ***$p < .001$

9.3.4.4.4 Logistic regression using the Schilder ATNR as a predictor variable and the reading test from the DST as an outcome variable (i.e., Reynolds et al.) (n = 51)

With only the constant included in the model, 66.7% of cases were correctly classified and this did not change with the introduction of the first predictor (Schilder) in block 1. The addition of the Schilder as a predictor improved the ability of the model to predict dyslexia status ($p < .05$) but the variable itself did not show any evidence of such predictive efficacy ($p > .05$). The R^2 calculation showed that 6.55% of the variance in dyslexia risk status was accounted for by the addition of the Schilder scores to the model.

The percentage of cases correctly classified in block 2 rose by 9.8% to 76.5%. The effect of adding these predictor variables is a significant increase in ability of the model to predict dyslexia status ($p < .001$).The Wald statistic identified the Schilder ($p < .05$), Visuo-motor ($p < .05$) and phonological ($p < .01$) scores as significant predictors of dyslexia status. The value of Exp *b* was less than 1 (see Table 59) for the Schilder scores, indicating that the risk of dyslexia increased as these scores went down (i.e., as presence of Schilder decreased). For Visuo-motor scores, dyslexia risk went up as these increased (i.e., as performance got worse). Finally, for phonological scores, dyslexia risk increased as these scores decreased (i.e., got worse). The confidence intervals for these predictor variables did not cross 1. The R^2 calculation showed that 34.5% of the variance in dyslexia risk was now accounted for by the entire model. The values for these predictors are shown in Table 59.

		95% CI for Exp *b*		
	B(SE)	Lower	Exp *b*	Upper
Step 1				
Constant	0.82 (0.8)			
Schilder	-0.19 (0.1)	0.68	0.83	1.0
Step 2				
Constant	10.4 (4.04)			
Schilder	-0.4* (0.18)	0.47	0.67	0.96
Balance	1.16 (0.85)	0.61	3.2	16.79
Visuo-motor	0.27* (0.13)	1.01	1.3	1.71
Fine-motor	-0.25 (0.13)	0.61	0.78	1.0
Phonological	-0.15** (0.05)	0.78	0.86	0.95

Table 59: Logistic regression using forced entry with Schilder score as first predictor and Reading as the outcome variable.
Note: Step 1: R^2 = 5.8 (Hosmer & Lemeshow), .08 (Cox & Snell), .11 (Nagelkerke). Model $\chi^2(1) = 4.25$
Step 2: R^2 = 5.67 (Hosmer & Lemeshow), .36 (Cox & Snell), .49 (Nagelkerke). Model $\chi^2(5) = 22.43$
* $p < .05$, ** $p < .01$, ***$p < .001$

Summary of predictive efficacy findings

The multiple regression analyses found that, where the outcome variable was the full DEST / DST score, the predictor variables were not significantly useful in improving the model to predict outcome, something which applied both when the full reflex score was used as the step 1 predictor and when the Schilder was used as the step 1 predictor. When logistic regression was employed, in which case dyslexia status was categorical (i.e., either at risk or not at risk), findings were similar. No predictor was useful, by itself, when the full reflex score was entered first, though the model in step 2 did improve significantly in its ability to predict dyslexia status. When the Schilder score was entered first, one predictor (i.e., balance) significantly contributed to the ability of the model (step 2) to predict dyslexia status.

Logistic regression was also used where the at risk level included those at mild risk for dyslexia. In this case, where full reflex was entered at step 1, there were three predictor variables which significantly improved the ability of the model to predict status. There were: visual-motor, fine-motor and phonological scores. Where Schilder was entered at step 1, fine-motor scores were lost and only visual-motor and phonological scores remained as significantly improving the predictive ability of the model.

Where reading score was the outcome variable, rather than the full DEST / DST score, there were more significant findings. Multiple regression confirmed the previous finding that neither full reflex score nor the Schilder score, when added at step 1, contributed significantly to the ability of the regression model to predict reading status, but in both cases, step 2 showed that the phonological score contributed significantly to the regression model's predictive ability. Where full reflex scores were entered at step 1, both reflex scores and fine-motor scores also contributed significantly to the model's predictive ability in step 2. Logistic regression showed that at step 1 the model was significantly better able to predict reading ability when Schilder was included at step 1, although there was no evidence for the Schilder score being a significantly good predictor by itself. The predictive ability of the model was not significantly improved when the full reflex score was entered at step 1 however. As before, phonological score contributed significantly to the regression model at step 2, when both the full reflex score and the Schilder score was entered at step 1. For the former (i.e., full reflex at step 1), balance, reflex and fine-motor scores also contributed significantly to the

regression model, and for the latter (i.e., Schilder at step 1), Schilder and visual-motor scores contributed significantly to the regression model. This is summarised in table form in Appendix 25.

To interpret the results of the multiple regression and logistic regression models, it may be tentatively concluded that:

- Neither "combined reflexes" nor the Schilder test predict dyslexia risk status at outcome. However, in both types of model higher levels of combined reflexes are associated with lower outcome reading scores.
- Poorer phonological skill, at time one, contributes significantly in both models to the prediction of increased dyslexic risk status and to poorer reading scores.
- Poorer fine-motor skills and poorer balance performance both contribute to the prediction of increased risk for dyslexia but also of better reading scores.
- Higher visual-motor skill performance contributes to the prediction of increased risk for dyslexia in both types of model.

Given the consistent role played by phonological skills in predicting dyslexia risk status, it is worthwhile examining the distribution of participants across groups in terms of a criterion of low phonological competence – presumably these are the participants for whom the experimental intervention might have had some effect.

A chi-square was run to investigate this, which showed that the proportions of low versus high phonological scorers was equivalent across groups at time 1($\chi^2(3) = 1.43$ p = .72). A chi-square matrix showing the proportions of high v low phonological scorers across group is presented in Table 60.

Group	Phonological status		
	Low	*High*	**Total**
Reflex	12	23	**35**
Balance	13	14	**27**
Placebo	7	9	**16**
Control	18	22	**40**
Total	**50**	**68**	**118**

Table 60: Proportions of low v high phonological scorers per group.

There was no evidence that those who initially scored poorly on the measure of phonological skill (i.e., below the 50th percentile) benefited more from an experimental programme than those in the control groups. First, the change in phonological score, from time 1 to time 3, was no different between any of the groups ($F_{(3, 115)} = .009$, $p = .999$), and, second, the DEST / DST scores were examined using an ANOVA to see whether there were differences between the high and low phonological scorers. This revealed no significant differences ($F_{(3, 115)} = 2.63$, $p > .05$).

9.3.4.5 Some final analyses

The final aim (aim 8) looked at the relationship between the phonological skills and tests of cerebellar functioning in reference to Liberman and Mattingly's (1985) motor theory of speech. In addition to this, relevant suggestions in the research literature rendered two further logistic regression analyses necessary. The first arose from the previously mentioned relationship between ADHD and dyslexia in the context of motor theory (e.g., Denckla et al., 1985; Wimmer et al., 1999). Thus, it was appropriate to examine whether the motor-based predictor variables were useful in classifying groups according to risk for ADHD. The second analysis arose from Fawcett and Nicolson's (1999) suggestion that perhaps only severe dyslexics score highly on such motor tasks. For this analysis, only those who scored under the 11th percentile on their DST reading task were considered "severe" and everyone else was classed as not severe (despite some of them being below average readers [i.e., 12-24th percentile]).

In both cases logistic regression was run using the largest sample possible as well as the largest sample possible minus those who had not performed at least 120 days of their programme. There were no differences in the results yielded by each analysis and, therefore, only one set of results is reported. In each case this represents the largest sample possible given the analysis in question. Both analyses satisfied the assumptions of standardised residuals, Cook's distance and centered leverage value

9.3.4.5.1 Aim 8: To investigate whether tests of phonological skills are related to performance on cerebellar tests in order to test this prediction from Liberman and Mattingly's (1985) motor theory of speech (n = 136)

Four Pearson[60] correlations were run to examine the relationship between phonological scores and tests of cerebellar function (taken at time 1). The cerebellar tests included the (gross) balance tests, the visual-motor tests and the fine-motor tests. An average of these three tests was also taken and this too was compared to the phonological score. All of the correlations showed a significant positive relationship[61], with balance, visual-motor and fine-motor skills getting worse as phonological scores got worse. The means and standard deviations of the test averages are shown in table 61 and the Pearson correlations are shown in table 62. Scattergrams showing the line of best fit are shown in Figures 15-18.

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[60] Non-parametric Spearman tests showed the same levels of significance.

[61] The scores for Phonological tests were scored in an opposite direction to all the other tests. An increase in Phonological score suggested an improvement in Phonological ability whereas an increase in any of the other test scores suggested a worsening of ability. This means that what might look like a negative relationship in the scattergram is in fact a positive relationship.

Test	Mean	Standard Deviation
Phonological score	55.71	16.12
Balance score	2.61	.72
Visual-motor score	8.96	3.66
Fine-motor score	15.91	5.12
Average of above	9.16	2.56

Table 61: Means and standard deviations of the phonological, balance, visual-motor, fine-motor and combined tests.

Comparison	r (2 tailed)	P	Direction
Phon & Balance	.41	< .000	Positive
Phon & Visual	.24	< .005	Positive
Phon & Fine	.48	< .000	Positive
Phon & Combined	.47	< .000	Positive

Table 62: Pearson correlations between the difference combinations of phonological and cerebellar tests.

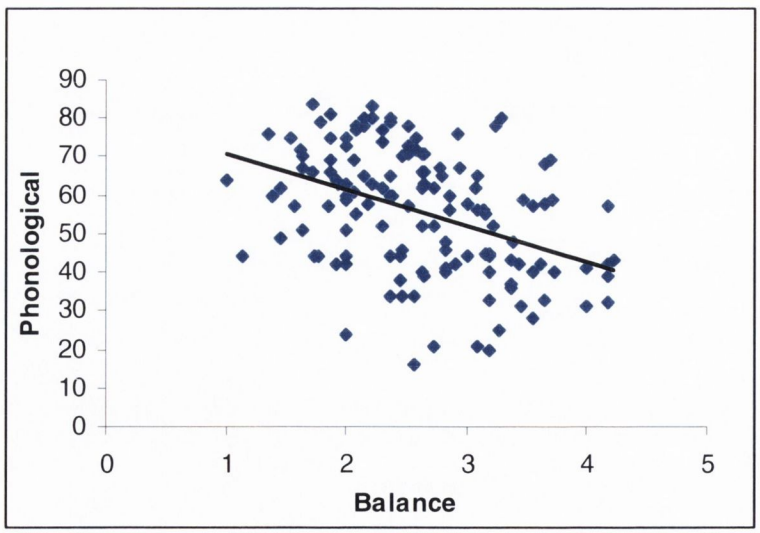

Figure 15: Scattergram showing the relationship between phonological scores and balance scores.

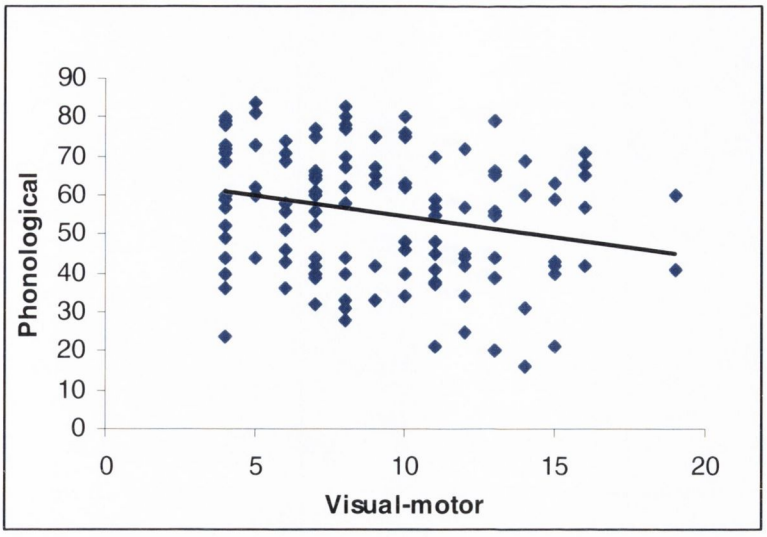

Figure 16: Scattergram showing the relationship between phonological scores and visual-motor scores.

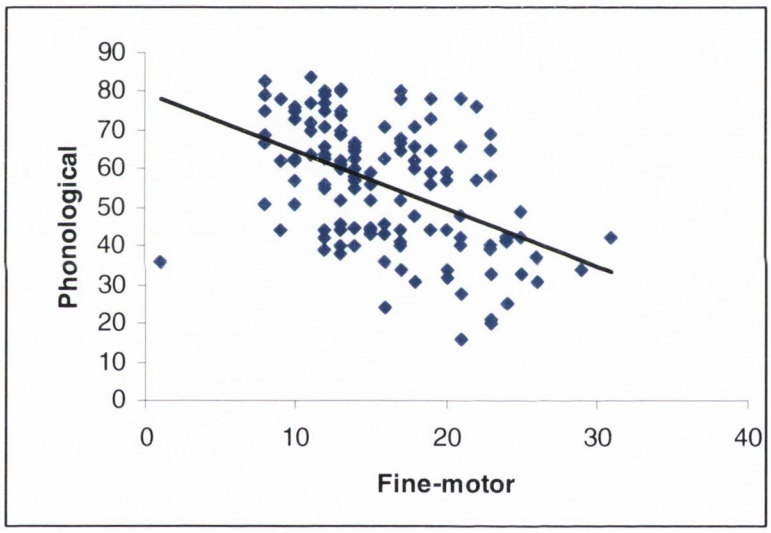

Figure 17: Scattergram showing the relationship between phonological scores and fine-motor scores.

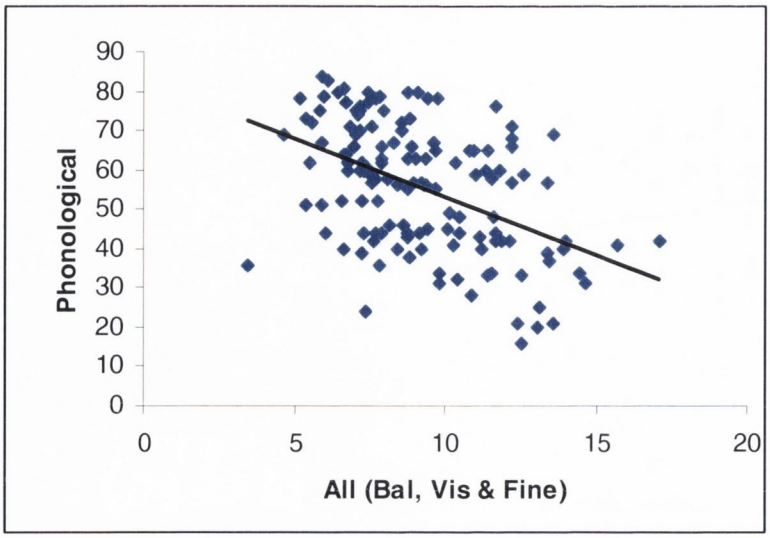

Figure 18: Scattergram showing the relationship between phonological scores and the average of the balance, visual-motor and fine-motor scores.

9.3.4.5.2 Logistic regression where ADHD is the outcome variable (n = 136)

For this sample the entire set of children were included (n = 136). Because there was no a priori reason for suspecting that phonological ability would be related to ADHD this was not included in the set of predictor variables (as it was when the outcome was dyslexia). Furthermore, although the predictor "reflex" was entered first on previous analyses, because of (1) recent claims of its relevance to this population and (2) Nicolson and Fawcett's theory linking cerebellar abnormality to literacy (including reading) via motor and motor-based phonological difficulties, these did not apply to ADHD in this case, so all four predictor variables (reflex, balance, visual-motor and fine-motor) were forcibly entered together in one step.

With only the constant included in the model, 77.2% of cases were correctly classified as either ADHD or not ADHD and this did not change when the set of predictor variables was added. In addition, the percentage of variance explained by the model was only R^2 = 1.4%. The addition of the variables as predictors did not improve the ability of the model to predict ADHD status beyond that of the constant ($p > .05$). An examination of the Wald statistic associated with each predictor variable revealed that none contributed significantly to the ability of the model to predict ADHD status ($p > .05$) (see table 63).

		95% CI for Exp *b*		
	B(SE)	Lower	Exp *b*	Upper
Constant	-2.37 (1.1)			
Reflex	0.11 (0.6)	0.35	1.12	3.56
Balance	0.25 (0.34)	0.66	1.3	2.51
Visuo-motor	-0.23 (0.06)	0.93	0.98	1.14
Fine-motor	-0.3 (0.05)	0.61	1.03	1.0

Table 63: Logistic regression using forced entry with all except the phonological predictor and ADHD as the outcome variable.
Note: Step 1: R^2 = 6.055 (Hosmer & Lemeshow), .015 (Cox & Snell), .023 (Nagelkerke). Model $\chi^2(4) = 2.038$
*p < .05, **p < .01, ***p < .001

9.3.4.5.3 Logistic regression using all predictor variables and reading as the outcome variable where the risk group have very low reading scores (i.e., < 11th percentile)

The sample size for this group was n = 75 because the full sample of those who had a DST score were included (the last use of such criteria removed those in the balance group thereby reducing it to n = 54). The phonological predictor variable was retained in this analysis because the outcome measure was reading and a 2-step model was again employed because of the previously mentioned importance of the reflex variable.

With the constant included in the model, 62.7% of cases were correctly classified and this did not change with the introduction of the first predictor (reflex) in block 1. The addition of the reflex as a predictor did not improve the ability of the model to predict (very poor) reading status (p > .05) and the variable itself did not show any evidence of such predictive efficacy (p > .05). The R^2 calculation showed that 0.24% of the variance in (very poor) reading status was accounted for by the addition of the reflex score to the model.

The percentage of cases correctly classified in block 2 rose by 6.6% to 69.3%. The effect of adding these predictor variables was a significant increase in the ability of the model to predict (very poor) reading status ($p < .05$). The Wald statistic identified only the phonological score ($p < .01$) as a significant predictor of (very poor) reading status. The value of Exp *b* was less than 1 (see table 64), indicating that the risk of (very poor) reading increased as these scores went down (i.e., as this ability worsened). The confidence intervals for this predictor variable did not cross 1 meaning it is generalisable to other samples. The R^2 calculation showed that 11.2% of the variance in (very poor) reading risk was now accounted for by the entire model. The values for these predictors are shown in table 64.

		95% CI for Exp *b*		
	B(SE)	Lower	Exp *b*	Upper
Step 1				
Constant	-1.07 (1.2)			
Reflex	0.31 (0.64)	0.39	1.37	4.8
Step 2				
Constant	5.08 (2.52)			
Reflex	0.82 (0.9)	0.38	2.27	13.46
Balance	-0.16 (0.52)	0.3	0.86	2.38
Visuo-motor	0.02 (0.08)	0.87	1.02	1.2
Fine-motor	-0.08 (0.09)	0.78	0.92	1.09
Phonological	-0.09** (0.03)	0.86	0.92	0.97

Table 64: Logistic regression using (very poor) Reading as the outcome variable.
Note: Step 1: R^2 = 2.18 (Hosmer & Lemeshow), .003 (Cox & Snell), .004 (Nagelkerke). Model $\chi^2(1) = .236$
Step 2: R^2 = 4.44 (Hosmer & Lemeshow), .14 (Cox & Snell), .19 (Nagelkerke). Model $\chi^2(5) = 11.13$
* $p < .05$, ** $p < .01$, ***$p < .001$

9.3.4.5.4 Reflex correlations

Given McPhillips et al.'s (2000) claim that a child's reflex score can be represented by his performance on the ATNR (specifically the Schilder test) two correlations were run to verify this. These were the averages of the following taken from testing time 1:

a. The Schilder v the Reflex scores (including ATNR)
b. The Schilder v Reflex scores (excluding ATNR)

Pearson[62] correlations showed that the Schilder test score average correlated significantly with the full reflex score average ($r = .553$, $p < .001$) which is partially retained when compared to the reflex score average minus any ATNR tests ($r = .251$, $p < .01$). Thus, it is apparent that, to an extent, regardless of whether or not the ATNR tests are removed from the full reflex score, the Schilder test score is related to the other scores. The ATNR test average, when correlated with the reflex test average (minus ATNR tests), showed a similar result ($r = .364$, $p < .001$). The scatter-plots of the first two relationships are shown in Figures 19 and 20.

Despite the significance of these findings, this showed that only about 6% of the variance of (other) reflex scores was predicted by the Schilder test. Therefore, the validity of the claim of McPhillips et al. is open to question.

[62] Non-parametric Spearman tests showed the same levels of significance.

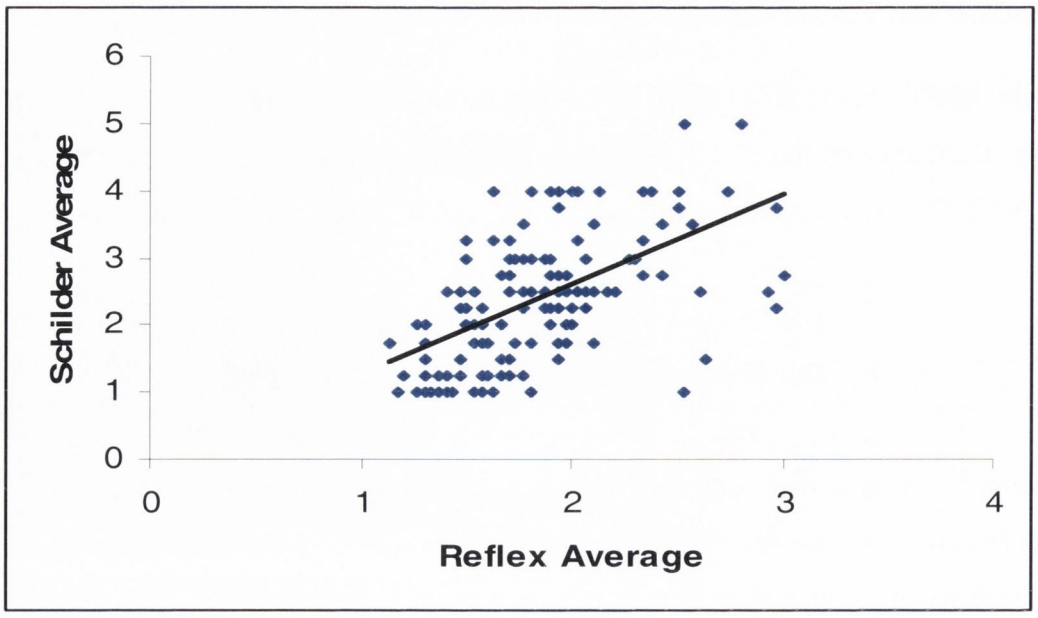

Figure 19: Scatterplot showing the relationship between the Schilder ATNR average and the other reflex score averages (including ATNR).

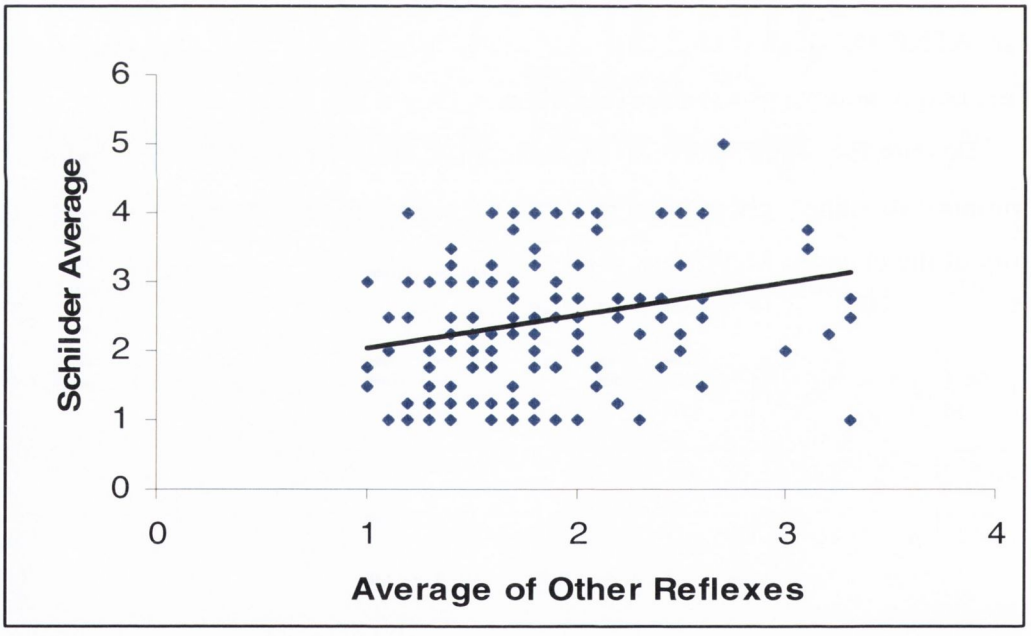

Figure 20: Scatterplot showing the relationship between the Schilder ATNR average and the other reflex score averages (excluding ATNR).

10 Chapter 10: Discussion

The discussion is divided up into five principal sections, consisting of: a review of the major and minor findings, implications of these findings, limitations of the current study, future work / recommendations and conclusions.

10.1 Review of findings

The current work was conducted in order to explore the validity of previous research which suggests that tests of cerebellar (motor) function may be useful in identifying those at risk of dyslexia and that exercise programmes aimed at targeting the cerebellum might alleviate the effects of dyslexia. Such suggestions present a novel and practical approach to dealing with this disorder, and come at a time when the efficacy of current diagnostic methods are themselves heavily criticised. The controversy over the use of IQs and the validity of intelligence tests, coupled with the difficulty of distinguishing between poor reading due to low intelligence and poor reading due to dyslexia in a person of low intelligence, makes this a necessary area of research. A younger sample of children was used here than in previous research because of the need to identify and ameliorate this disorder at as early an age as possible.

10.1.1 Treatment efficacy

Aim 1: To investigate whether a programme designed to integrate retained reflexes into higher order movements reduces the subsequent probability of risk for dyslexia, as measured by the DEST / DST.

Aim 2: To investigate whether stimulating the vestibular system through a programme of gross motor co-ordination reduces the subsequent probability of risk for dyslexia, as measured by the DEST / DST.

Given the strategic design of this study, evaluation of the effects of the experimental treatments is represented best through the chi square analyses, since this compares the

proportions "at risk" for dyslexia in the control groups with those "at risk" in the experimental groups and takes account of the expected distribution of 30-40% of participants at risk in each group. Thus, if a particular treatment was completely effective, we would expect 0% to be "at risk" at time 3 in the treatment group, but 30-40% at risk in the control group(s). The means analysis, of course, is challenged by comparing treatment effects in samples for whom it is expected that there will possibly be little or no effect for 60-70% and, therefore, provides a relatively weak test of the efficacy of the intervention.

A further point is that in order to refine the test of intervention efficacy, the relationship between predictor, and outcome variables, could have been determined exclusively for the control groups. Had reliable predictors emerged from this, only those participants who scored higher on the predictors could be used in the means analysis. However, because few treatment effects emerged from the chi square analyses, the analysis of predictors used the power of the largest possible sample of participants (i.e., all those minus the balance group as these increased significantly in dyslexia risk).

10.1.1.1 Brief review of findings

Balance v Reflex group:

The balance group scored significantly more highly at risk of dyslexia than the reflex group when all children were included in the analysis (n = 136), a finding which was retained when non-programme-compliant children were removed from the sample (n = 118) and when the "private 5" were removed from this sample (n = 113). This difference almost reached significance when the ADHD children were removed from the full sample (n = 105) and did reach significance when the ADHD children were removed from the programme-compliant sample (n = 89). When Reading-only was the outcome variable these groups were also significantly different.

Balance v Control group:

The balance group scored significantly more highly at risk of dyslexia than the Control group when the programme-compliant group were used (n = 118) and when the private 5 were removed from this sample (n = 113). This also approached significance where

the private 5 only were used (n = 88). Where reading-only was the outcome variable, there was also a significant difference between these two groups.

10.1.1.2 Findings

The overall results from the treatment efficacy analyses were disappointing. There were few differences between the groups in any sample and no support for the previously documented efficacy of the type of treatment programmes evaluated here. One clear finding that emerged was the severity of the balance group's dyslexia risk score (on the DEST / DST) and poor reading scores. The balance group's mean score on the DEST / DST was at the level deemed to be "at risk" of dyslexia, something not seen in any of the other groups. Furthermore, their reading scores were below average, unlike the other groups whose reading scores fell in the average range.

Where the full sample (n = 136) was used, the balance group scored at significantly *greater* risk of dyslexia than the reflex group, a finding which remained when the tighter programme-compliant sample was considered (n = 118). Thus, regardless of the amount of programme done there were obvious differences between the groups.

The difference between the balance and reflex group was lost when the 5 private children were removed and when the ADHD children were removed (for both the programme-compliant and the full sample). For the former of these, the only change in DEST / DST mean scores was a small rise in the mean at-risk score of the reflex group (from .62 to .65) which was sufficient to result in a non-significant difference between it and the balance group. Nevertheless, as mentioned previously, it suggests that the presence of those 5 children's DEST / DST scores served to lower the dyslexia risk when included, which could be interpreted as support for the dyslexia risk-reducing effects of the reflex programme when applied professionally or privately. However, no difference was found between the reflex and control (including placebo control) groups when these 5 children were isolated to form one, small reflex group. Hence, although the small sample seriously reduced the power of the test, the idea that the reflex programme has dyslexia risk-reducing effects was not supported here.

The removal of the ADHD individuals from these samples resulted in a decrease in mean DEST / DST scores for each of the groups. This suggests that ADHD

is associated with being at risk for dyslexia. However, despite this there were no differences between any of the groups where only the ADHD sample was analysed, either when the full sample or the programme-compliant sample was taken into consideration.

Among the programme-compliant children, a significant difference between the balance and control group also emerged. Although all of the mean dyslexia risk scores went up for the programme groups who were compliant, the balance group increased in dyslexia risk by 1 full point compared, to .4 and .1 for the reflex and placebo groups respectively. This could suggest that the more of their programme the children in the balance group did, the more highly "at risk" of dyslexia they became compared to the control group. If this was the case, it is hardly an encouraging finding, possibly implying a deleterious effect of participating in such a programme. It is possible, however, to interpret this as those children with the greatest difficulty persisting with their programme more so than those with less difficulty.

Although there was no DEST / DST pre-measure (due to the young age and the attention capacity of the children), the initial random allocation of children to groups would suggest that, prior to treatment, DEST / DST scores should be similar across groups. Although there was some artificial re-assignment, this was from the experimental groups into the control group. Had the control group scored significantly higher on this measure than the experimental groups, this re-assignment might hypothetically have accounted for some of that difference (with the explanation that such children come from families who are less motivated or disciplined in terms of actively promoting their child's abilities or tackling their weaknesses). The finding that the balance group were scoring, on average, at higher risk for dyslexia than the other groups would therefore suggest that the content of the balance programme is not effective in terms of reducing the child's risk of dyslexia, as measured by the DEST / DST. The lack of evidence for a significant difference between the reflex group and the control group suggests that "fetal" type movements – slow repetition of retained reflexes - are equally ineffective in reducing children's risk for dyslexia.

The higher "at risk" score for dyslexia in the balance group could be explained by a diversion of child and family attention from direct reading / academic home pursuits to one which has no *direct* bearing on literacy improvement, particularly because there is

no difference between this group's dyslexia risk and that of the placebo control group, which in principle involves a similar diversion of activity. However, there was a significant difference between the balance and the reflex group even though the latter also directed attention away from direct reading instruction / practice. This is not support in favour of the reflex programme, however, because the latter does not differ significantly from either the control group or the placebo-control group.

It cannot be said that the results are due to any qualitative differences between how well the groups engaged in their programme. When questioned about the amount of time spent on the programme daily and the degree of control / effort put into it, there were no statistical differences across the programmes.

The fact that there were no differences in the amount of time, or quality of performance, between those who were at risk for dyslexia and those who were not, suggests that the quality of execution of programme did not affect dyslexia status. By and large the findings were the same for those with and without ADHD although there was a statistically significant negative relationship between quality of programme performance and ADHD. Higher frequency of ADHD was associated with the programme movements being more rushed. This makes intuitive sense given the nature of ADHD. Correlations such as these do not allow for interpretations about causality, so it is not possible to say whether presence of ADHD in those who ultimately showed it, prevented them from doing the programme properly or whether not doing the programme properly resulted in ADHD being maintained.

Finally, all of the significant differences concerned one dependent variable (i.e., "dyslexia risk"). Although this was the major dependent variable in the study there were also five minor dependent variables (level of retained reflexes, balance skills, phonological skills, visual-motor skills and fine-motor skills). It is striking, and somewhat curious, that there was no change in any of these, especially in the reflex and balance scores over time, given that the two experimental programmes studied were designed around these variables. Although possible, it is improbable that the programmes employed were not sufficiently well-designed in order to improve such skills because they were based entirely on the relevant literature. It is more likely that such programmes in general either do not do what they claim to do (i.e., reduce retained reflexes, improve vestibular functioning) or they do not do so beyond the

extent to which typical childhood life-styles allow. As seen earlier, there was no significant difference between groups in the amount of (other) extra-curricular or sports activities engaged in, so it is possible that the extra 10 minutes per day required by such programmes is a relatively small and inconsequential addition. Thus, if such programmes are having no discernable affect on the very skills they are designed to improve, it is hardly surprising that dyslexia or reading scores are not improved in these groups.

10.1.1.3 Summary

To sum up the treatment efficacy of the two programmes explored in this study, there is no direct evidence to support the previous finding (i.e., McPhillips et al., 2000) that a programme designed to integrate retained reflexes into higher order movements reduces the subsequent probability of risk for dyslexia, as measured by the DEST / DST. There is not even any evidence in the current study to suggest that such a programme reduces retention of reflexes themselves significantly more than in the control groups (where scores were measured under "blind" conditions, and inter-rater reliability with another blind scorer, was high).

Similarly, there is no direct evidence to support the previous finding (i.e., Reynolds et al., 2003) that a balance programme based on vestibular stimulation (designed to target the cerebellum) reduces the subsequent probability of risk for dyslexia, as measured by the DEST / DST. As with the previous finding, there was no evidence to suggest that such a programme improves performance on general balance tasks significantly more than in the control groups.

Although both the reflex and the balance programme were supposed to target the cerebellum there is no evidence that other measures of cerebellar functioning (e.g., fine-motor skills, visual- motor skills) improved as a result of either programme.

10.1.2 Predictive efficacy

Aim 3: To investigate whether retained reflexes can discriminate between dyslexic and non-dyslexic children by predicting which children of a pre- or early-school sample will subsequently score "at risk" for dyslexia on the basis of the DEST / DST.

Aim 4: To investigate whether vestibular functioning, as measured by a battery of gross motor co-ordination tests (i.e., balance), can discriminate between dyslexic and non-dyslexic children by predicting which children of a pre- or early-school sample will subsequently score at risk for dyslexia on the basis of the DEST / DST.

Aim 5: To investigate whether fine-motor skills (i.e., "soft cerebellar signs") can discriminate between dyslexic and non-dyslexic children by predicting which children of a pre- or early-school sample will subsequently score at risk for dyslexia on the basis of the DEST / DST.

Aim 6: To investigate whether visuo-motor skills can discriminate between dyslexic and non-dyslexic children by predicting which children of a pre- or early-school sample will subsequently score at risk for dyslexia on the basis of the DEST / DST.

Aim 7: To investigate whether phonological skills can discriminate between dyslexic and non-dyslexic children by predicting which children of a pre- or early-school sample will subsequently score at risk for dyslexia on the basis of the DEST / DST.

Aim 8: To investigate whether tests of phonological skills are related to performance on cerebellar tests in order to test Liberman and Mattingly's (1985) motor theory of speech as incorporated in Nicolson and Fawcett's (1990) cerebellar deficit hypothesis.

10.1.2.1 Brief review of findings

The predictive efficacy of the individual test variables was generally poor. Either they were not significant as predictors, or where they were significant, the direction of the relationship matched that suggested by the theoretical literature on only a percentage of occasions. Consistent with the theoretical literature, there was one predictor which

significantly predicted the outcome in half of the analyses and did so in the suggested direction on all of these occasions. This was phonological ability.

10.1.2.2 Findings

With the exception of the phonological variable, examination of the individual predictor variables (in step 2) did not inspire much confidence in terms of using them to predict dyslexia score or status. Although many of them did show significant relationships with the outcome variables, the nature of this relationship often did not make sense or was inconsistent across different types of model. Individual predictor variables showed a significant relationship with the outcome variable on 17 out of 60 analyses; of these, only 11 were in the direction which the literature would have suggested (i.e., approximately 17%).

Models: Making use of the variables in one combined model was more useful for making predictions. This may reflect the fact that Step 1, represented by the initial (full) reflex or Schilder variable, accounted for only a *tiny* percentage of the variance of any of the outcome variables, thereby making it easy for the addition of other predictor variables to significantly improve upon this, particularly as they contained the phonological variable (percentage improvement is shown in Table 65). Regardless of whether the model was predicting scores, as when multiple regression was employed, or whether it was predicting status, as when logistic regression was employed, the full model was useful in predicting reading outcome, whether groups were divided according to above and below average performances or whether the "poor" category was particularly severe (i.e., <=11th percentile). With the addition of the full set of predictors, the outcome of "reading" had a bigger increase in variance explanation and classification ability than any of the other outcome variables. The variance percentage in reading explained by the model (where full "reflex" score is the entered in step 1) reached 61.5% and correctly classified 92.2% of the sample[63], which represented an improvement of 25.5% and 60.97% respectively compared to pre-step 1 (see Table 65). In general it made little difference to predictive efficacy whether the full reflex score or

[63] Percentage correctly classified ranges from 62.7% to 81.3% before Step 1, up to a range of 69.3% to 92.2% after Step 2.

the Schilder reflex score only was entered first. Entering the Schilder reflex score first had one advantage over the use of the full reflex score in predicting dyslexia status (using the full DEST / DST). However, both set of reflexes were equally good when dyslexia status was expanded to include those at "mild risk" of dyslexia.

For the most part, as the model improved in the amount of outcome variance that it explained, it also improved in its ability to correctly classify groups. This can be seen in Table 65 where, with the exception of the second and fourth rows, the rank of improvement for each analysis was the same (e.g., row one shows the smallest increase in terms of both predicted outcome variance and correct classification, whereas the last row shows the biggest increase in percentages for both).

1ST step variable / Outcome variable	Variance (% explained)			Classification (% correctly classified)		
	Pre	*Post*	*Difference*	*Pre*	*Post*	*Difference*
All entered / ADHD	N.A.	1.4%	**1.4%**	77.2%	77.2%	**0%**
Schilder / DESTandDST	.05%	22.2%	**22.45%**	81.3%	81.3%	**0%**
Full reflex / DESTandDST	1.4%	22.4%	**21%**	81.3%	83.5%	**2.2%**
All entered / Poor Reading	.24%	11.2%	**10.96%**	62.7%	69.3%	**6.6%**
Schilder / Reading	6.55%	34.5%	**27.95%**	66.7%	76.5%	**9.8%**
Full reflex / mild DESTandDST	1.4%	40%	**38.6%**	70.3%	84.6%	**14.3%**
Schilder / mild DESTandDST	.8%	42.7%	**41.9%**	71.1%	86.7%	**15.6%**
Full reflex / Reading	.53%	61.5%	**60.97%**	66.7%	92.2%	**25.5%**

Table 65: The percentages of (1) Explained outcome variance, (2) Correctly classified sample, divided into "pre" (i.e., before introduction of any of the variables), "post" (after introduction of all variables) and "difference" (difference between the two) using logistic regression. For the most part samples are presented in order of percentage increase.

10.1.2.3 Generalisability

Any predictive efficacy seen here is limited to the sample employed in this study. Whilst findings suggested predictive efficacy of some variables and models, multiple regression techniques suggested that this was not generalisable to other random samples or the true population. Thus, the reliability of the findings meant they must be treated with caution. This is especially so given that 83% of all the significant relationships yielded by the multiple regression analyses suggested directions which did not fit that suggested by the literature.

10.1.2.4 Summary

The reflex tests showed no ability to predict dyslexia score or status as measured by the DEST / DST. They did show some efficacy in predicting reading when it was the outcome variable, regardless of the method of regression used (i.e., multiple or logistic), and both of these relationships were positive; that is, the less evidence there was of retained reflexes, the better the reading score. The Schilder test predicted reading ability but suggested a negative relationship; the less evidence there was of a retained Schilder reflex, the more likely it was for reading to be classed as below average.

In their study McPhillips et al. (2000) claimed to find evidence to support the link between reading difficulties and control of movement, however, they failed to explore the prevalence of retained reflexes (the measurements of which were defined by them) in the normal population. Furthermore, it is impossible from their study to know if the presence of such reflexes is common across the dyslexic population because it did not include any dyslexics without retained reflexes. The authors claimed that they could not find any such dyslexics, but whether this is a result of sample bias (which they themselves suggest as a possibility) or reflects the fact that they do not exist, we cannot know. The current study found some support for the predictive utility

of (full) reflex scores in predicting those who scored above and below average at reading, although there was no evidence to support that it was useful for predicting dyslexia status or score, as measured by the DEST or DST. It could be that this relationship between reflex and reading is a result of these children's general development or ability. For example, those who are less intelligent may be both less likely to pick up reading easily and less likely to sustain the slow and careful movements involved in reflex testing or perhaps less likely to understand or attend to the instructions given to them. If this is the case, most tests could probably be used to identify such children, not least those of phonological ability. Thus, the use of evidence of retained reflexes as a tool for identifying those with dyslexia would be premature and indeed by 2004 there appeared to be a little more caution with regard to claims about persistent primary reflexes as *causes* of reading difficulties (McPhillips and Sheehy, 2004, p.317).

It is important also to note that this study shows that the Schilder tests generally failed to predict reading status, and the direction of the relationship in the logistic regression analysis was opposite to that suggested by McPhillips et al. This does not lend any support to their claims, therefore, particularly when combined with studies by those such as Hellebrandt et al. (1962) who claimed to find evidence of the ATNR in normal children and adults when tested in the quadruped position. Parr et al. (1974) also claimed that the ATNR was normal in children between the ages of 3 and 9 years. It has to be concluded, therefore, that the evidence for dyslexics' problems resulting from retained reflexes is at best inconsistent and is certainly inconclusive.

Balance was useful in predicting dyslexia status, as measured by the DEST / DST; this relationship suggests that as balance ability worsened, dyslexia was more likely. There was also a significant, negative relationship between balance and reading which is not in accordance with the literature. This suggested that as balance scores improved, reading scores worsened. This is a difficult finding to explain. It is possible to envisage, however, that certain temperaments might lead to differing pursuits amongst children. For example, those who engage in more active pursuits may put themselves in a position whereby they are regularly practising balance-type skills at the expense of reading related skills, where-as those who have other pass-times such as reading, watching television or playing cognitive games might be improving their reading, attention and symbol representation, but not their balance skills.

Fine-motor skills did have a positive relationship with dyslexia status (when this was expanded to include mild cases). Although this measure also showed predictive efficacy for reading, the relationships were negative for both sets of regression analyses, suggesting that as fine-motor ability improved, reading ability worsened. This is not in accordance with the literature and would be hard to account for theoretically.

Visual-motor skills appeared to predict dyslexia status when the mild cases were included, but the relationship in both cases (i.e., when full reflex and Schilder reflexes were the first entry), were negative. That is, as visual-motor scores improved, dyslexia became more likely. This too is hard to account for theoretically.

A positive relationship between phonological ability and dyslexia score was apparent when dyslexia outcome was expanded to include those at mild risk of the condition but not when the higher risk criterion was used. As previously mentioned, phonological ability also showed a positive relationship with reading, such that as phonological ability improved, poor reading was less likely.

There were positive relationships between phonological ability and three of the measures taken of cerebellar functioning. These three measures were combined to form an average and this too correlated positively with the phonological scores. The amount of shared variance between phonological ability and each of the other four variables was: balance, 16%; visual-motor, 6%; fine-motor, 25% and combined, 25%. Thus, there is some shared variance between these factors, which provides some evidence in support of the motor theory of speech.

The issues of predictive and treatment efficacy, as well as the implications for causal theories, have been discussed and it can be seen from these results that replicating previous findings has not been easy. Although there was some evidence for relationships between dyslexia or reading status and reflex, balance or fine-motor scores they did not inspire much confidence. Even less confidence is generated by the fact that not only did reflex and balance programmes have no apparent efficacy in reducing dyslexia risk or improving reading scores but that those groups showed no better performance on either reflex or balance scores at session 3. The regression

techniques employed warn against generalising these findings either to other random samples or to the true population and so it cannot be concluded that such measures would be of practical use.

10.2 Implications

10.2.1 Treatments

10.2.1.1 Reynolds et al., 2003: Balance programme

Reynolds et al. report that the balance group in their study showed significant gains above those of their control group on various cognitive skills which might contribute to reading (i.e., dexterity, verbal fluency and semantic fluency) as well as on reading itself. Scores on national standardised achievement tests showed significant improvements in terms of reading gain compared to the previous year. The current study could not attempt to replicate these exact findings because the sample was younger, some had not yet started school and the country of testing (Ireland) does not have annual national tests. However, an attempt to explore the effect of such a programme on DEST / DST functioning was possible and this was done in a way which addressed many of the previous criticisms of the original study (e.g., scoring was blind, there was a placebo control).

As previously mentioned, the scores of those in the balance group were high enough to be considered seriously at risk for dyslexia and significantly higher than those of the control group. Two explanations of this finding are possible: the sample assigned to the balance group were particularly at risk to begin with, or the programme contributed to this score. The former of these is unlikely due to the random assignment of children to groups, however, if it is the case, then the balance programme did not serve to reduce the "at risk" score to a desirable level, and thus, questions should be raised about the clinical efficacy of the programme (as distinct from its statistical reliability). If the latter is the case, it suggests that consideration should be given to abandoning such programmes out of consideration for the harm they could be doing. However, there is also no significant difference between the balance and the placebo-control group, which suggests that there is no specific positive or negative effect of a programme designed to target the cerebellum via vestibular stimulation.

Alternatively, the "at risk" outcome of the balance group could be due to the reassignment of some children from the balance group to the control group. These children could have been those with the least severe difficulties, and therefore, least parental motivation to engage in the programme. In this case, the balance group would be left with a greater ratio of more severely affected children than the control group. In reality this is unlikely to be the case for several reasons. First, there was no difference between the reflex group or the placebo group and the control groups which would have been subject to similar severity and motivation mixes. Second, of the three programmes to be engaged in, the balance one was the least difficult to implement and the most fun one to do; it is more likely that parents and children would have abandoned the other two programmes. Third, there was no statistical difference between the mean DEST / DST scores of those who left the balance group compared to those who remained in it.

Why the balance group should be associated with such high scores remains a mystery. It is unlikely that the time spent on such programmes detracted from more relevant learning because the placebo-control was not significantly different from the control, nor was the reflex group and yet these children spent similar amounts of time redirecting their attention to complementary programmes. Is it possible that the more active, boisterous nature of the balance programme (e.g., running, jumping, twirling) somehow affected the children's hyperactivity or concentration levels? Specific analyses of DEST / DST component tests was not undertaken, with the exception of reading, and so tasks designed to test working memory (e.g., rapid automatised naming, digit span) were not explored for differences across groups. However, there were no differences between the groups on measures of ADHD.

In their study of DDAT treatment efficacy, Reynolds et al. explored 4 different issues of relevance to this discussion.

1. Incidence of cerebellar / vestibular symptoms

The tests used to predict dyslexia status were administered on first contact with the child. Although incidence cannot be reported upon, the fact that these tests did not reliably discriminate between which groups were later classed as dyslexic and which were not, suggests that that there are not significant differences between the two populations. This supports McPhillips and Sheehy's (2004) finding that there

was no difference between dyslexics and non-dyslexics on measures of motor performance, which included balance tasks.

2. Effectiveness of DDAT intervention on cerebellar / vestibular symptoms

Although Reynolds et al. report improvements on various cerebellar / vestibular tests (i.e., eye-tracking and balance) within their experimental group, the improvements here were not significantly greater than those of the control group. The current study failed to show any significant differences between the balance group and either of the control groups on measures of cerebellar tasks, including; fine-motor coordination, visual-motor coordination and most of all, gross-motor coordination (i.e., balance). Thus, a programme based on balance tasks failed to change balance ability.

3. "Near transfer" to fundamental cognitive skills

4. "Far transfer" to literacy

In considering (3) and (4) above, any change in performance on outcome variables where a novel treatment is in use, should be judged in light of comparisons with a placebo group. This was not done by Reynolds et al; so although changes were evident in their study, it cannot be said with any certainty that this was due to the specific nature of their programme. The current study did employ a placebo-control and found that on measures of both dyslexia generally (using the DEST or DST) and on the subtest measure of reading, there were no differences between the balance and placebo-control groups.

The response in the scientific literature as a result of the Reynolds et al. study was critical (e.g., Rack et al., 2003; Snowling and Hulme, 2003). Such responses focussed on the methodological and statistical shortcomings of the study and what these meant for interpretation of findings, with a consequential concern over the misleading impression given to the public (see Snowling and Hulme, 2003). The current study's inability to replicate findings, whilst nevertheless employing sounder methodology, suggests that these criticisms were justified, and there is no case for the DDAT programme to claim that cerebellar or reading changes in children are a result of any specific aspect of their programme.

10.2.1.2 McPhillips et al. (2000): Reflex replication programme

The current study can offer no support for McPhillips et al.'s (2000) findings. Although the reflex group did not score at such high risk for dyslexia as those in the balance group, they did not differ significantly from those in either of the control groups particularly in the chi square analyses. As we have seen, the DEST / DST scores did reduce when only those engaging in the private versions of the reflex therapy were considered (i.e., reflex group n = 5; from .62 to .39 on the DEST / DST) and although there is no significant difference between these 5 children and the control group mean scores, the reduction itself is interesting. Although it is impossible to know what might happen with a larger sample, it could it be that, in this sample, the 5 children from the private versions of the therapy come from more motivated families. Given that the parents were already aware of their child's involvement in a programme (either in school or privately paid for by themselves), yet they also volunteered their child for inclusion in a university-based study, it seems plausible that they are the ones exposing their children to as much intervention as possible. Thus, lowered risk scores in this group could be due to the intense scrutiny put on the child, or inherited / learned motivational skills learned from the parent or the result of some uncontrolled variable (such as private tuition or receipt of resource hours in school). It seems unlikely that the location of the intervention should affect outcome; advantages in one location could be cancelled out by disadvantages in another. For example, it could be argued that children are more likely to comply with their teachers' instruction if the rest of the class is conforming than they might in the home where it might be easier to ignore or reject parents' request for compliance. On the other hand, it could be argued that parents are more able and likely to ensure that the correct movements are performed, given that they only have one child to monitor.

10.2.1.3 Treatment efficacy: Conclusion

One salient difference between the current study and that conducted by McPhillips et al. and Reynolds et al., was the age of the sample studies. The original McPhillips et al. study employed 8-11 year old children and the Reynolds et al. study 8-10 years olds, where-as the current study employed 3-7 year old children. A principal difference between their groups and the groups studied in this experiment was the quantity of their

respective prior exposure to language. Thus, it could be argued that older children somehow made use of their programme in a way that the younger ones could not. This allows the current study to conclude that treatment of cerebellar symptoms does not reduce dyslexia risk in *young* children, without necessarily challenging the effect it has on older children.

Cratty (1974; 1979) is reserved when it comes to the effects of motor programmes in general. His research with mentally retarded children showed changes in a variety of areas after the implementation of exercise programmes, but that one of the factors contributing to this included lower range IQ (i.e., <65). In addition, the kinds of positive outcomes included such things as improved vocational opportunities, improved social interactions due to improved "play" skills, reduction in hyperactivity due to a reduction of excess muscular tension, improved self-control and pacing as well as self-concept. These are relatively modest claims and seldom include rises in IQ or academic functioning.

It can only be concluded here that those either at risk of dyslexia, or showing early signs of the disorder, are possibly better off sticking to conventional teaching methods to enhance and practise directly their literacy skills. Despite the time-consuming nature and frustration that such methods may involve, the current findings suggest that these challenges will only be delayed, not avoided, if motor-treatments are exclusively pursued. This echoes the conclusion made by Mattson (1983) who claimed that such motor programmes deny the child proper remediation of his condition.

The finding that those with ADHD were less likely to comply with control of their programme movements and more likely to rush through them may be because the presence of their ADHD prevented them from adequately attending to and concentrating on what they were supposed to be doing. This is a finding which makes sense intuitively. However, it does not seem to have been a consideration of those organisations who offer motor programmes to such children. It would appear from this evidence however, that the reflex is a poor choice of intervention for children with ADHD.

10.2.2 Tests

The current study showed that although full reflex scores could not be used to predict dyslexia status / scores, they could be used to predict reading ability. Although this might initially seem to support McPhillips and Sheehy's (2004) findings, the Schilder ATNR score, when taken in isolation, had a relationship in the opposite direction to that suggested by McPhillips et al. (2000). This supports the conclusion of McEvoy (2002) failed to support McPhillips et al.'s (2000) finding that the Schilder ATNR test could be used to discriminate between dyslexic and non-dyslexic samples and also reported a weak inverse relationship between ATNR and dyslexia. As the Schilder ATNR could not discriminate between the dyslexic and non-dyslexic groups in the current study, it appears that the suggestion made some time ago by Parmenter (1975) that such reflexes are present in all children, not just those with learning difficulties, is possibly supported.

Furthermore, the current study showed that although there was a significant correlation between the Schilder ATNR score and the "other" reflex tests (i.e., STNR, TLR, Moro), the amount of shared variance that the Schilder ATNR had with the STNR, TLR & Moro was only about 6%. This adds weight to another of McEvoy's (2002) findings which found that the Schilder ATNR and the Ayres ATNR were not significantly correlated with each other. Crutchfield and Barnes (1993, p.166) clearly state that one retained reflex cannot promote or retard development and is unlikely to act as a good predictor of anything by itself. For this reason a cluster of reflexes must always be measured. Given the small amount of variance shared by the Schilder ATNR and other reflexes, cautious interpretation should be employed where one test is used to make gross interpretations about group characteristics or group change over time.

There are many different organisations (Appendix 2) and professions (e.g., teachers, psychologists, physiotherapists, physical therapists, occupational therapists, paediatricians) using such testing methods, particularly retained reflexes, and it is not known how consistently movements are measured across such a variety of users. The possibility of interpersonal and inter-agency differences emerging over time is quite likely given the observation of reflex elicitation differences between some of these groups as well as problems observed with some of the tests (see "Limitations" for a further discussion of this).

10.2.3 Theoretical implications

10.2.3.1 Neuro-maturational theory

Both Reynolds et al. (2003) and McPhillips et al. (2000) claimed that their respective programmes were effective in improving balance or reducing retained reflexes (depending on the movement programme used) in the experimental groups. In the former study they refer to the improvement in cerebellar / vestibular symptoms, as measured by posturography[64] and electronystagmography[65] and in the latter study they refer to the improvement in the high prevalence of persistent ATNR. Neither finding was supported when the current study explored the treatment efficacy of each programme on a variety of outcomes, including cerebellar / vestibular symptoms, as measured by fine-motor, visual-motor, gross-motor (i.e., balance) and even reflex scores. Either the programme may have worked in terms of "targeting the cerebellum" and the tests did not pick it up, or the tests did measure cerebellar functioning and there was no change. Whichever the case, it appears that either the treatment or the tests did not perform the function they were supposed to. This has quite profound implications for the "Neuro-maturational theory" which sparked off this line of research (e.g., Gesell, 1938; Delacato, 1959, 1966; Blythe and McGlown, 1979). The ideas have not stood up to scientific scrutiny in the past (e.g., Zigler and Seitz, 1975; Robbins, 1966a, 1966b; O'Donnell and Eisenson, 1969; Jackson and Dando, 1976; Sparrow and Zigler, 1978; Fagan et al., 1988) and this study fails to provide any support for them now. This provides further evidence against the neuro-maturational theory which should be re-examined in conjunction with subsequent challenging evidence such as that presented below.

The neuro-maturational / reflex theory of development, is based essentially on a hierarchical view of motor development, suggesting that hard-wired patterns determine the performance of movements in the form of reflexes. It has been accused of being overly simplistic, linear and uni-directional. A further weakness of such theories is that they don't properly describe how one level of performance gradually leads to another,

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[64] As measured on tests of sensory organisation, motor control and (motor) adaptation.
[65] Smooth pursuit, saccade latency / accuracy, optokinetic flow.

neither do they allow for the influence of the environment on motor development (Crutchfield and Barnes, 1993).

Thelan et al. (1981) suggested that muscles work together in patterns according to a pattern generator and particular patterns were invoked by the task at hand rather than simply being stimulated by prenatally determined muscular relationships. She challenged previously accepted ideas of motor development by demonstrating that movements were not necessarily or merely a direct response to stimuli, but that infants could in fact engage in purposeful and spontaneous movements (e.g., leg kicking; Thelan and Fisher, 1983a) and that such movements were related to other factors such as the infant's level of arousal (Thelan and Fogel, 1989). Thus, the infant does have some level of control over movements, and this represents a step in the direction of ultimate control. The predominant activity of the legs also challenged the cephalo-caudal theory of directional development because stereotypies of the legs and feet begin at just 4 weeks of age, earlier than arms or torso (Thelan and Fisher, 1983b); the proximo-distal theory of development was similarly questioned when she observed torso stereotypies occurring after arm stereotypies (Thelan, 1979). Cephalo-caudal directional development has also been dismissed by Prechtl (1986) in his ultrasound studies of fetal development.

Thelen and Fisher (1982) also suggested that movement performance depended on many different things, such as body mass, length, centre of inertia etc. For example, neuro-maturational theory suggested that the stepping reflex disappeared a few months after birth. However, Thelen and Fisher demonstrated that it was not eliminated at all, but simply prevented by the child's weight when it was that much heavier. Lying prone in water or on the floor, the infant would still be able to perform the movement. These factors, combined with the constant experience of "fitting movements to tasks", make the development of motor skills in the human infant an interactive process and not one which is formed by one simple top-down or bottom-up process. The idea that certain parts of the brain were of dominating importance in terms of development may also have been naïve and simplistic in its interpretation of how the brain works. The dense interconnection of perceptual, motor and cognitive processes mean that neurones can be activated in an almost infinite number of ways (Thelan, 2000).

10.2.3.2 Cerebellar Deficit Hypothesis (Nicolson et al., 2001)

As Newton (1985) points out, the neuro-maturational theory did not attempt to explain how planning, initiating or learning movements could occur. The cerebellar deficit hypothesis does allow for this and is more focussed upon the inter-relationships of centres to allow for movements. Memory of situations, plus internal and external information are all sent to the CNS from the sensory receptors and are then processed according to a motor goal which may be adjusted using both feed-forward information (e.g., taking information and sending it onwards) and feed-backward information (e.g., comparing execution to an "ideal" movement, in order to "adjust" the movement).

The theory also incorporates the motor theory of speech (Liberman and Mattingly, 1985) as an explanation for dyslexia, suggesting that a general motor problem would lead to a speech motor deficit which would itself result in rehearsal and articulation problems thereby contributing to a poor working memory and poor phonemic awareness. The finding in the current study that phonological skills correlated with different kinds of cerebellar tasks (i.e., fine-motor, visual-motor and balance tasks), with solid amounts of shared outcome variance (up to 25%), provide some initial support for the motor theory of speech. As previously mentioned, in order to provide more solid exploration of this theory, it would be useful to know whether those infants who had problems matching aurally perceived phonetic information to face / voice were the same ones who later developed dyslexic difficulties. Whether evidence of delayed phonological skill development represents an abnormality in the cerebellum is another matter. Certainly the cerebellum is involved in motor tasks, as we have seen, but the lack of consistent and clear results makes the causal link between the two less strong. Whilst some support was found for a positive predictive relationship between balance skill and dyslexia outcome (and fine-motor skill and dyslexia outcome), as measured by the DEST / DST, a negative relationship between balance and reading, as well as fine-motor skill and reading, confuses the issue. From a practical point of view, reading is the construct of interest when discussing dyslexia, and so this also needs to be clearly accounted for in any theory or findings. To suggest that reading scores will improve if balance skills worsen is counter to the argument made by Reynolds et al. (2003) in their support of the DDAT treatment.

It is apparent that a strong link exists between phonological ability and reading ability both from the literature generally and from the current study. Given the support

for the phonological deficit hypothesis as a descriptive theory for dyslexia, it is, therefore, surprising that phonological scores did not clearly identify those with (severe) dyslexia. This suggests that dyslexia is more than just a problem with reading or a problem with phonological decoding and that other measures are necessary in order to help identify this specific group. From the current findings a causal link (as opposed to simple association) between measures of cerebellar function and risk for dyslexia cannot be determined and thus it cannot be concluded that cerebellar deficiencies are a necessary and sufficient explanation of dyslexia.

In response to Fawcett and Nicolson's (1999) suggestion that it could be the more severe dyslexics who score poorly on cerebellar tests there was also no support. Their studies found consistent evidence of a correlation between dyslexia and poor performance on cerebellar tasks but they always used strongly dyslexic samples. Four samples were used to explore this in the current study: one with the higher cut-off point on the DEST (i.e., 0.9) to classify those who were at risk for dyslexia; one using the lower cut-off on the DEST (i.e., 0.6) to classify those at mild risk of dyslexia; one using reading outcome with the cut-off set at "below average" and one using reading outcome with the cut-off set at a lower percentile (i.e., at or below the 11th percentile). These analyses did not support Fawcett and Nicolson's theory as there were more significant individual predictors when the criteria were less stringent than when they were more so and, as we have seen, even these were inconsistent and often the relationship was in the opposite direction to that required to support the theory. Despite the methodological rigour employed by the Sheffield team, it is important to remember that most of their studies involved the same cohort of dyslexic children, recruited in their early teens and followed for over 10 years. The existence of sample exhaustion, sample compliance or even some behavioural anomaly within this relatively small group may be possible given the difficulty in replicating the Sheffield findings. The current study would suggest one of several things:

1. The cerebellar deficit hypothesis is not a valid explanation for dyslexia.
2. The tests used in the current study were not sensitive enough to pick up cerebellar abnormality.

3. There cerebellar deficit hypothesis goes some way towards explaining the causes of dyslexia but is not a complete enough theory to allow for reliable identification of dyslexics in this way.

Zeffiro and Eden (2001) suggest that the cerebellum may even be "an innocent bystander". Because of the number of complex circuits within which the cerebellum plays a part, they suggest that it is possible for damage to pre-cerebellar parts of these circuits to mimic cerebellar dysfunction.

10.2.3.3 Phonological Deficit Hypothesis

It has long been shown that phonological skill is a good predictor of future reading ability and of dyslexia, and the idea of a deficit in the phonemic awareness of language is not controversial. Thus, it is encouraging that, even given the limitations of the current study, phonological ability comes through as a solid predictor of dyslexia score and status and that its relationship with the dependent variables (be it dyslexia or reading) is in a theoretically consistent direction (i.e., as phonological ability improves the outcome is more promising). This finding adds further weight to the literature supporting a deficit in phonological awareness in those at risk of dyslexia and the early predictive use of such tests for dyslexia (e.g., Scarborough, 1990). Given the previously discussed studies which claim to have found differences between speech perception abilities of dyslexia "at risk" and control infants (i.e., Richardson et al., 2003; Paavo et al., 2002), the possibility of identifying, monitoring and even remediating at risk children as early as 6 months old presents an exciting and worthy line of research..

10.2.3.4 ADHD literature

Given previous research claiming that it is ADHD, and not dyslexia, which accounts for apparent motor (or cerebellar) differences between dyslexic and normal samples, those with ADHD and dyslexia were analysed"[66]. The lack of support for any treatment efficacy amongst these groups under either condition (i.e., the full sample or the programme-compliant sample) as well as the lack of evidence for motor / cerebellar

[66] It was not possible to isolate "pure ADHD" children because the samples would have been too small.

tests to predict those at risk of ADHD does not lend support to either the suggestion that cerebellar deficiencies are a marker of ADHD or that balance and reflex-based programmes are useful for reducing risk of ADHD compared to control and placebo-controlled groups. Thus, although the tests used were not exactly the same, the claims made by Denckla et al. (1985) and Raberger and Wimmer (2003) of the use of balance and other motor tests to identify those with ADHD was not supported. The findings neither lend support to the many centres offering relief from vaguely defined and general "neuro-developmental disorders" by prescribing such programmes (Appendix 2).

10.2.3.5 Definition of dyslexia

The theory of retained reflex importance and poor balance skills presented an exciting possibility for a behavioural test for dyslexia which might discriminate between dyslexic children of low IQ and general poor readers of low IQ. However, although the measures used in the current study claimed to target causal[67] aspects of dyslexia (i.e., children's cerebellar functioning) they were unable to discriminate between dyslexics and non-dyslexics. If they cannot do this then their theoretical status is seriously challenged and it means that we are not ready to abandon the socially-constructed discrepancy lead definition of dyslexia which is part based on IQ and attainment scores.

10.3 Limitations

10.3.1 Sample size

Although some of the initial samples may have met the recommended size for statistical power (i.e., where the fullest sample possible was used), subsequent removal of certain participants meant that the initial samples were reduced in size. It is possible therefore that there were group differences which could not be detected and this can only be ascertained by replicating the work using larger samples.

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[67] Rather than symptoms of dyslexia.

For example, the dyslexia score of those in the "private" reflex group was just .39 when this group was isolated (n = 5). This is a very low score, well below a "mild risk" level of dyslexia, and certainly well below the high risk levels of .88 and .98 seen in the larger samples. This could suggest that, given a larger sample of those who had performed the therapy privately, the results could have shown a significantly lower score for this group's dyslexia risk and, thus, a therapeutic efficacy for reflex based programmes. As previously mentioned, however, it is possible that this selected sample was not the same as the rest of the "random" sample that was in their group, uncontrolled factors, such as fussier parents or intelligence, could be responsible for these differences. Similarly, with the ADHD sample, it could be that differences failed to be detected because of the reduced sample size when these children were selected for analysis.

For this reason, p levels which approached significance were noted across the analyses (e.g., p = 0.086, p = 0.07, p = 0.06, p = 0.065, p = 0.058 – see emboldened values in Appendix 24) and given consideration. It can be seen in Appendix 25 that some of the analyses of the "full sample" showed probability levels of 0.01; however, the corresponding analyses of the "minimum days sample" (which was consequently a smaller sample) had probability levels which rose to 0.05, suggesting that loss of participants may have made it more difficult to detect differences.

10.3.2 Test items

Sample size also limited the power of the analyses such that individual tests could not be used to explore their predictive validity (with the exception of the Schilder test where relevant). For example, although we know that the phonological variable was a good predictor, we do not know whether all the tests making up this variable were contributing to this or if one or two components were responsible for the observed effect. This applies to all the other variables also, for example, Moe-Nilssen et al. (2003) suggested that dyslexics did significantly less well on tasks of dynamic balance than did controls, and Fawcett and Nicolson (2004) that there was a discrepancy in static and dynamic tasks between dyslexics and other poor-readers (with dyslexics doing less well than poor readers on static tasks and poor-readers doing less well than

dyslexics on static tasks). In retrospect, it is questioned whether it may have been worth dividing balance tasks into their two types; static and dynamic in order to investigate whether one had any better predictive efficacy over the other. Thus, although there is some support for the model's utility in predicting both reading outcome and correct classification, it is difficult to know whether to attribute this to individual subtests or the combination on which the measures were based.

10.3.3 Sample slippage

In order to avoid continuous sample slippage, those parents who wanted to contribute something to the study, but found the programmes too arduous (for whatever reason), were moved into the control group. It was possible that these children consisted of those with fewer problems or less risk of dyslexia which affected their parent's motivation for continuing, or indeed starting, the programme. Had this been the case it would explain why the higher risk children remained in the balance group, although it does not account for why the reflex and placebo groups were no different from the control group seeing as they were subject to similar slippage. However, although the mean DEST / DST score for those children who transferred to the control group was slightly lower than the mean for the other participants originally allocated to that group, they were not significantly different.

10.3.4 Pre-post measures

One of the most regrettable, but unavoidable, features of the study design was the lack of availability of a pre-test measure against which to judge Group x Time change on dyslexia risk status. The age range of the children did not allow for use of the DEST / DST at the outset and it is unlikely that the children would have been able to withstand assessment sessions of the required length anyway at their initial, younger ages. This makes interpretation of the balance and reflex group DEST / DST scores difficult because, although random assignment should have meant that both groups were initially equally at risk for dyslexia, it is impossible to know for certain whether or not this was the case.

10.3.5 Compliance

Programme and study compliance is a major factor in a study such as this, even considering that the parent participants were volunteers and should have been highly motivated as a result of their dyslexic backgrounds. Many recruited families withdrew entirely. In all, 42 of the original families (i.e., 23.6% - almost a quarter - of the original sample) withdrew. Several others never began, or abandoned, their programme early on, necessitating a move to the control group, as happened with a further 21 of the families (i.e., 11.8% of the original sample). Of those who managed to persist with their programme, a further 18 failed to do a minimum number of days to be considered appropriate (according to the minimum defined in Primary Movement schools). Of the original sample of 178 children / families, 38 were assigned to the control group, leaving 140 involved in programmes. Thus, of the original sample, only 56% performed a minimum level of the programme.

To what extent the nature of the programmes was responsible for interfering with this commitment is difficult to quantify. Parents reported that finding 10 minutes each day in a busy family routine was stressful, especially where there were other children to consider and especially if one of these (older) siblings was dyslexic, because any spare time was usually devoted to them. Others felt that the degree of concentration and control necessary for the programme was beyond the abilities of such young children. Some parents, or care-takers, felt that the programme would not have any real benefit and this may have affected motivation or quality of performance. However, it is possible, if not probable, that the poor level of commitment is a reflection of what would happen in the true population, because the same levels of parental attention and child concentration are needed for all of these programmes (although the expense of them normally might increase motivation somewhat). It would be interesting to know also whether these findings compare with compliance levels generally, regardless of the nature of the programme tasks.

Despite any inherent difficulties in the delivery of the programmes, there may have been ways to avoid them had more manpower and funding been available. Although each parent had access to the private mobile numbers of the researchers should queries arise, and an attempt was made in the summer of 2004 to contact all parents regarding their child's progress, it would have been ideal to contact each parent on a monthly or bi-monthly basis to maintain contact and encourage participation and

motivation. Alternatively, creating a website for parents to post questions, or encouraging them to use e-mail, both which could be dealt with on a daily basis, may have helped compliance.

Some parents did not have a video-recorder in the house, but did have a DVD player. Although most of these managed to view the video elsewhere, periodic reviewing may have been difficult, and so for these, it would have been preferable to have had DVDs of the exercises.

It would have been valuable also to regularly change the task so that children did not get bored and were constantly stimulated by the programme. Some attempt was made to do this but it is arguable that there was still a lot of repetition in the programmes. Perhaps with more man-power to design different programmes this too could have been less of an issue. Ideally the programmes would all have been specifically tailored to suit the developing needs of the child; this would also have combated the problem of repetition in each programme.

The stories which accompanied the movements were designed for pre- and early- school children and involved animals, giants and magic. Some of the children had reached the age of 7 years, and a couple 8 years by the end of the study and it could be that these stories were too young for them. Alternatively, some of the stories designed around the "Mother and Son Tales" may have been too difficult to engage the younger child. Parents were told that the stories were there to help the children in the initial stages and that, provided they continued to do the same exercises, in the same order correctly, they were welcome to change the stories to suit the child or eliminate them if need be. Perhaps this should have been made clearer before the children began the programmes so that parents could manage their children's interest better from early on.

Finally, the need to do the programme every single day may be expecting too much commitment. Perhaps a system which rewards children and families for their hard work would keep motivation up by allowing a rest day once a week or even the weekends off. With weekends free, the families might link the programme with school and the routine involved in going to school so that it becomes part of the daily routine and they are less likely to question doing it on a daily basis. In cases where parents felt it was a matter of building in regular breaks (e.g., weekends free; "four days on, one off") orbandon the programme entirely, the built in break was encouraged to retain numbers.

10.3.6 Age range of participants

One of the flaws of the study which has subsequently come to light is the age range of the participants who engaged in the study. This ranged from 3 to 7 years, thus spreading over 4 years and encompassing a range of abilities. The initial plans were to recruit those of 4 and 5 years, however, concern over sample size meant that the study was opened out to older and younger children. Whilst we stopped recruiting 3 year olds fairly promptly because of the finding that they found it difficult to concentrate for the full testing session, the 6 year olds were necessary to make the sample large enough for analysis.

Most reflexes are considered to have been integrated by the time the child is 6 months old and certainly by the time they reach toddler-hood and therefore there is little concern over the reflex tests employed here, however, the abilities of such children do differ across motor, fine motor, visual and phonological tests. It would have been ideal to run a pilot study prior to testing in order to ascertain norms for each different age group in order to allow for standardised comparison of groups at analysis.

In the case of the (gross) motor tests attempts were made to account for this age range. For example, for the one leg balance, 3 and 4 years olds were permitted to hold their arms out at shoulder height to aid balance and the length of time that they were expected to hold their balance was less than that expected of 6 year olds. This balance aid was also permitted in the walking tests of the Mann and Heel to Toe test, in addition to which they were also allowed to look down at their feet. It was also acceptable if children under 4 did not quite walk heel to toe. Malina (2004) suggested that there are several periods of brain maturation during childhood; the first occurring between the ages of 15 and 24 months, the second between the ages of 6 and 8 years and the others during adolescence. Thus, it is possible that some within this study had reached a point of brain maturation which affected their ability to move more smoothly than other in the study. However, no accommodations were made for chidren of lower or higher ages on the other tests. This means that for some children (i.e., those of 3 years) the tests may have been too hard and for others (i.e., 6 and 7 year olds) the tests may have been too easy (i.e., there may have been both floor and ceiling effects at work here). Examples of such tests is mentioned in the section on "Testing" within Limitations.

Thus, the need to standardise the tests, or recruit a narrower age range, would be advisable for future studies.

10.3.7 Reliability and validity of tests used

It is quite possible for tools to be reliable even if they are not valid and the tests used here are a possible example of this. Although inter-rater reliability was high, the fact that all testers could observe and measure the same behaviour says nothing about the tests' ability to measure the construct in question (i.e., cerebellar skills). This could either be because the tasks do not tap cerebellar skill or because they were not sensitive enough for this sample (e.g., perhaps the tests were too easy or difficult for everyone, thereby creating ceiling or floor effects) in which case they are poor discriminators. Fawcett et al. (2001) claimed that standardised motor batteries do not cover the necessary range of cerebellar abnormalities for these kinds of samples. However, as discussed in chapter 7, Brookes and Stirling (2005) reported that reducing Dow and Moruzzi's (1958) test battery did not reduce the test's validity.

10.3.8 Length of assessment

All tests relied upon the motivation and alertness of the child and it is possible that the testing sessions were just too long. At times 1 and 2 the sessions were one hour and at time 3 they were one and a half hours (the additional half an hour was due to application of the DEST or DST). With more manpower the DEST / DST could have been administered later in the day or week in order to avoid putting excessive demands on the children. Although 8 research assistants were available over the 2.5 year period of testing this was still not sufficient to allow for such extended time and lab-space it might have required.

10.3.9 Testing

The scoring of retained reflexes raises important issues relating to validity. Whilst the inter-rater reliability proved to be highly acceptable in this study, there were a couple of issues with McPhillips's scoring method[68] which generated concern about measurement differences. For example, with the ATNR (Ayres, i.e., quadrupedal position), the theoretical literature would suggest a bend in the contralateral or *opposite*

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[68] Taken from his training course, Dublin 2002.

arm. However, McPhillips's method instructed scoring to be directed at the homolateral arm which seems contradictory. In addition, Pacella and Barrera (1940) found that an ATNR (supine version) response will often suppress the grasp reflex on the side to which the head is turned but reinforce it on the occipital side, such that a movement in the fingers will be evident on the opposite side. McPhillips's scoring method, however, instructed observation and recording of finger movement on the side to which the head was turned, thereby monitoring something quite different from what Pacella and Barrera would advise. Scoring, therefore, may have failed to detect some subtle signs of this reflex, thereby rendering an inaccurate representation of reflex retention and inaccurate interpretation about its utility. Other aspects of McPhillips's scoring method are also questioned. For example, the fact that he measured most tests out of just 5 renders the issue of whether or not this provided a sufficiently large scale to detect change. Also, there is the question of whether some of the tests were in fact too difficult for the younger children in the current age group. McPhillips made use of some balance tests which required both eyes open and eyes closed. Although accommodations were made for younger children on some tests, the Mann Test and Finger to Nose Test, which required testing of eyes closed as well as open, was administered to all children. It is felt that this should have been omitted for children under the age of 5 years. These three examples demonstrate a serious problem: that is, although the same tests may apparently be in use across studies and agencies, if the examiners are watching for and measuring different responses, then there can be no consistency (i.e., reliability) across the groups, and if there is no reliability, then there can be no validity. Other examples of tests which may have been too difficult for the younger children are some of the fine-motor tests, such as the Finger to Thumb test. This is included in the Paediatric Early Elementary Examination (PEEX 2), however, this is normed for children between the ages of 6-9 years. Age appropriate tests for younger children can be found in the Paediatric Extended Examination at Three (PEET at age 3) or the Paediatric Examination of Educational Readiness: Ages 4-6 years (PEER) (Levine).

Despite ambiguities about what actually constitutes evidence of particular retained reflexes, measurement error could be reduced by using an objective, electronic device to detect movement and the extent of that movement (see "Future Work").

There is also the possibility for concern over the time devoted to obtaining evidence for retained reflexes. A study which tested for the ATNR on normal newborns found that only 60% demonstrated signs of the reflex, and furthermore, the chances of observing the reflex on one trial was 3 / 16 (Vassella and Karlsson, 1962). Pothier et al. (1983) suggested testing each reflex a number of times (up to 5). Thus, it maybe questioned whether each reflex was tested for thoroughly enough in the current study (two samples were taken for each reflex, with eyes open and eyes closed) following the protocol designed by McPhillips.

Finally, despite the fact that Brookes and Stirling (2005) did not find any correlation between tests of hypotonia (i.e., a state of reduced muscle tension) and reading or reading sub-skills (e.g., picture arrangement), Fawcett and Nicolson (2001) suggest that it is a good test for cerebellar dysfunction in dyslexic children, given that dyslexics scored particularly poorly on tests linked to the lateral parts of the posterior lobe and that hypotonia is a core cerebellar deficit linked to this specific area. In retrospect it may have been disadvantageous to omit this particular test and future work would be strongly advised to include it.

10.3.9.1 Other tests

Although care was taken to use motor reflex tests which had reliable discriminating power, Crutchfield and Barnes (1993, pps.274 & 424) voice concern over some commonly used tests for postural control of which the Romberg is one (Nashner, 1990). Furthermore, Crutchfield and Barnes also claim that visual observation, as a method of evaluating a vestibular-ocular reflex (VOR) (as used in this study) is often inaccurate. They go on to suggest that VOR is independent of the vestibulospinal contribution to postural control anyway, citing a study by Horak et al. (1988) which failed to confirm a relationship between VOR functioning and motor deficiencies. The same conclusion was supported by Shumway-Cook et al. (1987) who found no relationship between VOR scores and postural balance tests in learning-disabled children; in fact, although these children did show deficits in balance, they were normal on their VOR tests.

Cratty (1979) states that the "heel-to-toe" stand is seldom seen in children before middle childhood, and in evaluating the finger to thumb test, deQuiros and Schrager (1978) suggest observing the opposite hand for contractions or movements as this may indicate that the child has not yet achieved "laterality". This opposite-hand observation was not made in the present study but, in retrospect, perhaps it would have been informative to have included it.

Blaiklock (2004) showed that children were unable to perform phoneme deletion tasks until they developed word-reading skills. Thus, this task may not have been appropriate for children who had not yet had sufficient time in school to learn to read.This query may also apply to the Non-Word Repetition test.

10.3.9.2 Tests - conclusion

Thus, in conclusion, some tests used in this study, particularly the fine motor coordination and visual-motor tests, may not have been specific enough or may have involved too much subjectivity in scoring. These, incidentally, were not subject to any assessment concerning their inter-rater reliability in the way that the reflex and balance tests were.

Importantly, soft sign tests for cerebellar functioning, as used by Dow and Moruzzi (1958), Fawcett et al. (1996) and others, are only an indirect measure of cerebellar functioning, so failure to find any correlation between soft sign test scores and performance on the DEST may have been due to the lack of sensitivity of these tests. Furthermore, confining the number of tests used (compared to Dow and Moruzzi) was necessary for time purposes but this may also have had some effect on the validity of the test battery to identify those with cerebellar impairment (despite Brookes and Stirling's (2005) claim that a reduction in test items did not prejudice the validity of the battery when reduced from 14 to 8 tests). Finally, although Brookes and Stirling did find a significant correlation between tests of cerebellar functioning and reading, only some of the tests of cerebellar functioning they employed contributed to that correlation. It could be that a different selection of cerebellar tests was used here. According to Topka and Massaquoi (2002) only a small number of specific clinical signs may be viewed as being indicative of cerebellar dysfunction. Perhaps a more formal set of tests should have been used (e.g., the International Cooperative Ataxia Rating Scale (ICARS)) for a quantified scale of cerebellar ataxic signs (see Trouillas et

al., 1997). The importance of using a sufficiently broad battery of tests was demonstrated in a study by Kish et al. (1988) whose patient sample showed no abnormal responses on the Mini-Mental Status Examination (which is a simple mental test) despite having olivo-ponto-cerebellar atrophies. It took a fuller, more comprehensive testing battery to elicit subtle deficits across a broad range of skills, including, verbal, non-verbal, memory and frontal system function (Leiner et al., 1989).

10.3.10 IQ

It was not possible to administer IQ tests to the children involved in the study given t already extended testing time necessary. A full IQ test would have taken upwards of an ho and although subtests may have been used in lieu of a full test, it was hoped that rando allocation to groups would control for differences between groups. There is no way knowing whether or not this was in fact the case but it is something which should considered in future such work.

10.3.11Validity of the DEST / DST

There is good evidence for the predictive validity of the DEST (Nicolson and Fawcett, 2004) although this work has, to date, not been replicated, particularly on an Irish sample. The predictive validity of the DST is not known. Using the DEST / DST, however, the current study identified 30-39% of the sample as being at risk for dyslexia (depending on the cut-off point used). This is in line with expectations from research on the heritability of dyslexia.

Nevertheless, it appears that the motor-based interventions had no effect on reducing the risk of dyslexia using this measure, among this sample, and that motor-based tests were poor predictors of "at risk" status for dyslexia. However, these interpretations depend upon the ability of the DEST and DST to correctly identify the risk level of the child. If the DEST / DST failed to adequately identify those at risk of dyslexia, then there is no way of evaluating the validity of the predictor tests, or the interventions, in this study. If the DEST / DST only identified the worst of these children, then Fawcett and Nicolson's (1999) claim that cerebellar tests might only predict the most severe cases cannot be supported.

10.3.12Controls for comorbidity

Whilst it was possible to control for ADHD using the Conners' Scales, it was not possible to control for the potential co-morbidity of Developmental Coordination Disorder (DCD) (i.e., dyspraxia) beyond asking the parent whether or not this had been diagnosed (which was seldom the case).

10.3.13Retrospective questionnaire

Important for the analyses were questions on the retrospective questionnaire which asked about time and quality of effort spent on the programme each day. However, responses to these questions are open to the influence of social desirability, either consciously or not. This problem, of course, is not unusual in questionnaire methodology. Conscious faking could either be due to the desire to look compliant or to please the researcher. Subconscious faking could be due to a need to justify participation in the study to oneself and the effort of coming to the laboratory for three assessment sessions. Random assignment should have ensured that the tendency to fake was evenly distributed across groups, although this does not rule out the possibility that everyone misreported and that few engaged in the programme properly. Ideally there would have been some objective way to assess qualitative compliance.

10.4 Future work / Recommendations

10.4.1 Reliability of scoring

It was previously mentioned that measurement error could be reduced by using an objective, electronic device to detect movement in the assessment of motor reflexes. This could utilise automatic sensors, such as strain gauges, however, an alternative method was developed and explored in parallel with the current study, called "DysVideo" (in conjunction with the Department of Electronic and Electrical Engineering, Trinity College) and is currently in its final stages of completion. A computer programme analyses digital video-data, evaluating the precise degree of movement between initial position and maximum response to a stimulus (Kokaram et al., 2006; Denman et al., 2005). There is much potential in using such a method for

scoring movements, which could in principle apply to the more subtle movements of the eyes or fine-finger movements, as well, for example, as the degree of wobble involved in standing on one foot. However, the validity of such measurements would not be resolved.

Even without the use of objective methods for quantifying movements, further work is necessary to determine the reliability of the fine-motor and visual-motor tests used here. These were not subject to inter-reliability testing but in retrospect it is clear such testing would have been valuable. With the visual-motor tests in particular, too much unreliability may have been present in decision making, despite the scoring scale available. This particular test would possibly benefit from objective electronic measurement more than any other of the tests.

10.4.2 Alternative support for the CDH

The manifestations of inhibitory differences between dyslexics and non-dyslexics reported in the literature are worth exploring in order to evaluate further the cerebellar deficit hypothesis. Although not clearly replicated in this study, the previous evidence of retained primary reflexes in dyslexics suggests that they have problems with inhibition at the behavioural level. McEvoy (2004) found significantly increased evidence of faulty inhibition following alcohol inducement (measured both behaviourally using retained reflex tests (Goddard, 1996) and cognitively using the XY response inhibition task (Garavan et al., 1999), thereby showing a causal link between decreased inhibition and retained reflexes. To validate the claims of McPhillips et al. (2000), Reynolds et al. (2003) and Goddard (1996) and link them with the cerebellar deficit hypothesis would require more careful and direct exploration, of not just behavioural measures, but also cognitive and neurobiological measures.

There is strong evidence for dyslexics' difficulties with cognitive inhibition (e.g., Facoetti et al., 2003; Brosnan et al., 2002). The principal neurotransmitter responsible for inhibitory function in the human brain, and particularly the cerebellum, is GABA (Vicini, 2005). If the cerebellum is involved in neuro-developmental disorders, such disorders could be due to poor inhibition (e.g., ADHD – Nigg, 1999;

dyslexia – van der Schoot et al., 2002[69]) and dyslexia, ADHD and GABA could all be linked to chromosome 6 (Willcutt et al., 2002; Smith et al., 2001; Cutting et al., 1992; Hisama et al. 2001). Thus, it would be worth investigating the levels of GABA in dyslexics compared to controls (see also Grigorenko, 2003). The current research may fail to support the cerebellar deficit hypothesis from a predictive test or treatment efficacy approach, but this does not necessarily negate the entire theory. It may simply be that it is impossible to remediate a poorly developed cerebellum through movement exercises in the age range studied here. Psychobiological and cognitive support for the theory would add an important further dimension.

For the idea of cerebellar deficiencies to gain more credence it would also be useful to have a fuller account of the nature of the deficiencies and how they function (e.g., is it, as mentioned earlier, a disturbance of the inhibitory efferent projections from the neo-cerebellum?). Similarly, if successful treatments are based on remediating these deficiencies, there is a need to describe theoretically how they are doing this.

10.4.3 Predictive validity of DEST / DST

The finding that there were only 30-39% of dyslexics in our sample is based on the assumption that the DEST / DST tests could adequately detect all of those with (or would later manifest) dyslexia. Although this finding was not out of line with some previous heredity studies (as mentioned earlier), it was at the lower end of what the literature suggests. As also previously mentioned, the predictive validity of the DEST has not been replicated to-date on any other sample. The predictive validity of the DST is not known and needs to be further explored. In addition to providing this information generally, it would be useful to have data relating to an Irish sample. It was hoped that a sufficient number of children would have been officially diagnosed using a full psycho-educational assessment and that these details would furnish us with enough data to conduct chi-squares in order to investigate these questions. However, to date

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[69] Van der Schoot et al. (2002) used a stop-task to measure inhibition and found that "guessers" (i.e., fast, inaccurate readers) were more likely than non-guessers (i.e., slow, accurate readers) to have a weak association between response inhibition and inhibition of activity in the central motor structures. The subject of subtypes is not the focus of this work but at the very least van der Schoot et al.'s work gives some support to the idea of inhibition difficulties in those with specific learning difficulties.

this has not been the case. Approximately 11 children have had a full assessment, 10 of whom have been diagnosed as dyslexic. This does not give a large enough number of fully-assessed non-dyslexics to run a chi-square analysis. Although parents are generally aware enough of their children's difficulties to determine their progress, some of the children are probably considered too young to have a full psycho-educational assessment. Those who have made contact with private or NEPS[70] psychologists may be on waiting lists, which can be at least 6 months long. We are hoping to maintain contact with all parents however, and to collect as much of this information as we can, so that such results will be available within a couple of years.

10.4.4 Statistical analysis

One of the unfortunate aspects of the current study was the small percentage of those classified as "at risk" for dyslexia by the DEST / DST. Because of its dependence on means analysis, in theory, use of a MANOVA may be a relatively weak measure of treatment efficacy compared to a chi-square analysis in future such studies. This appears not to have presented too much of a problem here because there were no analyses in which the MANOVA failed to detect a difference that was already detected by the chi-square. Nevertheless, in order to avoid such concerns in future work it would be necessary to make as much effort as possible to capture a larger proportion of dyslexic individuals in the sample. An attempt was made to do this here by initially recruiting only males (or girls who were already showing difficulties in school), by ensuring that there was an immediate family history of the disorder and by enquiring about co-morbid disorders or medical problems which might explain any dyslexic-type difficulties (e.g., poor eye-sight, poor hearing). The use of pre-tests to determine risk level (see below) may also be employed or the use of a slightly older sample (i.e., early school years) rather than pre-school years so that the teacher can give an indication of how the child is performing relative to the class.

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[70] NEPS: "National Educational Psychological Service", run by the Irish government's Department of Education and Science.

10.4.5 Pre-post measures

The present study compared four randomly-assigned groups on one main outcome variable; a more definite way than this of measuring change over time and the effects of interventions is to employ a pre-post design. Although this was done in the present study in the case of motor-tests it could not be done using the dyslexia measures for reasons already outlined. Perhaps another such study could still recruit young children but ensure the lower cut-off age was 4.5 years so that the DEST could be administered at the start of the study. This could be administered on a separate day to elicit a reliable assessment of the child although this would create logistic difficulties in terms of man-power and time. Alternatively, there is now available a younger version of the DEST called the Pre-school Early Screening Test (PREST) designed for children aged 3 years, 6 months to 4 years, 5 months (Fawcett, Nicolson and Lee, 2001).

10.4.6 ADHD theory

Although using the ADHD measurement taken from the Conners' ADHD scale did not replicate the results of previous studies claiming ADHD was responsible for the motor abnormalities seen in dyslexics, the Conners' scale yields several other measures, including Oppositional Behaviour and Hyperactivity. Future research might subject these dimensions to outcome analysis in the evaluation of these earlier claims.

10.5 Conclusions

Methods of early identification and intervention are beneficial and welcome but the former must be reliable and valid and the latter must show clinical efficacy and effectiveness. Many have advocated the need for more rigorous and independent studies of movement-based exercises in ameliorating specific difficulties such as dyslexia (e.g., Whiteley and Pope, 2003) and this present study attempted to do just that. In response to the Reynolds et al. article, Peer (2003) wondered why we hear nothing from those who claim such programmes do not work (p.124). Satisfying the stringent "gold-standard"[71] methodology that validates claims of treatment-efficacy and

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[71] Double-blind, placebo-controlled, randomly assigned.

applying it to a longer time-frame takes years of careful preparation, wide recruitment and patient co-operation with, and from, the public. However, we are now in a position to let our findings be heard.

Although there are limitations to the current study, I do not believe they are sufficient to undermine its validity. Given the ability of the phonological variable to predict dyslexia score and dyslexia status in many of the analyses, the study cannot be easily criticised on the grounds of sample selection or size. If the other variables had the degree of predictive ability that the phonological tests had, this should have been evident. Although there was some small support for previous findings of group differences on motor variables, these were either inconsistent or difficult to interpret. Such findings suggest that motor tests are best avoided as a means of identifying those at risk of specific reading problems. It seems that although the present, formal method of diagnosing dyslexia (which does not rely on motor tests) may be less than ideal, it may, nevertheless, be the best system currently available. The present investigation of cerebellar-based tests for identifying dyslexia, judged on the basis of predictive validity, would suggest that the adoption of this criterion, as currently practiced, may at best lack sensitivity and at worst be completely inaccurate. The approach of identifying deficiencies in requisite skills by comparison with expected skills on the basis of IQ, would seem to be the less foolhardy approach of the two, and one which, given its standardisation, is worth defending.

Compared to the (test) predictive efficacy analyses, there was even less support for the claim that interventions based on (1) inhibiting persistent reflexes or (2) targeting the cerebellum through vestibular stimulation, were useful in reducing dyslexic symptoms. Looking again at Figure 11 it is easy to trace the origin of motor intervention programmes. They all link back to the same set of people whose claims were either not scientifically tested by them or whose programmes were criticised by more rigorous investigation. Although such programmes took a back seat for several decades they have now resurfaced albeit with different names. It is necessary to remind ourselves of previous findings and to re-investigate the claims using contemporary target samples. There were two main questions raised by Kavale and Mattson (1983) with regard to treatment research: (1) Does it work and (2) Does it cause undue harm, either financially, emotionally or by removing child from a better suited learning environment? In response to the first question, the weaknesses of the current study are acknowledged, however, the design of the study was sound and the sample size deemed

adequate, and on this basis it is felt that the claims of such treatments should be cautiously considered in light of the weak support generated here. The answer to the second question depends in part on what the client's feelings are in response to having engaged with a programme. However, given programme cost, parents, teachers and concerned adults would be advised to consider carefully the results of this study before committing themselves to them emotionally, financially or temporally as such time and money could be spent on some more useful pursuit of academic improvement

Despite the fact that the cerebellar deficit hypothesis was not overwhelmingly supported in this study, it should not be rejected out right. The theory has notable strengths in furthering our understanding of dyslexia. The descriptive model (Figure 8) accounts or allows for different levels of impairment, from the genetic, to the neurobiological, cognitive and behavioural, thereby fitting Frith's three level framework (Figure 2). It incorporates the phonological deficit hypothesis, the temporal processing deficit hypothesis and allows for the magnocellular hypothesis. It also fits nicely with the inclusive definition of dyslexia used by the Dyslexia Institute (see Introduction). The current study casts doubt on the use of soft-sign tests as sufficiently sensitive indicators of cerebellar abnormality and suggests that programmes designed to ameliorate this abnormality fail either because they are not targeting the cerebellum properly or because the cerebellum cannot be changed in this way. More precise (cerebellar) neurobiological research is suggested which examines the role of inhibition, either directly measuring relevant neurotransmitters associated with the cerebellum or indirectly by measuring behaviours associated with inhibition.

Rather than considering the cerebellum as a unitary functioning organ, it may be best to understand the cerebellum as a *system*, the subdivisions and functional compartmentalisations of which need to be properly appreciated before it will be possible to understand its deficiencies, and in order to design specific tests for those deficiencies.

A strong case can be made that future work in the exploration of developmental dyslexia should be multidisciplinary in nature, drawing from genetics, developmental embryology, endocrinology, nutrition, social studies, neuroscience, education and psychology. Replicative support for the cerebellar deficit hypothesis is one thing; explaining how, where and why deficits link to reading difficulties in quite another.

Science is slow to develop and the discipline of psychology is no different. The current work, and that on which it is based, may do nothing but lay a path in the direction of truth. Ultimately, scientific knowledge grows as old theories are falsified and replaced by new theories that are more powerful in explanatory depth and more predictive over a wider range of phenomena (Popper, 1963). Despite its limitations, this research may help to challenge or further some of the emergent claims concerning the causes and treatment of developmental dyslexia.

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APPENDIX 1

BALANCE TESTS

ONE LEG BALANCE

Demonstrate the test.
Ask children to watch and listen before attempting test.
Give 5 seconds practice.

With eyes open bend the knee and lift the foot off the ground.
Count seconds.
Repeat for other leg.

Standardization for age groups:

3 year old:	Place arms out at shoulder height to balance, lift foot off floor in front of body.
4 year old:	Place arms out at shoulder height to balance, lift foot off floor in front of body.
5 year old:	Place hands on hips, lift foot off floor in front of body.

Scoring:

Time in seconds: Left foot _____

Right foot _____

1 Perfect rock solid – 5 seconds at 4 years, 6 seconds at 6 years
2 Slight wobble, 20% elevation of hands
3 Sway, hands moving
4 Puts foot down within 5 seconds
5 Unable

Mann Test (Advanced Romberg)

Demonstrate the test.
Ask children to watch and listen before attempting test.
Give 5 seconds practice.

The child is to stand heel to toe with eyes open.
Repeat for eyes closed.

Standardization for age groups:

3 year olds:	Place arms out at shoulder height to balance
4-5 year olds:	Hands on hips

Scoring:

Eyes open	_____seconds
Eyes closed	_____seconds

1 Perfect rock solid (8 secs)
2 Feet wobbling
3 Sway, hands out
4 Steps out of position within 8 seconds
5 Unable

TANDEM WALK

Gross Muscle Co-ordination

Demonstrate the test.
Ask children to watch and listen before attempting test.
Give 5 practice steps.

The child is to walk heel to toe for 10 steps along a 4ft x 4 inch line.

Standardization for age groups:

Under 5: hands out and allowed to look down, not necessarily heel to toe, forwards only.
Six year olds: place hands on hips, walk heel to toe, forwards and backwards.

Stop counting steps immediately a foot comes off the line.

Scoring:

Forwards _____ steps
Backwards _____ steps

1 Perfect 10 steps
2 Cannot do heel to toe over 5 years despite correction
3 Major wobble
4 Under 10 steps/unable to keep foot on line
5 Foot down on first step/unable

4'' line without stepping off (8ft without swaying 20%).

49-54m: with hands on hips, toes touching heels walks backwards for 5 steps without stepping off.

WALKING ON TIPTOE

Demonstrate the test.
Ask children to watch and listen before attempting test.
Give 5 practice steps.

Place coloured line on floor.
Child to walk up to 10 steps on tiptoe.
Allowed to look down.
Stop counting if child comes off line or heel goes down.
From 5 years try backwards as well.

Standardization for age groups:

3 year old:	Place arms out at shoulder height to balance.
4 year old:	Place arms out at shoulder height to balance.
5 year old:	Place hands on hips.

Scoring:

Forwards _____ steps
Backwards _____ steps

1 Perfect (10 steps)
2 Cannot take small steps, large steps only/shuffle backwards
3 Sway 20%
4 Unable to stay on tiptoe (less than 10 steps)
5 Foot down on first step/Unable

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SLALOM WALK

Demonstrate the test.
Ask children to watch and listen before attempting test.
Give 5 practice steps.

Use diagonal line on floor
Child stands to left of line with hands on hips and lifts leg at knee and places foot on other side of line
Child to walk up to 10 steps
Allowed to look down
Make sure child understands instructions
Make sure testers steps are not too large
Stop counting if foot does not cross line, child overbalances and puts foot down

Scoring:

Forwards _____ steps

Backwards 6+? _____ steps

1 Perfect (min 10 steps)
2 Slight wobble & hesitance placing foot over line
3 Body sway
4 Less than 10 steps
5 Steps on line or puts foot down immediately

HOPPING ON THE SPOT

Demonstrate the test.
Ask children to watch and listen before attempting test.
Give 5 practice hops.

Valid from 4 years up
Place hands on hip
Hop on one leg
Score up to 10 hops
Repeat on other leg

From 8 years: hop forward on one leg.

Scoring no of hops: left foot_____

right foot_____

1 Perfect 10 hops
2 Wobble on landing/moving around
3 Quick hops not gaining any height/2nd foot resting on hopping leg
4 Less than 10 hops
5 Unable or not lifting hopping foot off floor

MARCH AND ABOUT TURN

Demonstrate first using outline square of green material
Ask children to watch and listen before attempting test.
Give 5 practice steps.

Swinging arms first on the spot
Lifting the knees start to march on the spot
March around the square.

Scoring:

Observe pattern and tick box

Homolateral
Crosslateral

1 Perfect crosslateral
2 Loses crosslateral reverts to homolateral
3 Mixed cross and homolateral gait
4 Full homolateral
5 Having difficulty marching at all

CRAWLING ON ALL FOURS

Demonstrate first.
Ask children to watch and listen before attempting test.
Give 5 practice steps.

The child is to crawl along the diagonal.

Watch for:
1. homolateral/cross pattern
2. Head flick
3. or opposite knee flick

Scoring:

Observe pattern and tick box

Homolateral
Crosslateral

1 Perfect crosslateral
2 Loses crosslateral reverts to homolateral
3 Mixed cross and homolateral gait
4 Full homolateral
5 Cannnot manage any crawling

Demonstrate First
Ask children to watch and listen before attempting test

Motor Control

1. **Finger-to-nose test**

Description:
Eyes open
Stand with feet together
Bring elbows out to shoulder height
Bring alternate **finger tips** to **tip of nose 10 times**

Try with eyes closed.

Scoring:
1 Perfect
2 Compensating, bringing head to nose, or **side** of finger to nose
3 Hitting side of nose
4 Hitting face
5 Unable

Eyes open	**1**	**2**	**3**	**4**	**5**
Eyes closed	**1**	**2**	**3**	**4**	**5**

Demonstrate First
Ask children to watch and listen before attempting test

Motor Control

2. Heel on Shin

Description:
Child sits on chair
The left heel is brought to rest at the top of the
right shin just below the knee.
Push the heel down the opposite shin and push out at the end.
Try once only.
Repeat with right heel.

Scoring:
1 Perfect
2 Heel goes to side of leg
3 Inside of foot goes down leg

5 Unable to do it.

Left	1 2 3 5
Right	1 2 3 5

Notes:

Autistic and Aspergers children will struggle with these tests – indicating problems with the cerebellum/brain stem and other balance regions of the brain

Demonstrate First
Ask children to watch and listen before attempting test

Adiadochokinesia (rapid alternate movement)

3. Fingers

DEMONSTRATE FORWARDS AND BACKWARDS

Description:
Child sits on chair
Starting with the index finger on the left hand, the child brings finger and thumb together once and continues with the other fingers in a rhythmic tapping manner, forward and back.
Tester to count at least 10 consecutive taps.
Repeat with right hand.

Scoring:

- left hand
- right hand

1 Perfect, tapping has rhythmic ripple
2 Can do it but conscious of it
3 Can do it but slow and laboured
4 Can't do movement with one or more fingers
5 Unable to attempt it

Notes:
Usually the difficulty is with bringing little finger and thumb together.

Demonstrate First
Ask children to watch and listen before attempting test

Adiadochokinesia (rapid alternate movement)

4. Rapid reversal of hands

DEMONSTRATE SLOWLY – THERE MUST BE A DEFINITE UP AN DOWN MOVEMENT OF HANDS

Description:
Child sits on chair
Placing palms face down on knees, turn one palm up
Slowly reverse their orientation so palm up goes down and down goes up
Demonstrate and start slowly
There must be definite up/down rhythm
Child should attempt 10 rotations

Scoring:
1 Perfect, speed is fast and automatic
2 Slower rhythm
3 Really thinking about it but can do it
4 Cannot turn opposite hands
5 Unable to attempt it

Notes:

It is unusual that the child would fail on one test alone, there should be a pattern.

Demonstrate First
Ask children to watch and listen before attempting test

Adiadochokinesia (rapid alternate movement)

5. Finger and Thumb (Incy Wincy) (10 rotations)

Description:
Child sits on chair
Demonstrate slowly
Child places opposing index finger and thumb together
Twist hands release the lower two to meet on top
Give 5 practice goes
Time 10 rotations
Can break and continue

Scoring:

Time 10 rotations in _____ secs

Able 1 **Unable 5**

Notes:

Dyslexia Early Years Project

Name_____

Child ID _____

In school

Familiar with Letterland scheme

Phonological Tests

Letter Name Knowledge

Using the plain lower case flashcards ask the child to randomly name the 26 letters of the alphabet. Hold up a card for 5 seconds asking them to name letter and then continue to the next letter. Place an X in the box for an error. Score correct letters out of 26.

A B C D E F G H I J K L

M N O P Q R S T U V W X

Y Z

Total correct: _____/ **26**

Dyslexia Early Years Project

Name_____

Child ID _____

Phoneme Deletion Test:

The child is required to remove the first phoneme (sound in itialics) of the words below.
Say to the child *' in the word **bus** if I take out the ' **/b/'** I am left with .....**us**'* supplying the answer. Continue with the demonstration stimuli.

Demonstration stimuli: *b*-us *s*-ad *p*-ie *c*-ow

Now say to the child *' in the word **bear** if I take out the ' **/b/'** I am left with?...'* Do not supply the answer.

Test stimuli: *b*-ear *s*-it *j*-am *t*-in *c*-ake *t*-oy *c*-up *m*-an

Total correct: _____**/ 8**

Dyslexia Early Years Project

Name_____

Child ID _____

Non-Word Repetition Test:

The child is required to repeat non-words between one and four syllables in the following order:

1. Simple non-words:

sa	zi	mo	cu
le	ka	ru	fa
te	fu		

___ **/ 10**

2.

hond	grall	tull	clird
fot	nate	sep	bift
smip	thip		

___ **/ 10**

3.

hampent	bannow	diller	pennel
ballop	sladding	prindle	rubid
glistow	tafflest	barrazon	bannifer
thickery	skiticult	commerine	dopelate
brasterer	glistering	trumpetine	finneriser
contranponist	stoppograttic	penneriful	loddenapish
perplisteronk	empliforvent	commerciate	blonterstaping
woogalamic	frescoveril		

___ **/ 30**

REFLEX TESTS

SCORING 1-4 gives description in range

5 = UNABLE due to anxiety, muscle tone, unable to stay up on all fours, keep arms in position etc.

Demonstrate First
Ask children to watch and listen before attempting tests

ATNR: LYING DOWN

Test to each side – eyes open and eyes closed

The tester kneels behind the subject lying on the floor. Arms placed at 45 degrees to the body with elbows bent. Hands loosely placed palm down and fingers spread and with tips touching floor. The tester places hands on subjects head taking care not to cover ears.

- Tester turns head to the left and counts 5 seconds
- Returns head to midline for 1 second
- Tester turns head to the right and counts 5 seconds
- Repeat with eyes closed.

Scoring:

N.B. only score side of body to which head is turned

Arms:

1	**No movement on the side to which the head is turned**
2	**Tremor**
3	**Fingers move**
4	**Hand and arm move**

Feet:

1	**No movement**
2	**No score**
3	**No score**
4	**Feet move**

Left arm:	eyes open	1 2 3 4
Left leg:	eyes open	1 4
Right arm:	eyes open	1 2 3 4
Right leg:	eyes open	1 4

Left arm:	eyes closed:	1 2 3 4
Left leg:	eyes closed:	1 4
Right arm:	eyes closed:	1 2 3 4
Right leg:	eyes closed:	1 4

ATNR LYING DOWN

ATNR: AYRES TEST

Test to each side – eyes open and eyes closed

The tester kneels in front of the subject on the floor. The subject assumes table position on all fours with hands facing forward palms down, looking at the floor. Make sure arms are not locked or twisted. **Watch for twisting of body in compensation.** The tester places hands on subjects head taking care not to cover ears.

- Tester turns head to the left and counts 5 seconds
- Returns head to midline for 1 second
- Tester turns head to the right and counts 5 seconds
- Repeat with eyes closed.

Scoring:

N.B. only score side of body to which head is turned

Arms:

1 No movement on the side to which the head is turned
2 Tremor
3 Elbow flicks –trying to keep arm straight
4 Definite bend in the arm

Left arm:	eyes open	1 2 3 4
Right arm:	eyes open	1 2 3 4
Left arm:	eyes closed:	1 2 3 4
Right arm:	eyes closed:	1 2 3 4

Observe for twisting of body in compensation. Let subject relax and try again.

STNR

The child does this themselves do not guide the head.

With the child in the table position, sideways to the camera, arms facing forward, ask the child to put their head down looking between the knees. Hold for 5 seconds.

Observe arms

1 No movement
2 Tremor in the arms
3 Flick in the arms
4 Definite bend

Then ask the child to bring the head up and look forward at the ceiling

Observe trunk and legs

1 No movement
2 1 lurch backwards and corrects it
3 lurch backwards x 2
4 Goes backwards but does not correct it and sometimes sits on heels

Repeat for eyes closed.

Scoring:

Trunk involvement	1 2 3 4
Arm involvement	1 2 3 4
Evidence with creeping on all fours	1 2 3 4

STNR: TABLE

ATNR: SCHILDER TEST

Test to each side – eyes open and eyes closed

The tester stands behind the subject. The subject stands with feet together, arms held out in front at shoulder height. Hands are relaxed and palms down. The tester places hands on subjects head taking care not to cover ears.

- Tester turns head to the left and counts 5 seconds
- Returns head to midline for 1 second
- Tester turns head to the right and counts 5 seconds
- Repeat
- Rest as arms will be tired
- Repeat as above with eyes closed.

Scoring:

N.B. Only score side of body to which head is turned

Arms:

Observe does the head pull the arms in its direction

1 No movement or 0-10^0 in children under 5 years
2 Movement 10-25^0 in direction of head movement
3 Movement 25 – 45^0
4 Arms move +45^0

Score drop (e.g. opt out of the task)

1 Drop of 0-10^0 in children under 5 years
2 Drop of 10-25^0
3 Drop of 25 – 45^0
4 Drop of +45^0

Arm movement

To left: eyes open 1 2 3 4
To right: eyes open 1 2 3 4

To left: eyes closed: 1 2 3 4
To right: eyes closed: 1 2 3 4

Drop

To left: eyes open 1 2 3 4
To right: eyes open 1 2 3 4

To left: eyes closed: 1 2 3 4
To right: eyes closed: 1 2 3 4

ATNR: Schilder

TLR – UPRIGHT TEST

Stand behind the child in case of loss of balance no need to touch child. Talk through movement with child

Child should stand with feet together facing forward, eyes open
Child should bring head down to look at toes
Pause for 5 seconds
Bring head to midline
Take head up to look at ceiling
Pause for 5 seconds
Bring head back to midline
Repeat with eyes closed

Head down

Look for flexor movement in the legs, do knees move, also note sway:

1 No movement
2 Tremor in the knees
3 Little flicks in the knee
4 Definite bend in knees

Head back

Look for extension in the arms

1 No movement
2 Definite sway in body
3 Sway with arms
4 Loss of balance

MORO REFLEX - UPRIGHT TEST

Do not use sudden noises this will upset child and you will loose their trust.

Talk through the test and then perform twice with eyes open and twice with eyes closed.

Stand with feet together like a tree
Keep the head in midline
The tester places hands on the child's waist
"the tree is going to blow gently in the wind"
Guide the child 2-3 inches forward feet remaining still
The back to neutral
The rock child gently back on heels for 2-3 inches
Observe when they opt out
Do they step out – can't stop themselves
Repeat with eyes closed

1 No problems with backwards and forwards
2 On backward movement arms come up
3 They go backwards but fall out of it
4 Opt out of forwards/backwards movement

Toes coming up is normal, listen for breathing on backwards movement – intake of breath.

MORO: STANDING

Neurological Dysfunction as a significant factor in children with Dyslexia
Blythe, S.G., BDA International Conference. Dyslexia: At the Dawn of the Century, April 2001.

Oculo-Motor Functioning Tests:

1. Visual Tracking

Test Position: Standing with feet together, arms by the sides.

The child is instructed to follow the coloured top of a pencil with his/her eyes without moving the head. The pencil is held at a distance from the face equivalent to the distance between the end of the child's fingertips and the elbow, three inches below eye level.
The tester moves the stimulus slowly from side to side for **10 movements**.
Eye movements are observed for:

Scoring: once they have the flow score for stable performance

Ability to follow the movements of the pencil without anticipation	1
Jumping	2
Head movement.	3
Unable to focus on and follow pencil	5

2. Hand-Eye Tracking

Test position: Standing feet together, arms by the sides.

The procedure is the same for test no. 1 except that the child holds the pencil in his/her hand and is asked to move the pencil from side to side without moving the head. Let the child choose which hand.

Scoring: once they have the flow score for stable performance

Ability to follow the movements of the pencil without anticipation	1
Jumping	2
Head movement	3
Unable to focus on and follow pencil	5

3. Fixation with Eyes

Test Position: Standing with feet together, arms by the sides.

The child is instructed to fixate on the coloured top of a pencil with his/her eyes, and instructed to hold that fixation while moving the head from side to side **10 times.** The pencil is held at a distance from the face equivalent to the distance between the end of the child's fingertips and the elbow, three inches below eye level. When the movement is stable the eyes are observed for any deviation from the fixation points the head moves – ocular-reflex.

Maintains fixation point	**Yes 1**
	No 5

4. Near Point Convergence and Re-establishment of Binocular Vision

The child is instructed to focus on the coloured point of the non-writing end of a coloured pencil **held at eye level at a distance of arm's length from the face** and is instructed to follow the coloured dot as it is moved towards the subject until the eyes converge.

The child is then asked to say, "stop" when the image begins to blur/or they feel a strain on the eyes. The pencil is then moved away and the child was asked to say, "stop" when the coloured end becomes clear again. The distance between the point going out of focus and returning to focus again is measured. If the distance exceeds **6 to 8 centimetres**, re-establishment of binocular vision is slow.

_____ cms

Convergence OK	**1**
Convergence Slow	**4**

APPENDIX 2

Relevant centres currently selling movement-based therapies for people with neuro-developmental disorders:

- Neuro-Developmental Therapy (Ireland): NDT.
- Brain Gym
- The Handle Institute
- The Miriam Bender Achievement Centre
- Jean Ayres's Sensory Integration
- The Bobath Approach
- SMART (Lyelle Palmer)
- BIRD (David McGlown)

NDT: (Neuro-Developmental Therapy – Ireland):

The sole affiliate of INPP-Chester. It offers reflex-based exercised to treat Dyslexia, Dyspraxia, Attention Deficit Disorder (ADD), Attention Deficit Hyperactivity Disorder (ADHD), Co-ordination difficulties, DAMP, DCD & immature behaviour.

www.ndt-ireland.com

Brain Gym:

Based in Ventura, California, Brain Gym aims to use the "science of movement" to improve learning. It uses what it calls "Educational Kinesiology" which is the study and application of posture and movement, to enhance, among other things, academic achievement.

www.braingym.org

The Handle Institute:

An exercise programme, based in the Seattle, Washington, which offers to treat Learning Disability (i.e. Dyslexia, Dyscalculia, Dysgraphia and other SLDs), ADHD, Autism, Visual-perceptual-motor Dysfunction, Behaviour Disorders (e.g. Oppositional Behaviours, Bi-polar Disorder), Apraxia / Dyspraxia, Memory and Organisational Disorders, Aphasia and Language Disorders, and Tourette's Syndrome.

www.handle.org

The Miriam Bender Achievement Centre:

Based in Indianapolis, Indiana, this is an STNR inhibiting programme which claims to treat ADHD behaviours.

www.stoppingadhd.com

Jean Ayres's Sensory Integration:

Based in Torrance, California, the Ayres Clinic was founded by Jean Ayres to treat those with Sensory Integration Disorders (historically learning disorders).

https://mmm1106.verio-web.com/sensor/index.htm

The Bobath Approach:

This therapy began in the 1940s and initially aimed to suppress reflexes in people with Cerebral Palsy by increasing postural tone. It is based in London.

http://www.bobath.org.uk/

SMART (Lyelle Palmer):

Also known as "Boost Up", this is run by Dr. Lyelle Palmer from Minneapolis, Minnesota. SMART stands for "Stimulating Maturity through Accelarated Readiness Training". By using visual, auditory, vestibular and kinaesthetic inputs it claims to build the underlying readiness of students for school-based learning. This is designed to be used by all students in the education system.

www.actg.com

BIRD: Brain Injury Rehabilitation and Development (David McGlown):

This charity was founded in 1982, near Chester in England and is designed to offer treatment to those of all ages suffering from brain injury or learning difficulties. Using a "unique" method, known as "Developmental Reflexive Rehabilitation", this programme also aims to inhibit primitive reflexes which have either not developed properly or have been re-released due to brain injury. They offer help to those with Dyslexia, Dyspraxia, ADD, ADHD, Asperger's,

http://www.b-i-r-d.org.uk/index.htm

APPENDIX 3



Early Years Project

INSTRUCTION MANUAL

Movement Programme 1

Dyslexia Early Years Project
Dept. of Psychology
TCD
Tel: 608 2095
www.dysvideo.org

Contents

Letter to Parents	Page 2
Exercise 1: Instructions for parents	Page 6
Exercise 1: Stories for the children	Page 14
Exercise 2: Instructions for parents	Page 17
Exercise 2: Stories for the children	Page 22
Exercise 3: Instructions for parents	Page 24
Exercise 3: Stories for the children	Page 30
Exercise 4: Instructions for parents	Page 32
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Timetable	Page 42

Dear Parent/Guardian,

Thank you for agreeing to allow your child take part in the Dyslexia Early Years Project.

This manual is to help guide you in the movement programme you will be doing with your child over the course of the next year. The video-tape illustrates each of the movements described in the manual. It would be useful to look at the video first to get an idea of the movements.

We would like your child to spend 10 minutes each day doing some movements. It may be useful to build them into your daily routine, using a quiet room, perhaps before bedtime.

Refer to the timetable at the back of this manual for the exercise for each particular day and tick the box when it is done, leave it blank if no exercise was done that day.

Please do not hesitate to contact us at the numbers/emails below if you have any questions about any aspect of the programme. We want you and your child to enjoy this programme and if we can help in that please ask for advice at any time.

This project could have an enormous influence in helping children at risk for dyslexia. Your family's commitment to participation is very much appreciated.

Yours faithfully,

Erika Doyle *edoyle4@tcd.ie* *087 9746032*
Elaine Smith *esmith@tcd.ie* *086 3845473*

Dyslexia Early Years Project
Rm 1.15
Dept. of Psychology
Aras an Phiarsaigh
Trinity College
Dublin 2.
Tel: 608 2095
www.dysvideo.org

Getting Started

Once you have viewed the video please refer to the back of your manual for timetable of the exercises. Fill in the start date and tick the box when the exercise has been completed by your child each night. Try out your exercise and do not worry if your child does not complete the movements properly in the first few nights. Remember it is not a memory or learning game. The important thing is that the child concentrates on doing the movements competently for the required amount of time and eventually becomes accomplished at the movements after a few weeks. The stories are just to help keep your child's interest and keep the movements flowing.

The manual with your timetable filled in, and the video are to be returned to the Dyslexia Research Group at the end of your child's movement programme. They remain the property of Trinity College Dublin. These materials

are only to be used in the context of your child's participation in this study.

Programme 1

Instructions for Parents

Exercise 1: 12 weeks duration

Exercise 1 should be done by your child every night for 12 weeks. There are stories for each exercise to help make it fun. Sit the child in a low chair so that their feet are flat on the ground before they begin. You may find it comfortable to sit opposite the child and tell one of the stories on page 14 at a slow pace as you go through each stage of the exercise.

For Exercise 1 follow these steps:

Start Position:

1. Sit the child in a low chair crossing right arm over left placing the hands palms down at the shoulder.
2. Cross right foot over left.
3. Place the head at midline. This is where the head is in the centre position in relation to the body, not slightly up/down, left or right.



First Movement: Right Side

4. Keeping arms and feet in position, very slowly move the head to the right so that the chin is in line with shoulder. At first allow 5 seconds for this movement the first night. Gradually increase the time for this movement over the week so that by the end of week 1 it lasts for 30 seconds.



Encourage slow head movement lengthening the time each night until 30 seconds is reached.

Second Movement: Right Side

5. Holding the head in this position, extend the right hand slowly out to the right at shoulder height. At first allow 5 seconds for this movement. Gradually increase the time for this movement over the week so that by the end of week 1 it too lasts for 30 seconds.



Do not worry if this time is not achieved within the first week. Encourage slow head movement and full arm extension until the full time is reached.

---

6. Right Hand

Keep the arm in this position for 10 seconds. One way of doing this is for the child to count to 10 on fingers and thumb ***(thumb to forefinger, then to middle finger, to ring finger and then little finger, then work from little finger back to thumb and forward again until you count to 10).***



Do not worry if your child cannot fully manage this at first, encourage them to continue.

Third Movement: Right Side

7. Holding the head in position with the forefinger of the left hand placed on the chin, bring the right arm back to starting position. At first allow 5 seconds for this movement. Gradually increase the time for this movement over the week so that by the end of week 1 it lasts for 30 seconds.



---

8. Removing the finger bring the head back to the midline. At first allow 5 seconds for this movement. Gradually increase the time for this movement over the week so that by the end of week 1 it lasts for 30 seconds.



Encourage slow head movement lengthening the time each night until 30 seconds is reached.

Now change the child's sitting position and repeat the exercise with head and left arm:

Changing Start Position:

9. With the child sitting in the low chair, this time crossing left arm over right placing the hands palms down at the shoulder.
10. Cross left foot over right.
11. Place the head at midline.



First Movement: Left Side

12. Keeping arms and feet in position, very slowly move the head to the left so that the chin is in line with the shoulder. At first allow 5 seconds for this movement. Gradually increase the time for this movement over the week so that by the end of week 1 it lasts for 30 seconds.



Second Movement: Left Side

13. Holding the head in this position, extend the left hand and arm slowly out to the left at shoulder height. At first allow 5 seconds for this movement. Gradually increase the time for this movement over the week so that by the end of week 1 it lasts for 30 seconds.



Do not worry if this time is not achieved within the first week. Encourage slow head movement and full arm extension until the full time is reached.

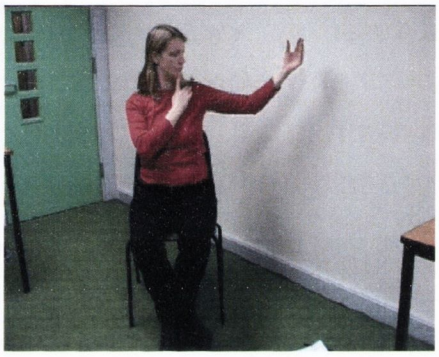
Keep the arm in this position for 10 seconds. One way of doing this is for the child to count to 10 on fingers and thumb ***(thumb to forefinger, then to middle finger, to ring finger and then little finger, then work from little finger back to thumb and forward again until you count to 10.***



Do not worry if your child cannot fully manage this at first, encourage them to continue.

Third Movement: Left Side

14. Holding the head in position with the forefinger of the right hand placed on the chin, bring the right arm back to the start. position. At first allow 5 seconds for this movement. Gradually increase the time for this movement over the week so that by the end of week 1 it lasts for 30 seconds.



15. Removing the finger bring the head back to the midline. At first allow 5 seconds for this movement. Gradually increase the time for this movement over the week so that by the end of week 1 it lasts for 30 seconds.



The full exercise can be done with eyes closed when mastered.

Goal: **After the first week this exercise should take about 4 minutes per night, moving head and arm out and back on each side.**

We do want your child to enjoy these exercises. Once your child is familiar with the movements you may wish to use some of the stories overleaf to maintain interest, or make up your own story, or you may prefer not to use a story at all.

Programme 1

Suggested stories for Exercise 1

A Dragon Fly in flight:

First Movement:
Move your head to follow the purple dragon fly as he flies from one flower to the next ***(slow head movement to the right/left).***

Second Movement:
Keeping your eyes on the dragon fly watch as he lands on the flower ***(hold head position).***
Slowly with your right/left hand, show what flower he flew to ***(right/left arm extension).*** Point to where he is ***(hold position).***
Count how long he stays on the flower ***(fingers to thumb).***

Third Movement:
Show him the way back to his flower ***(arm movement back to crossed over start position).***
Moving your head watch as the dragon fly flies back to his flower ***(move head back to midline start position).***

Repeat the story for head and left arm movement and legs crossed left over right as per instructions.

The Spaceship:

First Movement:
A little space ship flies across the night sky. Move your head to watch as it flies over to the moon ***(slow head movement to the right/left).***
Keeping your eyes on the spaceship, watch it land on the moon ***(hold head position).***

Second Movement:
With your arm trace the path it took across the night sky ***(right/left arm movement).***
Point to where it landed ***(hold head and arm position).***
Count how long it stays there ***(fingers to thumb).***

Third Movement:
Keep watching the spaceship but trace its way home with your arm ***(arm back to crossed over start position).***
Then move your head as you follow the spaceship on its way home to earth ***(head back to midline start position).***

Repeat the story for head and left arm movement and legs crossed left over right as per instructions.

A little bird flying:

First Movement:
Moving your head follow the little bird as he flies from his nest to the next tree ***(slow head movement to the right/left).***

Second Movement:
Watch as he lands on the branch ***(hold head position).***
Slowly with your right hand, show what tree he flew to ***(right/left arm extension- hold position).*** Pointing to where he is count how long he stays in that tree ***(fingers to thumb).***

Third Movement:
Show him the way back to his nest ***(arm movement back to start position).***
Moving your head watch as the bird flies back to his nest ***(head back to midline start position).***

Repeat the story for head and left arm movement and legs crossed left over right as per changing position instructions.

Programme 1

Instructions for Parents

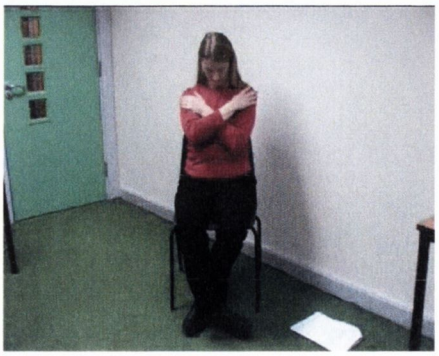
Exercise 2: 4 weeks duration

Exercise 2 should be done by your child every night for 4 weeks. There are stories for each exercise to help make it fun. Sit the child in a low chair so that their feet are flat on the ground before they begin. You may find it comfortable to sit opposite the child and tell one of the stories on page 22 at a slow pace as you go through each stage of the exercise.

For Exercise 2 follow these steps:

Start Position:

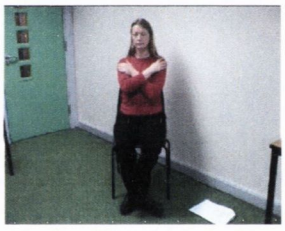
1. Sit the child in a low chair crossing right arm over left placing the hands palms down at the shoulder.
2. Cross right foot over left
3. Place the head down on the chest



First Movement:

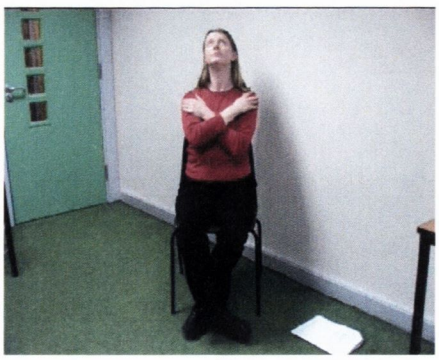
4. Keeping the arms and feet in position, very slowly lift the head so the chin is past the mid-line (but not so far back as to hurt the neck) and look at the ceiling. At first allow 5 seconds for this movement. Gradually increase the time for this movement over the week so that by the end of week 1 it lasts for 30 seconds and by the end of week 2 it lasts for 1 minute. Do not worry if this time is not achieved within the first week. Encourage slow head movement until the goal of 1 minute is reached.





Second Movement:

5. Keep the head in this position for 10 seconds.



Third Movement:

6. Bring head slowly back down to chest. At first allow 5 seconds for this movement. Gradually increase the time for this movement over the week so that by the end of week 1 it lasts for 30 seconds and by the end of week 2 it lasts for 1 minute.



Now change the child's sitting position and repeat exercise with the head:

Changing Start Position:

This is not shown in the video, however it is similar to above just follow the instructions below:

1. Still sitting in a low chair, the child now crosses left arm over right placing the hands palms down at the shoulder.
2. Cross left foot over right
3. Place the head down on the chest



First Movement: Changed position

4. As before keeping arms and feet in position, very slowly lift the head so the chin is past the mid-line (but not so far back as to hurt the neck) and look at the ceiling. At first allow 5 seconds for this movement. Gradually increase the time for this movement over the week so that by the end of week 1 it lasts for 30 seconds and by the end of week 2 it lasts for 1 minute. Do not worry if this time is not achieved within the first week. Encourage slow head movement until the goal of 1 minute is reached.

Second Movement: Changed Position

5. As before keep the head in this position for 10 seconds.

Third Movement: Changed Position

6. Bring head slowly back down to chest. At first allow 5 seconds for this movement. Gradually increase the time for this movement over the week so that by end of week 1 it lasts for 30 seconds and by the end of week two it lasts for 1 minute. Again do not worry if this time is not achieved within the first week. Encourage slow head movement until the goal of 1 minute is reached.



This can be done with eyes closed when mastered.

Goal: **After the first week this exercise should take about 4 minutes per night, bringing the head up and down and then crossing arms and feet in the changed position to repeat head movement.**

We do want your child to enjoy these exercises. Once your child is familiar with the movements you may wish to use some of the stories overleaf to maintain interest, or make up your own story, or you may prefer not to use a story at all.

Programme 1

Suggested stories for Exercise 2

The Little Daisy:

First Movement:
A little plant in the ground lifts his head out of the soil slowly to look at the sun ***(lift head from chest slowly).***

Second Movement:
The sun helps him grow strong ***(hold head position looking at the ceiling).***

Third Movement:
The sun goes down, the little daisy brings his head down slowly and goes to sleep ***(bring head slowly back down to chest).***

Repeat story for exercise with legs crossed left over right as per instructions.

The Spaceship Lands:

First Movement:
A little spaceship pod lands in the garden. A small door on the top of the pod slowly opens up and back ***(lift head slowly from chest).***

Second Movement:
A spaceman looks out ***(hold head position looking at the ceiling).***

Third Movement:
The door slowly closes again ***(bring head slowly back down to chest).***

Repeat story for exercise with legs crossed left over right as per instructions.

The Bird Asleep:

First Movement:
A bird is asleep on a branch. She snores and lifts her head slowly ***(lift head slowly from chest).***

Second Movement:
She holds her head up ***(hold head position looking at the ceiling),***

Third Movement:
and snoozing again, drops her head slowly down to her chest. ***(Bring head slowly back down to chest).***

Repeat story for exercise with legs crossed left over right as per instructions.

Programme 1

Instructions for Parents

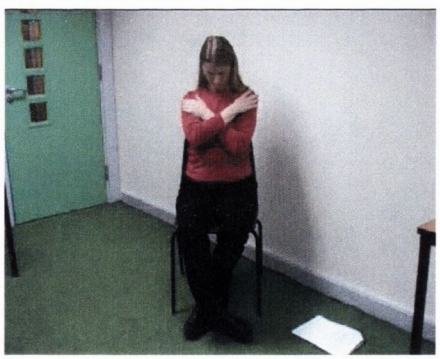
Exercise 3: 4 weeks duration

Exercise 3 should be done by your child every night for 4 weeks. There are stories for each exercise to help make it fun. Sit the child in a low chair so that their feet are flat on the ground before they begin. You might find it most comfortable to sit opposite the child and tell one of the stories on page 30 at a slow pace as you go through each stage of the exercise.

For Exercise 3 follow these steps:

Start Position:

1. Sit the child in a low chair crossing right arm over left placing the hands palms down at the shoulder.
2. Cross right foot over left
3. Place the head down on the chest

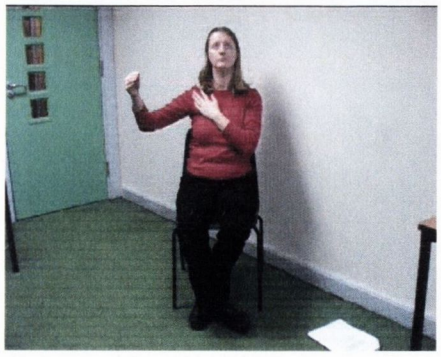


First Movement: Right Side

4. Keeping feet in position, slowly lift the head up and the right arm out simultaneously, so the chin is past the mid-line (but not so far back as to hurt the neck), and look at the ceiling, with the right arm in full extension. At first allow 5 seconds for this movement. Gradually increase the time for this movement over the week so that by the end of week 1 it lasts for 30 seconds and by end of week 2 it lasts for 1 minute.

   Do not worry if this time is not achieved within 2 weeks. Encourage slow head movement and full arm extension until the goal of 1 minute is reached.





Second Movement: Right Side

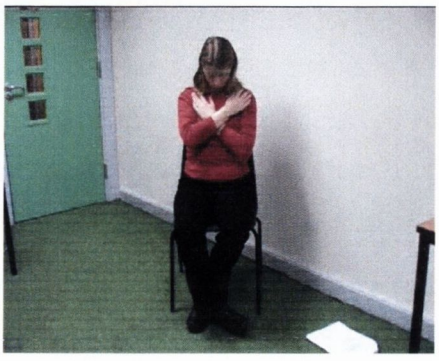
5. Keep the arm and head extended for 10 seconds. One way of doing this is for the child to count to 10 on fingers and thumb ***(thumb to forefinger, then to middle finger, to ring finger and then little finger, then work from little finger back and forward again until you count to 10).***



Do not worry if your child cannot fully manage this, encourage them to continue.

Third Movement: Right Side

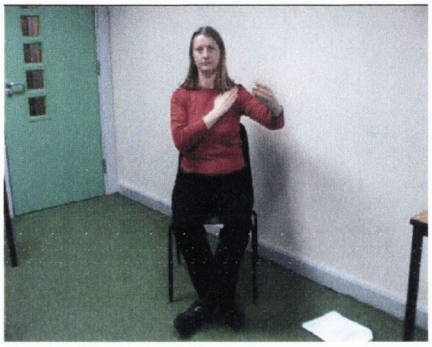
6. Simultaneously bring the head slowly back down to chest and the right arm back to start position. At first allow 5 seconds for this movement. Gradually increase the time for this movement over the week so that by end of week 1 it lasts for 30 seconds and by end of week 2 it lasts for 1 minute.



Now change the child's sitting position and repeat exercise with the head and left arm:

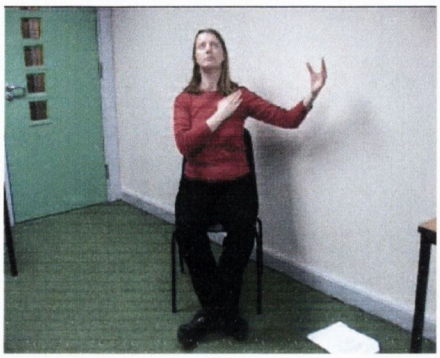
Changing Start Position:

1. Still sitting in the low chair, the child now crosses left arm over right placing the hands palms down at the shoulder.
2. Cross left foot over right
3. Place the head down on the chest



First Movement: Left Side

4. Keeping feet in position, slowly lift the head up and the left arm out simultaneously, so the chin is past the mid-line (but not so far back as to hurt the neck), and look at the ceiling, with the left arm in full extension. At first allow 5 seconds for this movement. Gradually increase the time for this movement over the week so that by the end of week 1 it lasts for 30 seconds and by the end of week 2 it lasts for 1 minute. Do not worry if this time is not achieved within the first two weeks. Encourage slow head and arm extension until the goal of 1 minute is reached.



Second Movement: Left Side

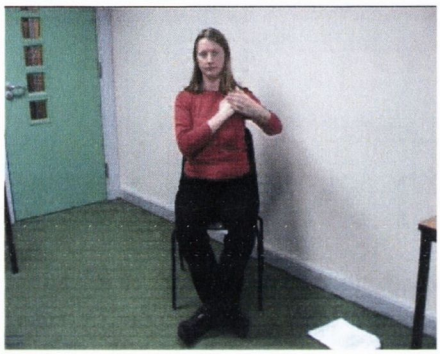
5. Keep the arm and head in this position for 10 seconds. One way of doing this is for the child to count to 10 on fingers and thumb ***(thumb to forefinger, then to middle finger, to ring finger and then little finger, then work from little finger back and forward again until you count to 10).***



Do not worry if your child cannot fully manage this at first, encourage them to continue.

Third Movement: Left Side

6. Simultaneously bring the head slowly back down to the chest and the left arm back to start position. At first allow 5 seconds for this movement. Gradually increase the time for this movement over the week so that by the end of week 1 it lasts for 30 seconds and by the end of week 2 it lasts for 1 minute.



Do not worry if this time is not achieved within the first week. Encourage slow head and arm extension until the goal of 1 minute is reached.

This can be done with eyes closed when mastered.

Goal: **<u>After the first week this exercise should take about 4 minutes per night, moving head up and arm out and back on each side.</u>**

We do want your child to enjoy these exercises. Once your child is familiar with the movements you may wish to use some of the stories overleaf to maintain interest, or make up your own story, or you may prefer not to use a story at all.

Programme 1

Suggested stories for Exercise 3

The Little Daisy Begins to Grow:

First Movement:
The little daisy lifts his head and the petals on his right side at the same time, to warm in the sun. ***(Lift head and right arm slowly from chest).***

Second Movement:
The sun makes him strong ***(hold head position looking at the ceiling).***

Third Movement:
He folds his head and petals closed again. ***(Bring head and arm slowly back down to start position).***

Repeat story for the exercise with head/left arm and legs crossed left over right as per instructions.

The Little Spaceman is not too sure:

First Movement:
The little spaceman opens the door on the top and the door on the right side of his ship ***(lift head and right arm slowly from chest).***

Second Movement:
He checks to see if it is safe to come out ***(hold head and arm position looking at the ceiling).***

Third Movement:
He hears someone coming so he closes the top and side door slowly so as not to be heard ***(bring head and arm slowly back to start position).***

Repeat story for the exercise with head/left arm and legs crossed left over right as per instructions.

Little Bird Wet in the Rain:

First Movement:
The little bird got wet in the rain. She lifts her head and right wing to dry in the sun ***(lift head and right arm slowly from chest).***

Second Movement:
hold position.

Third Movement:
When she is dry she folds her wing and brings her head back down ***(bring head and arm slowly back to start position).***

Repeat story for the exercise with head/left arm and legs crossed left over right as per instructions.

Programme 1

Instructions for Parents

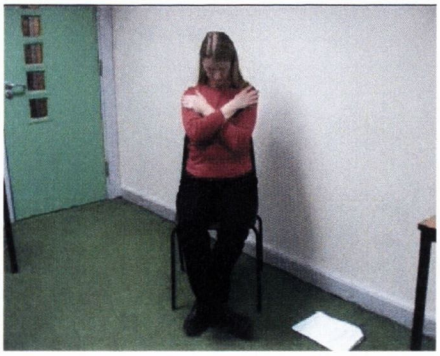
Exercise 4: 4 weeks duration

Exercise 4 should be done by your child every night for 4 weeks. There are stories for each exercise to help make it fun for the children. Sit the child in a low chair so that their feet are flat on the ground before they begin. You may find it comfortable to sit opposite the child and tell one of the stories on page 39 at a slow pace as you go through each stage of the exercise.

For Exercise 4 follow these steps:

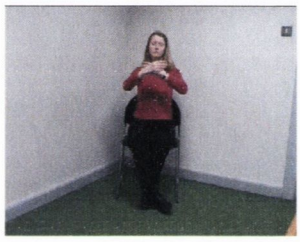
Start Position:

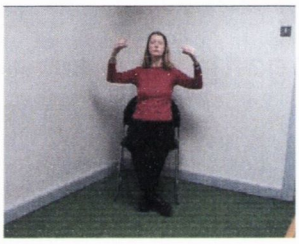
1. Sit the child in low a chair crossing right arm over left placing the hands palms down at the shoulder.
2. Cross right foot over left
3. Place the head down on the chest



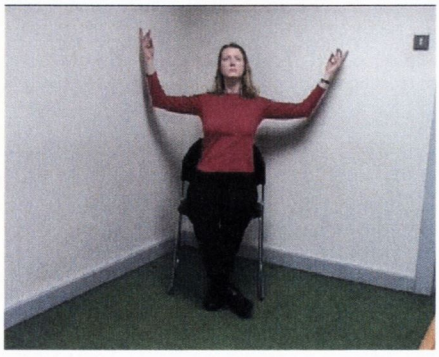
First Movement:

6. Keeping feet in position, slowly lift the head and two arms simultaneously, so the chin is past the mid-line (but not so far back as to hurt the neck), and look at the ceiling with both arms in full extension to shoulder height. At first allow 5 seconds for this movement. Gradually increase the time for this movement over the week so that by the end of week 1 it lasts for 30 seconds and by the end of week 2 it lasts for 1 minute.





Encourage the child to move head slowly and bring both arms slowly to full extension. Do not worry if it takes longer to achieve the 1 minute goal.



Second Movement:

6. Keep the arms and head in this position for 10 seconds. The child should count to 10 on both hands ***(thumb to forefinger, then to middle finger, to ring finger and then little finger, then work from little finger back and forward again until you count to 10).***

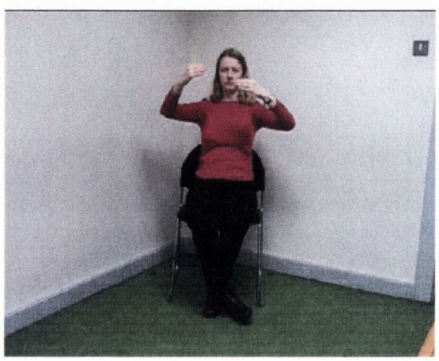
   Do not worry if your child cannot fully manage this at first, encourage them to continue.





Third Movement:

6. Bring head and arms slowly back down to start position. At first allow 5 seconds for this movement. Gradually increase the time for this movement over the week so that by end of week 1 it lasts for 30 seconds and by the end of week 2 it lasts for 1 minute.



Now change the child's sitting position and repeat exercise with the head and both arms:

Changing Start Position:

1. Still sitting in a low chair, the child crosses left arm over right placing the hands palms down at the shoulder.
2. Cross left foot over right
3. Place the head down on the chest



First Movement: Changed Position

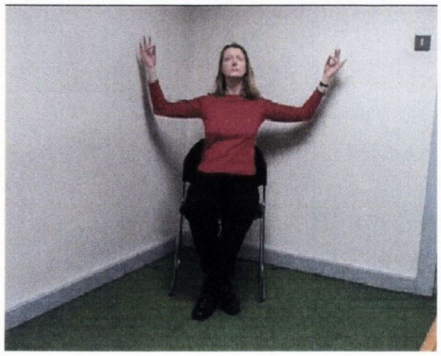
4. Keeping feet in position, slowly lift the head and two arms simultaneously, so the chin is past the mid-line (but not so fat back as to hurt the neck), and look at the ceiling with arms in full extension to shoulder height. At first allow 5 seconds for this movement. Gradually increase the time for this movement over the week so that by the end of week 1 it lasts for 30 seconds and by the end of week 2 it lasts for 1 minute.



Encourage the child to move head slowly and bring both arms slowly to full extension. Do not worry if it takes longer to achieve the 1 minute goal.

Second Movement: Changed Position

5. Keep the arms and head in this position for 10 seconds. The child should count to 10 on both hands ***(thumb to forefinger, then to middle finger, to ring finger and then little finger, then work from little finger back and forward again until you count to 10).***

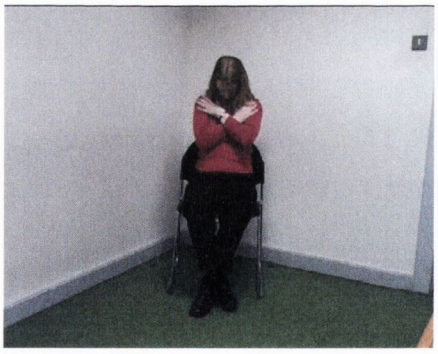


Do not worry if your child cannot fully manage this at first, encourage them to continue.

Third Movement: Changed Position

6. Bring head and arms slowly back down to start position. At first allow 5 seconds for this movement. Gradually increase the time for this movement over the week so that by end of week 1 it lasts for 30 seconds and by the end of week 2 it lasts for 1 minute. Do not worry if this time is not achieved within the first week. Encourage slow head and arm retraction until the goal of 1 minute is reached.





Finish Position

This can be done with eyes closed when mastered.

Goal: Exercise 4 should take 4 minutes per night, moving head up and arms out to full extension and repeat with left foot over right

We do want your child to enjoy these exercises. Once your child is familiar with the movements you may wish to use some of the stories overleaf to maintain interest, or make up your own story, or you may prefer not to use a story at all.

Programme 1

Suggested stories for Exercise 4

The Little Daisy:

First Movement:
The little daisy lifts her head and all her petals to warm in the sun ***(lift head and extend both arms slowly).***

Second Movement:
The sun makes her strong ***(hold head position and arm extension, looking at the ceiling).***

Third Movement:
She drops her head and folds her petals closed again. ***(Bring head and arms slowly back to start position).***

Repeat story for exercise with legs crossed left over right as per instructions.

The Little Spaceship gets ready to fly home:

First Movement:
The little spaceman opens the top door and the side doors of his ship ***(lift head and extend both arms slowly).***

Second Movement:
He checks to see if there is anyone about ***(hold head position and arm extension, looking at the ceiling).***

Third Movement:
It is time to fly home so he closes the top and side doors slowly so as not to be heard ***(bring head and arms slowly back to start position).***

Repeat story for exercise with legs crossed left over right as per instructions.

The Little Bird sunning herself:

First Movement:
The little bird is sunning herself one afternoon. She lifts her head and stretches out both wings to the sun ***(lift head and extend both arms slowly.)***

Second Movement:
Hold position.

Third Movement:
When she is tired she folds her wings and brings her head back down to have a sleep ***(bring head and arms slowly back to start position).***

Repeat story for exercise with legs crossed left over right as per instructions.

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Start Date: _____

Child Name: _____

Day	Exercise	Page	Tick
1	1	6	
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336	4	32	

Day	Exercise	Page	Tick
337	4	32	

End Date:_____

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Comments

Please feel free to comment on any problems encountered on any particular dates of your programme. Please include the relevant date of your comment.

APPENDIX 4



Early Years
Project

INSTRUCTION MANUAL

Movement Programme 2

Dyslexia Early Years Project
Dept. of Psychology
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Dear Parent/Guardian,

Thank you for agreeing to allow your child to take part in the Dyslexia Early Years Project.

This manual is to help guide you in the movement programme you will be doing with your child over the course of the next year. The video-tape illustrates each of the movements described in the manual. It would be useful to look at the video first to get an idea of the movements.

We would like your child to spend 10 minutes each day doing some movements. It may be useful to build them into your daily routine, using a quiet room, perhaps before bedtime.

Refer to the back of your manual for timetable of the exercises. Tick the box once the exercise has been completed by your child each night. Try out your exercise and do not worry if your child does not complete the exercises properly in the first few nights. Remember it is not a memory or learning game. The important thing is that the child concentrates on doing the exercises competently for the required amount of time and eventually becomes accomplished at the movements after a few weeks. The stories are just to help keep your child's interest and keep the movements flowing.

The manual with your timetable filled in, and the video are to be returned to the Dyslexia Research Group at the end of your child's movement programme. They remain the property of Trinity College, Dublin. These materials are only to be used in the context of your child's participation in this study.

Please do not hesitate to contact us at the numbers/emails below if you have any questions about any aspect of the programme. We want you and your child to enjoy this programme and if we can help in that please ask for advice at any time.

This project could have an enormous influence in helping children at risk for dyslexia. Your family's commitment to participation is very much appreciated.

Yours faithfully,

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INSTRUCTIONS TO PARENTS

This movement programme lasts one week and is repeated again each week for 48 weeks. The programme is made up of 2 different sets of games one set called "John's Big Adventure" and one called "Traditional Stories."

You will need some area of the house that has a space at least 4 metres wide where the child can perform his movements. Each movement should be performed in as accurate a way as possible. Static movements (i.e. those that can be performed on the spot) should be continued for up to **10** seconds. Dynamic movements (i.e. those that involve movement along the 4 metre line) should be repeated up to **6** times. That is, up an imaginary line 6 times and down the line 6 times.

Please ensure that the area you use has plenty of protection (e.g. mats, carpets, cushions, pillows, blankets) so that the child does not hurt himself if he should lose his balance.

John's Big Adventure

This set of games is made up of 4 games, one for each day:

- Day 1: John in the jungle.
- Day 2: John and the giant.
- Day 3: John and the animals.
- Day 4: Simon Says.

Traditional Stories

This part of the programme consists of 3 games, one for each day. Unlike the previous section these do not form one continuous story. They are loosely based on traditional stories from other cultures but parts have been adapted to incorporate the necessary movements. The stories are:

- Day 5: The Goddess of Luck.
- Day 6: Lucky Hans
- Day 7: The Magic Doll

These movements should last about 10 minutes each night. If the game does not last that long then it should be extended or even repeated to bring the time up to 10 minutes.

Do not worry if 10 minutes seems too much for your child. Encourage him to do whatever he can without becoming exhausted. Do not worry either if it takes some weeks for him to become familiar with the movements.

For all stories you may, if you wish, use your own child's name instead of the one used here.

John in the Jungle

(Game 1 - day 1)

You may use your child's name instead of John in the story to keep him interested.

Have an imaginary line inside the house about 4 metres long (perhaps the length of the sitting room or bedroom) where the child can perform these movements.

Each movement should be performed in as accurate a way as possible. Dynamic movements (i.e. those that involve movement along a line) should be repeated up to 6 times. Static movements (i.e. those that can be performed on the spot) should be continued for a period of **10** seconds.

Game 1: John in the Jungle

(Instructions)

Once upon a time there was a little boy called John who was in the middle of the jungle and he had to go home for his dinner. But first he had to do lots of things to get out of the jungle.

John sees a river and on the river there is a boat.

John runs down to the river to the boat.
(RUN to warm up – run up and down 6 times).

Carefully he puts one leg into the boat,
(STAND on one foot - 10 seconds).

Then he puts the other leg into the boat.
(STAND on two feet - 10 seconds).

He starts to row while standing, first on the left,
(LEAN to side and SIMULATE ROWING - 10 seconds).

then he rows on the other side.

(LEAN to other side SIMULATE ROWING - 10 seconds).

But then the boat wobbles and wobbles.

(WOBBLE - 10 seconds).

So John sits down in the boat and rows one side,

(KNEEL on knees and sit back on heels - SIMULATE ROWING - 10 seconds).

then the other.

(KNEEL on knees and sit back on heels - SIMULATE ROWING ON OTHER SIDE - 10 seconds).

Then John sees an airplane at the side of the river.

He paddles to the side of the river.

(**PADDLE** twice each side).

John gets out of the boat - first one leg,

(STAND on one foot - 10 seconds).

then the other leg.

(STAND on two feet - 10 seconds).

He runs to the airplane.

(RUN up and down 6 times).

First the airplane needs to be wound up. He winds up the airplane.

(ARM WHIRLS – 10 in one direction and 10 in the other. If your child cannot manage all of this just encourage them to do whatever they can).

Then he gets into the plane, first one foot,

(STAND on one foot - 10 seconds).

then the other.

(STAND two feet together - 10 seconds).

The plane starts to move,

(WALK SLOWLY with arms extended at 90 degrees - 2 lengths).

then it moves faster.

(WALK FASTER, arms still extended - 2 lengths).

then it moves really fast.

(POWER WALK with arms still extended - 2 lengths).

Then the bump, bump, bump and the plane takes off!

(JUMP with 2 feet together, arms still extended - 10 PACES, then RUN back to start).

The plane is flying through the sky.

(RUN with arms still extended – up and down 6 times).

John sees the edge of the forest and decides to land, he slows down.

(From RUN to WALK slow down gradually, with arms still extended).

John lands the plane, first one wheel come down.

(HOP on one foot, arms still extended – 6 paces).

then the other wheel.

(JUMP with 2 feet together, arms still extended - 6 paces).

The plane lands.

(WALK, with arms still extended up and down 6 times along path. Arms may then go down).

John gets out of the plane – first one leg,

(STAND on one foot - 10 seconds).

then the other leg.

(STAND on two feet together - 10 seconds).

John is still not home but now he is tired so he decides to have a rest and continue his journey tomorrow. So John lies down on the ground and goes to sleep.

Estimated time: 4 minutes. Repeat if necessary.

John and the Giant

(Game 2 - day 2)

You may use your child's name instead of John in the story to keep him interested.

Have an imaginary line inside the house about 4 metres long (perhaps the length of the sitting room) where the child can perform these activities.

Each activity should be performed in as accurate a way as possible. Dynamic movements (i.e. those that involve movement along a line) should be repeated up to **6** times. Static movements (i.e. those that can be performed on the spot) should be continued for a period of **10** seconds.

Game 2:John and the Giant

(Instructions)

John sees a giant at the bottom of the hill. The giant is so big maybe he can see John's house and tell him how to get home for his dinner.

John decides to roll down the hill

(ROLL on ground, at first very slow and controlled).

He starts to roll faster and faster

(ROLL faster).

When he gets to the bottom of the hill John sees 2 big fences. He runs at the first fence and takes a big jump and jumps right over the fence.

(JUMP over the fence).

Then he runs at the second fence and takes a big jump and jumps right over the fence.

(JUMP over second fence).

Then he walks up to the big giant.
John asks the giant if he will show him how to get home for his dinner and the giant says he will help John go home for his dinner but only if John shows him what a plane looks like because the giant has never seen a plane.

So John **runs** with arms outstretched to show the giant what a plane looks like.
(RUN up and down 6 times with arms outstretched).

But the giant enjoyed seeing what a plane looked like so much that he wanted John to show him what a train looks like because he has never seen a train either.

So John walks making "chew chew" movements with his arms to show the giant what a train looks like.
(WALK up and down 6 times making "chew chew" noises).

But the giant enjoyed that so much that he wants to know what it is like to ride a horse because he has never seen anyone riding a horse.
So John gallops to show the giant what a horse looks like.

(GALLOP up and down 6 times).

But the giant enjoyed that so much that he wants to know what a car looks like because he has never seen a car either.
So John jogs using his arms to make a steering wheel motion to show the giant what a car looks like.
(JOG up and down 6 times).

But the giant enjoyed that so much he wants to know what a helicopter looks like because he has never seen a helicopter either.
So John twirls to show the giant what a helicopter looks like.
(TWIRL along the path with arms outstretched, once forward in one direction and once back in the other direction).

But the giant enjoyed that so much he wants John to show him what a pogo stick looks like because he has never seen a pogo stick either.
So John bounces to show the giant what a pogo-stick looks like.
(BOUNCE up and down the path 6 times).

Then the giant tells John that he will help him go home for his dinner if he plays a game with him first.

The giant wants John to play a game with him called "Tug of War." The giant says if John wins the game he will help him go home

(PLAY tug of war – continue for a 10 tugs – let child win).

John wins the game.

The giant tells John the animals will help him get home and he will bring John to the animals in his wheelbarrow. John gets in the wheelbarrow.

(WHEELBARROW - 2 lengths)

But then John is so tired again that he has to lie down for another sleep. He decided to wait till tomorrow before letting the giant take him to the animals.

Estimated time: 4 minutes. Repeat if necessary

John and the Animals

(Game 3 - day 3)

You may use your child's name instead of John in the story to keep him interested.

Have an imaginary line inside the house about 4 metres long (perhaps the length of the sitting room) where the child can perform these activities.

Each activity should be performed in as accurate a way as possible. Dynamic movements (i.e. those that involve movement along a line) should be repeated up to 6 times. Static movements (i.e. those that can be performed on the spot) should be continued for a period of 10 seconds.

Game 6:John and the Animals

(Instructions)

First John meets a kangaroo. He asks the kangaroo if he knows how John can get home. The kangaroo doesn't know but he says the gorilla will know and he will help John get to the gorilla. But John must pretend to be a kangaroo if he wants help.

John and the kangaroo skip together to the gorilla's house.
(SKIP up and down 6 times).

They meet the gorilla. John asks if the gorilla knows how he can get home. The gorilla says he doesn't know but that the ants will know and he will help John get to the ants but John must pretend to be a gorilla.

John and the gorilla rock together to the ants' house.
(ROCK up and down from side to side, shifting weight from one leg to the other whilst making gorilla impressions with the arms curled under the armpits - up and down 6 times).

They meet the ants. The ants are very busy. John asks if they know how he can get home. They say they do not know but that the horse might know and they will bring him to the horse but he must pretend to be an ant.
John and the ants march together to the horse's house.
(MARCH up and down 6 times using opposite arm and leg).

They meet the horse. John asks the horse if he knows how John can get home and the horse says he doesn't know but that the frog might know. He will take John to the frog but John must pretend to be a horse.

John and the horse gallop together to the frog's house.
(GALLOP up and down 6 times - *Change leading leg half way*).

They meet the frog. John asks the frog if he knows how John can get home. The frog says he does not know but that the crab might know and he will bring John to the crab but John must pretend to be a frog.

John and the frog leap together to the crab's house.

(LEAP once then CRAWL along rest of path, turn and repeat. Do this up and down 6 times - *do not worry if your child finds this difficult. Encourage them to do only what they can manage*).

They meet the crab. John asks the crab if he knows how he can get home and the crab says that the only person who can help John is the ostrich. He will bring John to the ostrich but John must pretend to be a crab.

John and the crab crawl together to the ostrich's house.
(CRAWL on all 4s up and down 6 times).

They meet the ostrich. John asks the ostrich if he knows how he can get home and the ostrich says he does not know but that the eagle might know and he will bring John to the eagle but first John must pretend to be an ostrich.

John and the ostrich walk step towards the eagle's house.
(WALK up and down 6 times, making each 3rd step a BALANCE ON ONE FOOT - "step, step, hold on one foot, step, step,

hold on one foot etc." Ensure the foot balanced on changes each time).

They meet the eagle. John asks the eagle if he knows how he can get home and the eagle says he does not know but that the giraffe might know and he will bring John to the giraffe but first John must pretend to be an eagle.

John and the eagle fly to the giraffe's house.
(RUN up and down path 6 times, pretending to fly, arms out like wings flapping).

They meet the giraffe. John asks the giraffe if he knows how he can get home and the giraffe says he does not know but that the bat might know and he will bring John to the bat but first John must pretend to be a giraffe.

John and the giraffe walk to the bat's house.
(WALK up and down path 6 times, slowly, raising each leg high alternately).

They meet the bat. John asks the ostrich if he knows how he can get home and the bat says he does not know but that Simon might know and he will bring John to Simon tomorrow but first John must have a rest and go to sleep like a bat.

John and the bat sleep like bats do.

(BEND over and hang head upside-down for 10 seconds, FLAPPING arms like bat wings).

Estimated time: 4 minutes. Repeat if necessary.

Simon Says

(Game 4 - day 4)

You may use your child's name instead of John in the story to keep him interested.

Have an imaginary line inside the house about 4 metres long (perhaps the length of the sitting room) where the child can perform these activities.

Each activity should be performed in as accurate a way as possible. Dynamic movements (i.e. those that involve movement along a line) should be repeated up to **6** times. Static movements (i.e. those that can be performed on the spot) should be continued for a period of **10** seconds.

Game 4: Simon Says

(Instructions)

(To be done twice: first time with eyes open, second with eyes closed.)

John meets Simon. He asks Simon if he knows how John can get home. Simon says he can help John get home but only if he plays a game with him called "Simon Says" where John has to do everything Simon says. John thinks this sounds like a fun game so he agrees.

Simon says clap your hands and John claps his hands.
(STAND STILL & CLAP HANDS x 10).

Simon says do jumping jacks and John does jumping jacks.
(JUMPING JACKS x 10).

Simon says do windmills and John does windmills.
(WINDMILLS x 10. Spin left x 5, spin right x 5 - *if this causes too much dizziness for your child you may omit or reduce it).*

Simon says touch your toes and John touches his toes.

(TOUCH TOES – 10 times. Child is permitted to bend their knees).

Simon says stretch up and John stretches up.

(STRETCH UP – 10 seconds – *keep head in midline, looking straight ahead*).

Simon says stretch to the left and John stretches to the left.

(STRETCH TO LEFT – 5 seconds).

Simon says stretch to the right and John stretches to the right.

(STRETCH TO RIGHT – 5 seconds).

Simon says shake your left leg and John shakes his left leg.

(SHAKE LEG – 5 seconds).

Simon says shake your right leg and John shakes his right leg.

(SHAKE LEG – 5 seconds).

Simon says hang upside down and John hangs upside down.

(HANG HEAD AND UPPER BODY DOWN - 10 seconds).

Simon says sit down, cross legs and bounce one.

(DO BOTH LEGS - 5 seconds each).

Simon says sit on the ground, cross legs and rock.

(ROCK - side to side for 5 seconds, then forward and backward for 5 seconds).

Simon says pretend to be pop-corn.

(BUNNY HOP x 5).

Simon says do a tumble.

(TUMBLE x 2 - *help child if unable*).

Then Simon was so happy with John that he said he would take him home to his mummy and daddy for his dinner. He told John to shut his eyes and then he would find himself at home. So John shut his eyes and when he opened them he was back at home. His mum had made him his favourite dinner. He sat down and told his

mum and dad all about his big adventure in the jungle, with the giant, with the animals and with Simon.

Estimated time: 4 minutes. Repeat if necessary.

THE END

SECTION 2

TRADITIONAL STORIES

(Games 5-7)

The Goddess of Luck

(Game 5 - day 5)

This story is loosely based on a traditional Nepalese story.* Because the actions do not run for the entire length of the story the child may sit down when they are finished and listen to the rest of the story.

You may use your child's name instead of the one in the story if you feel it would help keep your child interested.

Have an imaginary line inside the house about 4 metres long (perhaps the length of the sitting room or bedroom) where the child can perform these activities.

Each activity should be performed in as accurate a way as possible. Dynamic movements (i.e. those that involve movement along a line) should be repeated up to **6** times. Static movements (i.e. those that can be performed on the spot) should be continued for a period of **10** seconds.

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* As retold by Josephine Evetts-Secker (1998) in "The Barefoot Book of Mother and Son Tales," Barefoot Books, Great Britain.

The Goddess of Luck

(Instructions)

Once upon a time high up in the mountains in Nepal there lived anwoman and her son who were very poor. They were so poor that sometimes they had to beg for food. One day the boy decided he did not like being poor and asked his mother why they were so poor. But she did not know why they were so poor so the little boy decided that he would set off to look for the Lord of the Universe to ask him why they were so poor.

So one day the boy set off looking for the Lord of the Universe. He walked for hours and hours and hours.
(Child should WALK up and down the line 6 times).

After walking a long time the boy reached a thick jungle. He was so tired from walking that he stopped and looked around.
(Child should MARCH on spot while the next part of the story is told).

While the boy was standing there along came the god and goddess Siva and Parvati and they saw the little boy standing all alone. The boy told them that he and his mother were very poor and he was trying to find the Lord on the Universe to ask him why they were so poor. Parvati felt sorry for the boy and wanted to give him something nice. But Siva said they could not give the boy anything unless he skipped up and down for them.

(Child should SKIP up and down 6 times along the line).

Then Siva said they could not give the boy anything unless he marched for them.

(Child should MARCH up and down 6 times along the line).

Then Siva said they could not give the boy anything unless he hopped for them.

(Child should HOP, on one foot then the other up and down 6 times along the line – or whatever they can manage).

Then the god and goddess were happy so they gave the boy a gold necklace. The boy was so happy he started to run all the way home to show his mother.

(Child should RUN up and down the line 6 times).

But then the boy got tired and slowed down. Soon he was walking he was so tired.

(Child should WALK up and down the line 6 times).

Then the boy got even more tired and soon he was crawling on the ground.

(Child should CRAWL up and down the line 6 times).

Finally the boy got so tired he decided to have a little sleep. He lay down on the ground and began to sleep.

(Child should lie on the ground and pretend to sleep).

But while the boy was asleep a big eagle flew by and saw the gold necklace sparkling. The eagle swooped down and flew off with the necklace. When the boy woke up he was so sad he cried all the way home.

(Child should WALK up and down line 6 times).

When he got home his mother did not believe his story and they were still really poor. So the boy decided that the next day he would try to find the Lord of the Universe again. When morning

came the boy left the house and marched off with much determination.

(Child should MARCH up and down the line 6 times).

Once again he met the god and goddess Siva and Parvati. He told them his sad story about losing the necklace. Parvati felt sorry for the boy and wanted to give him something else nice. But Siva said they could not give the boy anything unless he did jumping jacks for them.

(Child should do jumping jacks on the spot for 10 seconds).

Then Siva said they could not give the boy anything unless he did lots of stretches for them.

(Child should stretch to the left 10 times, then stretch to the right 10 times, then touch his toes 10 times – child is permitted to bend the knees).

Then the god and goddess gave the boy a diamond ring. The boy was so happy he started to run all the way home to show his mother.

(Child should RUN up and down the line 6 times).

After running for so long the boy was thirsty and so he stopped by a stream to drink the water. But while he was bending over the ring fell out of his pocket and into the stream where it was quickly swallowed by a big fish. The boy was so upset that he cried all the way home.

(Child should WALK up and down the line 6 times).

When he got home his mother did not believe his story and they were still really poor. So the boy decided that the next day he would try to find the Lord of the Universe again. When morning came the boy was feeling a bit better and again full of determination so he left the house and skipped off with much determination.

(Child should SKIP up and down the line 6 times).

Once again he met the god and goddess Siva and Parvati. He told them his sad story about losing the diamond ring. Parvati felt sorry for the boy and wanted to give him something else nice. But Siva said they could not give the boy anything unless he did a funny walk for them.

(Child should SWAY from side to side up and down the line 6 times).

Then Siva said they could not give the boy anything unless he did running jumps for them.

(Child should run then jump once (as if jumping over a fence) each time along the line. Run up and down the line 6 times).

Then Siva said they could not give the boy anything unless he did spins for them.

(Child should SPIN in one direction up and down the line 3 times then in the other direction 3 times – if they cannot manage this encourage them to do only what they can manage).

Then the god and goddess gave the boy some diamonds. The boy was so happy he started to run all the way home to show his mother.

(Child should RUN up and down line 6 times).

But when he got home his mother wasn't there. He left the diamonds in the house and went out looking for her. When he found her he brought her home only to find that a thief had broken into the house and stolen all the diamonds so his mother did not believe his story and they were still poor. So once again

the boy got up the next day determined to find the Lord of the Universe. He left his house and walked a long way.

(Child should WALK up and down line 6 times).

Once again he met the god and goddess Siva and Parvati. He told them his sad story about losing the diamonds. They felt so sorry for him that they decided this time to go to the Goddess of Luck to ask her to help the boy.

At this point the child may sit down and listen to the rest of the story.

The Goddess of Luck heard the story and thought the little boy's spirit was great. She came down to meet him and gave him a copper coin. The little boy was overjoyed and went straight home to show his mother. His mother was very happy. They saw a fisherman walking along the road selling fish so the little boy bought a fish. When he cut open the fish guess what he saw? He saw his diamond ring in the fish's belly! But that was only the first bit of luck because then the little boy needed some wood to stoke the fire to cook the fish so he went outside and climbed a tree. When he got to the top of the tree he saw an eagle's nest and guess what was inside the eagle's nest? His gold necklace! But that was only the second bit of luck because when he got back

home the thief was standing at his front door. The thief had felt so bad about stealing the diamonds that he had decided to give them back to the little boy. So then there were three bits of luck. The little boy now had food and a gold necklace and a diamond ring and lots of diamonds. This is how the Goddess of Luck helped the poor mother and her son to become rich!

Estimated time: 4 minutes. Repeat if necessary.

THE END

Lucky Hans

(Game 6 - day 6)

This story is loosely based on a traditional German story.*

You may use your child's name instead of the one in the story if you feel it would help keep your child interested.

Have an imaginary line inside the house about 4 metres long (perhaps the length of the sitting room) where the child can perform these activities.

Each activity should be performed in as accurate a way as possible. Dynamic movements (i.e. those that involve movement along a line) should be repeated up to **6** times. Static movements (i.e. those that can be performed on the spot) should be continued for a period of **10** seconds.

* As retold by Josephine Evetts-Secker (1998) in "The Barefoot Book of Mother and Son Tales," Barefoot Books, Great Britain.

Lucky Hans

(Instructions)

Once upon a time there was a young boy called Hans who had worked for his master faithfully for several years. One day Hans decided he had worked enough for his master and told him that he now wanted to go home to his mother. His master was sad to let him go but agreed to let him go home and as a reward for having been such a good worker gave him a huge lump of gold.

The lump of gold was so big that Hans could only carry it on his back. When he set off for home he had to carry it on his back. **(Child should WALK up and down the line 6 times pretending to hold the gold on their back, keeping the face forward at all times).**

While he was walking along Hans met a man on horseback. Hans thought it would be great to have a horse and not have to hurt his own feet on the road. So Hans decided to give his gold to the man in return for the horse.

Hans got up on to the horse slowly -first raising one leg over it then straddling it.

(Child should BALANCE on one leg of their choice for about 5 seconds then stand legs apart on both feet in a straddle position for 5 seconds).

When he was up on the horse Hans was delighted and started to canter on the horse.

(Child should GALLOP gently up and down line 6 times, pretending to ride the horse - ensure they change leading leg half way).

Then the horse started to go faster and faster and faster. Soon the horse was galloping along so fast that Hans was having trouble staying on top of the horse.

(Child should RUN fast up and down 6 times pretending to ride the horse).

But then the horse was running so fast that Hans got thrown off the horse. Just then a farmer passed by with a cow. He saw Hans was having problems with the horse and offered to swap the cow for the horse. Hans was happy to get rid of the horse. It was

hard to control it. But now he had a cow! Hans jumped up on the cow.

(Child should BALANCE, as before, on one leg of their choice for about 5 seconds then stand legs apart on both feet in a straddle position for 5 seconds).

The cow began to walk with Hans on top.

(Child should WADDLE from side to side up and down line 6 times).

Then Hans was very thirsty so he decided to milk the cow for a drink.

(Child should bend over and pretend to the milk the cow for 10 seconds).

But nothing would come out of the cow and finally she kicked Hans hard. Just as she kicked the boy a butcher passed by with a pig. The butcher told Hans that the cow was no good for milk but he would swap the pig for the boy's cow if the boy hopped on one foot for him.

(Child should hop on the spot for as long as they can manage).

Then the butcher said he would only swap the cow for the pig if Hans hopped on the other foot, so Hans hopped on the other foot.

(Child should hop on the other foot for as long as they can manage).

Then the butcher said he would only swap the cow for the pig if Hans promised to be kind to the pig and talk to it while crawling along with it. The boy agreed and so Hans and the pig went on their merry way with Hans crawling along side the pig and talking to it.

(Child should CRAWL up and down the line 6 times like the pig).

But then Hans's knees and hands began to get sore. He saw a boy with a goose and thought how much easier it would be to have a goose. So Hans offered to swap the pig for the goose. The boy said he would only swap the goose for the pig if the boy tried to keep the goose amused, because it was a very nice goose and liked to see funny things. So Hans agreed to keep the goose amused and swapped the pig for the goose. When Hans had the goose he did lots of things to make the goose laugh.

(Child should do jumping jacks 10 times).

The goose thought the jumping jacks were very funny but then wanted to see more funny things so Hans tried to walk like the goose to amuse him.

(Childs should WALK up and down the line 6 times flapping arms like wings).

The goose thought that was funny but then wanted to see more funny things so Hans rolled along the ground to try and make the goose laugh.

(Child should ROLL along the ground up and down the line 6 times).

But then Hans was tired of having to make the goose laugh. He saw a scissor-grinder walking along who was whistling merrily to himself. The scissor-grinder told Hans that his job made lots and lots of money. Hans thought it would be nice to have a grindstone so that he too could make lots and lots of money. He told the scissor-grinder that he would like a grindstone and the scissor-grinder told Hans that he would give him one in return for the goose. So Hans gave the scissor-grinder the goose and the

scissor-grinder gave Hans a large stone. So Hans went on his merry way carrying the stone on his back because it was so heavy. **(Child should WALK up and down the line with arms raised 6 times pretending to carry the stone on his back).**

But then the stone got so heavy that Hans couldn't carry it anymore. He stopped to take a drink from a well and as he was drinking he accidentally dropped the stone into the well. But Hans did not mind at all. He was so happy that now he didn't have to carry a heavy stone. So he jumped up and skipped all the way home to his mother thinking how lucky he was not to have anything now to carry.
(Child should JUMP as high as possible, CLAP their hands 5 times and SKIP up and down the line 6 times).

Estimated time: 4 minutes. Repeat if necessary.

THE END

The Magic Doll

(Game 7 - day 7)

This story is loosely based on a traditional Wabanaki story. *

You may use your child's name instead of the one in the story if you feel it would help keep your child interested.

Have an imaginary line inside the house about 4 metres long (perhaps the length of the sitting room) where the child can perform these activities.

Each activity should be performed in as accurate a way as possible. Dynamic movements (i.e. those that involve movement along a line) should be repeated up to **6** times. Static movements (i.e. those that can be performed on the spot) should be continued for a period of **10** seconds.

* As retold by Josephine Evetts-Secker (1998) in "The Barefoot Book of Mother and Son Tales," Barefoot Books, Great Britain.

The Magic Doll

(Instructions)

Once upon a time there was a very good boy called Kayak. One day, when he was old enough, he decided to leave home. His mother was very sad to let him go but knew that he had to grow up. The chief of the tribe saw Kayak leave home and wanted to test how good Kayak was. He hobbled along in front of Kayak and dropped a little box to see if Kayak would be an honest boy and give it back to him. Kayak saw the old man drop the box and was tempted to keep it but instead he picked it up and ran after the old man and gave it back to him. The old man knew then that Kayak was indeed a good boy and told him he could keep the box because it was a lucky box and inside was a magic doll who could grant wishes when Kayak needed them on his journey. Kayak said thank you to the man then went on his journey.

Kayak walked for days and days looking for a village where he might get some food and some rest.

(Child should WALK up and down line 6 times).

Eventually he got to the village of the Micmac people and Kayak went to meet the chief of the village. Because the chief was an important man, Kayak had to bow for a long time in front of him.

(Child should BOW and hold the position for 10 seconds).

The chief had a very beautiful daughter and Kayak immediately fell in love with her. He asked the chief if he could marry his daughter. But the chief said Kayak could only marry his daughter if Kayak could jump as high as the sky. Kayak knew that he could not jump as high as the sky so he got out the lucky box and opened it. He asked the magic doll to make him be able to jump as high as the sky.

(Child should JUMP as high as they can 10 times).

The chief was angry that Kayak was able to jump as high as the sky. Kayak thought that now he would be able to marry the chief's beautiful daughter but the chief said he could only marry his

daughter if he could rock better than a boat. Kayak knew that he could not rock better than a boat so he got out the lucky box and opened it. He asked the magic doll to make him be able to rock better than a boat.

(Child should SIT on the floor knees bent and feet together and ROCK from side to side 10 times).

The chief was angry that Kayak was able to rock better than a boat. Kayak thought that now he would be able to marry the chief's beautiful daughter but the chief said he could only marry his daughter if could fly better than a bird. Kayak knew that he could not fly better than a bird so he got out the lucky box and opened it. He asked the magic doll to make him be able to fly better than a bird.

(Child should hold out their arms at 90 degrees to body and RUN up and down line 6 times flapping their arms like a bird).

The chief was angry that Kayak was able to fly better than a bird. Kayak thought that now he would be able to marry the chief's beautiful daughter but the chief said he could only marry his daughter if he swims like a fish. Kayak knew that he could not swim better than a fish so he got out the lucky box and opened it.

He asked the magic doll to make him be able to swim better than a fish.

(Child should do either breast-stroke or front-crawl like movements with the arms – whichever is easier, as they RUN up and down line 6 times).

The chief was angry that Kayak was able to swim better than a fish. Kayak thought that now he would be able to marry the chief's beautiful daughter but the chief said he could only marry his daughter if he could march better than the best soldier in the army. Kayak knew that he could not march better than the best soldier in the army so he got out the lucky box and opened it. He asked the magic doll to make him be able to march better than the best soldier in the army.

(Child should MARCH up and down line 6 times).

The chief was angry that Kayak was able to march better than the best soldier in the army. Kayak thought that now he would be able to marry the chief's beautiful daughter but the chief said he could only marry his daughter if he could roll better than a ball. Kayak knew that he could not roll better than a ball so he got out

the lucky box and opened it. He asked the magic doll to make him be able to roll better than a ball.

(Child should ROLL on the ground up and down the line up to 6 times if they can manage but fewer if they cannot).

The chief was angry that Kayak was able to roll better than a ball. Kayak thought that now he would be able to marry the chief's beautiful daughter but the chief said he could only marry his daughter if he could shake better than a leaf. Kayak knew that he could not shake better than a leaf so he got out the lucky box and opened it. He asked the magic doll to make him be able to shake better than a leaf.

(Child should SHAKE each limb for 10 seconds – fewer if they cannot manage it, first the arms and then the legs).

The chief was angry that Kayak was able to shake better than a leaf. Kayak thought that now he would be able to marry the chief's beautiful daughter but the chief said he could only marry his daughter if he could hop for a mile. Kayak knew that he could not hop for a mile so he got out the lucky box and opened it. He asked the magic doll to make him be able to hop for a mile.

(Child should HOP up and down the line 6 times, switching feet half way. If the child cannot manage this much encourage them to do whatever they can).

The chief was angry that Kayak was able to hop for a mile. Kayak thought that now he would be able to marry the chief's beautiful daughter but the chief said he could only marry his daughter if he could skip better than a kangaroo. Kayak knew that he could not skip better than a kangaroo so he got out the lucky box and opened it. He asked the magic doll to make him be able to skip better than a kangaroo.

(Child should SKIP up and down the line 6 times).

The chief was angry that Kayak was able to skip better than a kangaroo. Kayak thought that now he would be able to marry the chief's beautiful daughter but the chief said he could only marry his daughter if he could crawl better than a spider. Kayak knew that he could not crawl better than a spider so he got out the lucky box and opened it. He asked the magic doll to make him be able to crawl better than a spider.

(Child should CRAWL on all fours up and down the line 6 times).

The chief was angry that Kayak was able to crawl better than a spider. Kayak thought that now he would be able to marry the chief's beautiful daughter but the chief said he could only marry his daughter if he could leap the length of 10 men. Kayak knew that he could not leap the length of 10 men so he got out the lucky box and opened it. He asked the magic doll to make him be able to leap the length of 10 men.

(Child should take a RUNNING LEAP along the line – run, leap – then turn around and repeat up and down 6 times).

Then the chief was no longer angry. He saw that Kayak obviously had the strength and courage of a great man and he knew this would be a good person for his beautiful daughter to marry. He told Kayak that he was a good boy and that he could now marry his beautiful daughter. Kayak was so happy he ran up and down shouting at the top of his voice and clapping hard.

(Child should RUN up and down line 6 times clapping their hands).

Kayak married the chief's beautiful daughter and they lived happily ever after.

Estimated time: 4 minutes.Repeat if necessary.

THE END

LOG BOOK & TIME-TABLE

Start Date: _____

Day	Exercise	Page	Tick
1	1	6	
2	1	6	
3	1	6	
4	2	12	
5	2	12	
6	2	12	
7	3	17	
8	3	17	
9	3	17	
10	4	23	
11	4	23	
12	4	23	
13	5	29	
14	5	29	
15	5	29	
16	6	38	
17	6	38	
18	6	38	
19	7	45	
20	7	45	
21	7	45	

Day	Exercise	Page	Tick
22	1	6	
23	2	12	
24	3	17	
25	4	23	
26	5	29	
27	6	38	
28	7	45	
29	1	6	
30	2	12	
31	3	17	
32	4	23	
33	5	29	
34	6	38	
35	7	45	
36	1	6	
37	2	12	
38	3	17	
39	4	23	
40	5	29	
41	6	38	
42	7	45	

Day	Exercise	Page	Tick
43	1	6	
44	2	12	
45	3	17	
46	4	23	
47	5	29	
48	6	38	
49	7	45	
50	1	6	
51	2	12	
52	3	17	
53	4	23	
54	5	29	
55	6	38	
56	7	45	
57	1	6	
58	2	12	
59	3	17	
60	4	23	
61	5	29	
62	6	38	
63	7	45	

Day	Exercise	Page	Tick
64	1	6	
65	2	12	
66	3	17	
67	4	23	
68	5	29	
69	6	38	
70	7	45	
71	1	6	
72	2	12	
73	3	17	
74	4	23	
75	5	29	
76	6	38	
77	7	45	
78	1	6	
79	2	12	
80	3	17	
81	4	23	
82	5	29	
83	6	38	
84	7	45	

Day	Exercise	Page	Tick
85	1	6	
86	2	12	
87	3	17	
88	4	23	
89	5	29	
90	6	38	
91	7	45	
92	1	6	
93	2	12	
94	3	17	
95	4	23	
96	5	29	
97	6	38	
98	7	45	
99	1	6	
100	2	12	
101	3	17	
102	4	23	
103	5	29	
104	6	38	
105	7	45	

Day	Exercise	Page	Tick
106	1	6	
107	2	12	
108	3	17	
109	4	23	
110	5	29	
111	6	38	
112	7	45	
113	1	6	
114	2	12	
115	3	17	
116	4	23	
117	5	29	
118	6	38	
119	7	45	
120	1	6	
121	2	12	
122	3	17	
123	4	23	
124	5	29	
125	6	38	
126	7	45	

Day	Exercise	Page	Tick
127	1	6	
128	2	12	
129	3	17	
130	4	23	
131	5	29	
132	6	38	
133	7	45	
134	1	6	
135	2	12	
136	3	17	
137	4	23	
138	5	29	
139	6	38	
140	7	45	
141	1	6	
142	2	12	
143	3	17	
144	4	23	
145	5	29	
146	6	38	
147	7	45	

Day	Exercise	Page	Tick
148	1	6	
149	2	12	
150	3	17	
151	4	23	
152	5	29	
153	6	38	
154	7	45	
155	1	6	
156	2	12	
157	3	17	
158	4	23	
159	5	29	
160	6	38	
161	7	45	
162	1	6	
163	2	12	
164	3	17	
165	4	23	
166	5	29	
167	6	38	
168	7	45	

Day	Exercise	Page	Tick
211	1	6	
212	2	12	
213	3	17	
214	4	23	
215	5	29	
216	6	38	
217	7	45	
218	1	6	
219	2	12	
220	3	17	
221	4	23	
222	5	29	
223	6	38	
224	7	45	
225	1	6	
226	2	12	
227	3	17	
228	4	23	
229	5	29	
230	6	38	
231	7	45	

Day	Exercise	Page	Tick
232	1	6	
233	2	12	
234	3	17	
235	4	23	
236	5	29	
237	6	38	
238	7	45	
239	1	6	
240	2	12	
241	3	17	
242	4	23	
243	5	29	
244	6	38	
245	7	45	
246	1	6	
247	2	12	
248	3	17	
249	4	23	
250	5	29	
251	6	38	
252	7	45	

Day	Exercise	Page	Tick
169	1	6	
170	2	12	
171	3	17	
172	4	23	
173	5	29	
174	6	38	
175	7	45	
176	1	6	
177	2	12	
178	3	17	
179	4	23	
180	5	29	
181	6	38	
182	7	45	
183	1	6	
184	2	12	
185	3	17	
186	4	23	
187	5	29	
188	6	38	
189	7	45	

Day	Exercise	Page	Tick
190	1	6	
191	2	12	
192	3	17	
193	4	23	
194	5	29	
195	6	38	
196	7	45	
197	1	6	
198	2	12	
199	3	17	
200	4	23	
201	5	29	
202	6	38	
203	7	45	
24	1	6	
205	2	12	
206	3	17	
207	4	23	
208	5	29	
209	6	38	
210	7	45	

Day	Exercise	Page	Tick
253	1	6	
254	2	12	
255	3	17	
256	4	23	
257	5	29	
258	6	38	
259	7	45	
260	1	6	
261	2	12	
262	3	17	
263	4	23	
264	5	29	
265	6	38	
266	7	45	
267	1	6	
268	2	12	
269	3	17	
270	4	23	
271	5	29	
272	6	38	
273	7	45	

Day	Exercise	Page	Tick
274	1	6	
275	2	12	
276	3	17	
277	4	23	
278	5	29	
279	6	38	
270	7	45	
281	1	6	
282	2	12	
283	3	17	
284	4	23	
285	5	29	
286	6	38	
287	7	45	
288	1	6	
289	2	12	
290	3	17	
291	4	23	
292	5	29	
293	6	38	
294	7	45	

Day	Exercise	Page	Tick
295	1	6	
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297	3	17	
298	4	23	
299	5	29	
300	6	38	
301	7	45	
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303	2	12	
304	3	17	
305	4	23	
306	5	29	
307	6	38	
308	7	45	
309	1	6	
310	2	12	
311	3	17	
312	4	23	
313	5	29	
314	6	38	
315	7	45	

Day	Exercise	Page	Tick
316	1	6	
317	2	12	
318	3	17	
319	4	23	
320	5	29	
321	6	38	
322	7	45	
323	1	6	
324	2	12	
325	3	17	
326	4	23	
327	5	29	
328	6	38	
329	7	45	
330	1	6	
331	2	12	
332	3	17	
333	4	23	
334	5	29	
335	6	38	
336	7	45	

End Date: _____

Comments

Please feel free to comment on any problems encountered on any particular days of your programme. Please include the relevant date of your comment.

APPENDIX 5



Early Years Project

INSTRUCTION MANUAL

Movement Programme 3

Dyslexia Early Years Project
Dept. of Psychology
TCD
Tel: 608 2095
www.dysvideo.org

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(8) The Slow-Fast Game----------Page 64

Timetable/ Log book----------Page 69

Dear Parent/Guardian,

Thank you for agreeing to allow your child to take part in the Dyslexia Early Years Project.

This manual is to help guide you in the movement programme that you will be doing with your child over the course of the next year. The video-tape illustrates each of the movements described in the manual. It would be useful to look at the video first to get an idea of the movements.

We would like your child to spend 10 minutes each day doing some movements. It may be useful to build them into your daily routine, using a quiet room, perhaps before bedtime.

Refer to the back of your manual for a timetable of the exercises. Tick the box once the exercise has been completed by your child each night. Try out your exercise and do not worry if your child does not complete the exercises properly in the first few nights. Remember it is not a memory or learning game. The important thing is that the child concentrates on doing the exercises competently for the required amount of time and eventually becomes accomplished at the movements after a few weeks. The stories are just to help keep your child's interest and keep the movements flowing.

The manual with your timetable filled in, and the video are to be returned to the Dyslexia Research Group at the end of your child's movement programme. They remain the property of Trinity College Dublin. These materials are only to be used in the context of your child's participation in this study.

Please do not hesitate to contact us at the numbers/emails below if you have any questions about any aspect of the programme. We want you and your child to enjoy this programme and if we can help in that please ask for advice at any time.

This project could have an enormous influence in helping children at risk for dyslexia. Your family's commitment to participation is very much appreciated.

Yours faithfully,

Erika Doyle *edoyle4@tcd.ie* *087 9746032*

Elaine Smith *esmith@tcd.ie* *086 3845473*

Dyslexia Early Years Project
Dept. of Psychology
Aras an Phiarsaigh
Trinity College
Dublin 2.
Tel: 608 2095

DIRECTIONS TO PARENTS

This movement programme lasts a total of 12 months. There are 8 different exercises carried out through stories and games. The sequence of stories and games will be in a random order.

For 4 of the stories the parent must take responsibility for moving the child's limbs and for the 4 face movement games the child has to imitate the parent's face movement or expression.

Parental Help Stories

There are 4 different stories in which the parent is actively engaged.

- 1: The Teddy game
- 2: Mr. Jelly
- 3: Finding Santa
- 4: Walking in the jungle

Parental Help Stories: During these stories the child should lie comfortably on the ground on blanket or rug, or on a bed. Ask the child to let their body 'go floppy'. The parent must take full responsibility for the weight of every limb in every movement. Don't let the child control any movements. Each movement should be held for a

period of approximately 10 seconds. If the parent wishes they may count aloud to 10.

Face Games

There are 4 different games which involve face movements.

- 5: Simon Says
- 6: Party Food
- 7: Snap
- 8: The Slow-Fast Game

The child should sit comfortably in a high backed chair and only move the facial muscles. The parent demonstrates and asks the child to hold the expression for five seconds.

Finally, these exercises should last about 10 minutes each night. If the game does not last that long then it should be extended or even repeated to bring the time up to 10 minutes.

Parental Help Games

The Teddy Game

(Instructions)

This game makes use of 2 teddies or dolls. One teddy (or doll) is put on each side of the child. The game requires the child *to* guess which teddy/doll is where or, with the control of the parent, to exercise the arms and legs.

When in the lying position the child should lie comfortably on the ground on a blanket or rug. When in the sitting position the child should sit comfortably in a high backed chair or fully supported on a bed. The teddies should be placed on either side of the child, far enough away so the child can see both teddies by moving his eyes **but without having to move the head.**

Ask the child to let their body 'go floppy' and to allow the parent to move their limbs. Young children may find this difficult so you may have to demonstrate. The parent must take <u>full</u> responsibility for the weight of every limb in every movement. Don't let the child control <u>any</u> movements. The child's eyes should

be closed while the movement is in progress but can be opened once the child has guessed "which teddy." If the guess is right the parent is recommended to say "very good" in a positive and encouraging way and if wrong the parent should say "oops" or some other such child friendly word. It is very important that the child does not move their head when they open their eyes to look at the teddy. If the teddy is so close he cannot see it without moving his head then move the teddy further away from the child's body.

While the parent is manipulating the child's arms it is suggested that they locate themselves behind the child's head and when manipulating the child's feet and legs they may move to the front of the child. These, however, are only suggestions and if you find a method which is more comfortable (e.g. remaining at the child's feet throughout), it is acceptable to adopt that method.

All manipulations should be held for 10 seconds.

The process should last about 10 minutes. If it is shorter then repeat the game again. All movement of limbs must be done in a slow and deliberate manner.

The Teddy Game

(Story 1 of 4)

Ask the child to lie down and be very still: The parent is to manipulate the arms. Explain to the child that he must guess which teddy is where when asked and that he must practise his arm and legs exercises like teddy does.

Part 1

1. Kneel above the child's head to manipulate arms.
2. The child should shut his eyes.
3. Parent should lift the child's right arm, supporting all weight and point it towards the teddy on the right.
4. Ask the child to guess which teddy he is pointing towards, then allow him to open his eyes. Respond appropriately ("Very good" or "Oops!").
5. Child should shut eyes again and parent should lift the child's left arm, supporting all weight and point it towards the teddy on the left. Ask him to guess which teddy they are pointing at. Respond appropriately. (You may now begin to switch the teddies' positions for at least three times to make the game more fun).
6. Now put a teddy at the end of each foot.
7. Parent should raise the right leg, pointing it towards the

teddy on the right and ask the child to guess teddy.

8. Respond appropriately.
9. Parent should raise the child's left leg, pointing it toward the teddy on the left and ask the child to guess teddy.
10. Respond appropriately.
11. You may now switch the teddies' positions three times to make the game more fun.

Continue in this fashion, moving the teddies from arms to feet and asking the child to guess for as long as you wish or until child tires of game.

Part 2

1. Tell the child that he and teddy must practise their arm exercises so that they can climb trees properly.
2. Parent should hold up one of the child's arms and bend it forwards and backwards. (It is permitted to bend the arm at the elbow).
3. Repeat above with the other arm.
4. Now parent should hold up both the child's arms and bend them backwards and forwards as if climbing a tree.
5. Tell the child that teddy now has to practise his leg exercises so that he can run very fast.

6. Parent should hold up one of the child's legs and in a similar manner bend it at the knee forwards and backwards.
7. Repeat above with the other leg.
8. Now parent should hold up both the child's legs and bend them backwards and forwards as if running.

Part 3

1. Tell the child that teddy has to practise his arm exercises again so that he can swim properly.
2. Parent should hold up one of the child's arms move it up and back then outwards and return to the side, as if doing back-crawl.
3. Repeat above with the other arm.
4. Now parent should hold up both the child's arms and bend them backwards and forwards as climbing a tree.
5. Tell the child that teddy now has to practise his leg exercises again to help his swimming.
6. Parent should hold up one leg, bend it at the knee then move it in a circular motion as if doing the breast-stroke.
7. Repeat above with the child's other leg.

8. Now hold up both the child's legs and bend them backwards and forwards together.

Estimated time: 4 minutes. Repeat if necessary to bring the time up to 10 minutes.

THE END

Mr. Jelly

(Instructions)

Child should begin by standing. A soft comfortable floor with blankets or rugs should be prepared.

The procedure involves enactment of a story, which is narrated by the Parent. The parent should read the script prompted by the word "parent," and should then move the child according to the directions as instructed in the bold type beneath.

Ask the child to let their body 'go floppy' and allow the parent to move their limbs. The parent must take full responsibility for the weight of the child's limbs when manipulating them, except for very beginning of story when child is standing (for purposes of story). All movement of limbs must be done in a slow and deliberate manner.

Mr. Jelly

(Story 2 of 4)

Child should stand while this introduction is being read.

Once upon a time there was a man called Mr. Jelly and his very good friend called Mrs. Nelly. Sometimes Mr. Jelly turns all wobbly and can't stand up!

1) Parent wobbles and gently pretends to fall to floor then encourages child to do the same and gently ensures he "falls" to the ground so he is now in a lying position.

2) **Parent:** "One day Mr. Jelly and Mrs. Nelly are at the seaside and Mr. Jelly wants to go for a swim but he can't stand up!"

3) **Parent:** "Mr. Jelly tries to lift one leg but it wobbles too much."
(Parent lifts child's right leg and gently shakes it from side to side before a gentle return to the ground).

4) **Parent:** "Mr. Jelly tries to lift the other leg but it wobbles too much."
(Parent lifts child's left leg and gently shakes it from side to side before a gentle return to the ground).

5) **Parent**: "Mr. Jelly then tries to lift both legs but that doesn't work either."

(Parent lifts both legs and shakes both of them together before a gentle return to ground.)

6) **Parent:** "But Mr. Jelly wanted to go for a swim so much that Mrs. Nelly decided to help him. She decided to drag him all the way to the sea."

(Parent lifts the child's arms and swings them from side to side, pretending to drag them. Ensure child stays firm and does not move any part of his own body).

7) **Parent:** "When he is in the sea Mrs. Nelly has to help Mr. Jelly to swim. She helps him do the breast-stroke with his arms."

(Parent holds both child's arms and moves them in a circular motion in front of body).

8) **Parent:** "Then she helps him to do the breast-stroke with his legs."

(Parent holds both legs and moves them in a circular motion).

9) **Parent:** "Then she helps him to do the front crawl."

(Parent holds both arms and brings them one at a time towards her in a front-crawl fashion).

10) **Parent:** "Then she helps him to kick his legs."

(Parent picks up legs and moves them in an up and down fashion slowly getting faster and faster).

11) **Parent:** "Then a BIG wave comes" and Mr. Jelly goes under the water."

12) **Parent:** "Under the water he sees lots of fish swimming around."

(Arms of child should be down along sides of body. Hold wrists and flap them outwards and inwards whilst referring to the fish).

13) **Parent:** " When he comes back to the top of the water Mrs. Nelly decides they should go back to the beach and have a picnic so she drags him back to the beach."

(Parent pretends to drag child using legs, as before).

14) **Parent:** "Mrs. Nelly helps Mr. Jelly to dry himself, first she dries his legs,"

(Parents takes one leg and gives it a gentle shake from left to right and then takes the other leg and gives it a gentle shake from left to right).

15) **Parent:** "then she dries his arms."

(Parents takes one arm and gives it a gentle shake from left to right and then takes the other arm and gives it a gentle shake from left to right).

16) **Parent:** "Mrs. Nelly has a picnic. She puts out sandwiches and cakes and lemonade."

17) **Parent:** "Mr. Jelly wants a sandwich so he tries to lift one arm but he can't. He tries once, then he tries again."

(Parent slowly lifts right arm high then drops it with a gentle return to the ground x 2 times).

18) **Parent:** "Mr. Jelly tries lifting his other arm but he can't, he tries once then he tries again."

(Parent slowly lifts left arm high then drops it with a gentle return to the ground x 2 times).

19) **Parent:** "Mrs. Nelly has to help Mr. Jelly. She puts a sandwich in his hand and helps him to eat it."

(Parent pretends to put food in child's hands and moves them alternately to the child's mouth making munching noises).

20) **Parent:** "Then Mr. Jelly tries to get a drink but he can't. He tries lifting his arm once and then again."

(Parent slowly lifts right arm again high then drops with a gentle thud x 2 times).

21) **Parent:** "Mr. Jelly tries with the other arm once then again."

(Parent repeats with the other arm x 2 times).

22) **Parent:** "Mrs. Nelly helps him get a drink."

(Parent pretends to put bottle in child's right hand and moves it towards the mouth, making glugging noises).

- This is end of session, rest is simply for closure).

23) **Parent:** "SUDDENLY Mr. Jelly finds he can move again! He moves one leg then the other."

(Parent encourages child to shake both legs)

24) **Parent:** "Then he moves his arms."

(Parent encourages child to move both arms).

25) **Parent:** "Mr. Jelly gets up. He says thank you to Mrs. Nelly for a lovely day at the sea-side and for being so nice to him and helping him to swim and to eat his lunch. Then he goes home."

Estimated time: 4 minutes. Repeat if necessary to bring the time up to 10 minutes.

THE END

Finding Santa

(Instructions)

A soft comfortable floor with blankets or rugs should be prepared.

Procedure involves enactment of a story, which is narrated by the Parent. The parent should read the script prompted by the word "parent," and should then move the child according to the directions as instructed in the bold type beneath.

Ask the child to let their body 'go floppy' and allow the parent to move their limbs. The parent must take full responsibility for the weight of the child's limbs when manipulating them. All movement of limbs must be done in a slow and deliberate manner.

Finding Santa

(Story 3 of 4)

There once was a little toy soldier who fell out of Santa's sack on Christmas Eve. Santa was very busy rushing around to all the little boys' and girls' houses and so he didn't even notice the soldier fall out of the sack. The soldier didn't know which house he was meant to go to so he decided he should find Santa again as fast as possible. The soldier had to do lots of things before he could find Santa.

1) Child is lying on ground with arms down by sides.
(Parent positions herself above the head of the child).

2) **Parent:** "The soldier lay on the ground wondering where Santa was. He looks all around for Santa, first to the left and then to the right."
(Parent lifts left arm and points it to the left; then lifts the right arm and points it to right. Then lift and point either hand to the front).

3) **Parent:** "The soldier was sure Santa must be somewhere near so he had another good look. He looked, right, left and straight ahead."
(Repeat above a second time).

4) **Parent:** "The soldier couldn't see Santa but he did see a bicycle, so he decided to get on the bicycle and go looking for Santa. He climbs on the bike and puts one foot on the pedal and the other foot on the other pedal."

(Parent moves to end of child and lifts one foot then the other, pretending to put them on the pedals).

5) **Parent:** "Then the soldier began to cycle the bike."

(Holding the 2 feet the Parent begins to circle them in a pedalling motion).

6) **Parent:** "The soldier pedalled faster and faster looking for Santa. He looked everywhere, in gardens, in houses, in shops, he pedals up the hill, he pedals down the hill, but he couldn't find Santa."

(Parent begins to pedal faster, beginning slowly then gradually speeding up. Slow down gradually on cycling motion).

7) **Parent:** "The soldier sees a big river. He wonders if Santa in on the other side of the river. He walks to the river."

(Parent lifts feet and makes walking motion).

8) **Parent:** "At the river the soldier sees a boat. He gets into the boat and sails across the river. The boat is very rocky."

(Child must keep body still. Parent should lift the arms and move them up and down as if wobbling on a rocky boat. These start off gradually).

9) **Parent:** "There were lots of little waves and lots of BIG waves."
(Parent should move the arms gently for the little waves and more pronounced for the big ones).

10) **Parent:** "At the other side of the river the soldier gets out of the boat. He cannot see Santa. He looks all around; to the left, to the right and up and down."
(When speaking Parent again moves right arm to right; then repeats with left arm; then repeats with either arm forwards).

11) **Parent:** "But he cannot see Santa. However, he does see a helicopter. He gets in to the helicopter and it starts to fly. He can see all around."
(Parent holds both arms and raises them slowly along the floor up and down, up and down, above the head and back. Repeat several times while saying...)

12) **Parent:** "...the soldier sees lakes, the soldier sees forests, the soldier sees birds flying in the sky, the soldier sees lots of people, the

soldier sees houses and then do you know what the soldier sees? He sees SANTA!! He sees Santa on the other side of the river."

(All the while be moving the arms up and down).

13) **Parent:** "The soldier lands the helicopter and goes running back to the river."

(Parent simulates running by lifting the feet alternately in a hurried running motion).

14) **Parent:** "The soldier gets into the boat again and sails back through the waves to the other side."

(Parent does same as before, lifting arms and moving them up and down gently for small waves and more so for large ones).

15) **Parent:** "The soldier runs back to the bicycle."

(Parent again lifts feet making a running motion with them).

16) **Parent:** "The soldier gets on to the bike, first one foot and then the other. "

Again, lift first the left and then the right leg as if stepping on to a bicycle.

17) **Parent:** "Then the soldier starts to cycle very fast."

Simulate a cycling motion rotating the feet in a slow then faster cycling manner whilst saying...

18) **Parent:** "...the soldier cycled faster and faster, passed all the houses and passed all the gardens and passed all the shops, hoping he could find Santa.

19) **Parent:** "As the soldier came round a corner do you know who he saw?"
(Ask child. If they say "Santa" reply "Very good, he saw Santa." If they say nothing or get the answer wrong tell them he saw Santa.)

20) **Parent:** "The soldier jumped off the bike and went running up to Santa" (Parent again simulates running by lifting feet and doing an up-down motion). "Santa was very pleased to see him and gave him a big hug"(give child hug). "The soldier was very happy to be back with Santa again."

Estimated time: 4 minutes. Repeat if necessary to bring the time up to 10 minutes.

THE END

Walking in the Jungle

(Instructions)

A soft comfortable floor with blankets or rugs should be prepared.

Procedure involves enactment of a story, which is narrated by the parent.

Ask the child to let their body 'go floppy' and allow the parent to move their limbs. The Parent must take all responsibility for the weight of the child's limbs when manipulating them. All movement of limbs must be done in a slow and deliberate manner.

Walking in the Jungle

(Story 4 of 4)

Once upon a time there was a little boy who wanted to go for a walk in the jungle. (Child is lying comfortably on the ground with arms straight down by sides).

1) **Parent:** "The little boy walked for ages to get to the jungle."
(Parent sits at child's feet and lifts them both alternating their movement up and down, up and down to simulate walking).

2) **Parent:** "Finally the little boy could see the jungle but first he had to walk through a lot of mud. It was very hard to walk through the mud and it took a long time."
(Very slowly the Parent takes the right leg and bending it at the knee moves it upwards then downwards as if it is treading down into mud, then very slowly she lifts the left leg and does the same).

3) **Parent:** "When the little boy got out of the mud he walked into the forest. He could lots and lots of trees. Suddenly it was VERY windy and the trees began to wave around and around."

(Parent lifts both arms together and holding them up, waves them from left to right).

4) **Parent:** "When the wind stopped there was a fairy standing in front of the little boy. The fairy told the little boy that if he clapped his legs 5 times and then clapped his hands 5 times he could see any animal he wanted in the jungle."

5) **Parent:** "The little boy was so excited he immediately clapped his hands and feet 5 times saying he wanted to see lots of birds."
(Parent picks up both feet together and claps them 5 times. Then she picks up both hands and claps them 5 times).

6) **Parent:** "SUDDENLY there were lots of birds in the jungle. The little boy could see birds with big wings flying around."
(Parent holds both of child's arms out straight at 90 degrees to body and very slowly waves arms up and down together 5 times).

7) **Parent:** "Then the little boy could see birds with small wings too."
(Parent bends child's arms at elbow and places hands on hips then moves them more quickly up and down by the elbows).
8) **Parent:** "The little boy then wanted to see monkeys so he clapped his legs then his hands and said that he wanted to see monkeys."

(Parent picks up both feet together and claps them 5 times. Then she picks up both hands and claps them 5 times).

9) **Parent:** "Suddenly the monkeys appeared in the trees and the little boy could see them swinging from tree to tree."
(Parent picks up boy's hands and swings simultaneously them in an exaggerated and slow manner from side to side).

10) **Parent:** "The boy saw a family of monkeys – there was the Daddy monkey **(make very large swinging movements)**, then there was the Mummy monkey **(make slightly smaller swinging movements)**, then there was a boy monkey **(make little swinging movements)** then there was a baby monkey **(make tiny little swinging movements)**.

11) **Parent:** "Then the little boy wanted to see zebras, so he clapped his feet and he clapped his hands and said that he wanted to see zebras."
(Parent claps feet 5 times then hands 5 times).

12) **Parent:** "Suddenly the little boy lots of zebras. They were galloping along **(Parent lifts both legs and rotates them in a cyclical movement)**. "He saw a family of zebras. He saw a Daddy zebra **(make big galloping movements with the legs)**, then he saw a Mummy zebra **(make slightly smaller galloping movements with legs)**, then he saw a

boy zebra **(make little galloping movements)** then he saw a little baby zebra **(make tiny galloping movements).**

13) **Parent:** "Then the little boy wanted to see elephants so he clapped his feet and clapped his hands and wished to see elephants." (Clap hands and feet x 5).

14) **Parent:** "Suddenly there were lots of elephants nearby. The boy could see a family of elephants all with long trunks. He could see a Daddy elephant **(Parent lifts child's right arm and holds it in front of child's face swinging it from side to side as much as possible),** then the little boy saw a Mummy elephant **(repeat as above with slightly smaller movements),** then the little boy saw a boy **elephant (repeat as above with smaller movements),** then the little boy saw a baby elephant **(repeat as above with tiny movements).**

15) **Parent:** "After that the fairy told the little boy that it was time to go home. The little boy was happy because he was very tired. He walked out of the jungle."
(Parent should make walking motions with feet alternately as before).

16) **Parent:** "Then the little boy had to wade back through all the mud" **(Parent should lift child's legs alternately as before making effortful strikes through the mud).**

17) **Parent:** "After that the boy had to walk a long way home again" **(Parent should make walking movements).**

18) **Parent:** "The little boy got home then just in time for bed."

Estimated time: 4 minutes. Repeat if necessary to bring the time up to 10 minutes.

THE END

Face Games

Instructions

Simon Says

This game is very similar to the 'Simon Says' children's game but there is no 'catching out' of the child. The aim is to get the child to copy the faces you are making. Facial movements should be done slowly and should be held for at least 5 seconds.

Child sits in a chair with a high back so that the head is supported. Sit opposite him and ask him to **keep his head and body still** and to copy everything that Simon does. The parent demonstrates. If the child copies the movement properly congratulate him (e.g. saying "very good"). If the child copies it wrongly or ignores it persist until they do it right. Make sure the child can see exactly what you are doing. With the teeth licking exercises especially, open your mouth to allow the child to see what you are doing. The game may be played with the child giving the instructions once they know the particular movements.

Do not worry if it takes time for your child to be able to do each face game.

Simon Says

(Game 1 of 4)

1. **Parent:** "Simon says scrunch up your nose."

(Parent scrunches up nose - ensure that child copies action).

2. **Parent:** "Simon says smile."

(Using the orbital muscles round the mouth smile on both sides of the mouth i.e. smile normally)

3. **Parent:** "Simon says smile with this side of your mouth only."

(Parent smiles with one side of the mouth only. Encourage the child to copy).

4. **Parent:** "Simon says now smile with the other side of the mouth only."

(Parents smiles with the other side of the mouth only - Ensure child smiles on other side and holds for 5 seconds).

5. **Parent:** "Simon says puff out both cheeks and hold."

(Parent puffs out cheeks and holds for 5 seconds.

6. **Parent:** "Simon says puff out one cheek only and hold."

(Parent puffs out one cheek and holds for 5 seconds).

7. **Parent:** "Simon says puff out the other cheek only and hold."

(Parent puffs out the other cheek and holds for 5 seconds - Ensure child has puffed out different cheek).

8. **Parent:** "Simon says bite your lower lip."

(Parent bites lower lip bringing upper jaw over the lower one).

9. **Parent:** "Simon says bite your upper lip."

(Parent bites upper lip, bringing lower jaw up over the upper one).

10. **Parent:** "Simon says stick out your tongue."

(Parent sticks out tongue).

11. **Parent:** "Simon says wave your tongue around."

(Parent sticks out tongue and waves it around).

12. **Parent:** "Simon says lick your chin."

(Parent licks chin with tongue).

13. **Parent:** "Simon says try to reach your nose with your tongue."

(Parent tips nose with tongue if possible. Ask the child to try, do not worry if the child cannot manage it).

14. **Parent:** "Simon says lick your lips."

(Parent licks lips starting at the top and going the whole way around).

15. **Parent:** "Simon says lick your lips this way."

(Parent licks lips in the other direction).

16. **Parent:** "Simon says lick your top teeth."

(Parent licks upper teeth in one direction).

17. **Parent:** "Simon says lick your teeth the other way."

(Parent licks upper teeth in the other direction).

18. **Parent:** "Simon says lick your bottom teeth."

(Parent licks bottom teeth in one direction).

19. **Parent:** "Simon says lick your bottom teeth the other way."

(Parent licks along the lower teeth in the other direction).

20. **Parent:** "Simon says lick all your teeth."

(Parent places tongue between the upper teeth and lips and moves it in one direction and continues down onto the lower teeth and across until a circle has been completed).

Part 2

21. **Parent:** "Simon says lick your bottom teeth this way."

(Parent licks along the lower teeth in one direction).

22. **Parent:** "Simon says puff out this cheek only and hold."

(Parent puffs out one cheek and holds for 5 seconds)

23. **Parent:** "Simon says smile."

(Smile normally)

24. **Parent:** "Simon says try to reach your nose."

(Parent tips nose with tongue if possible).

25. **Parent:** "Simon says smile with this side of your mouth only."

(Parent smiles with one side of the mouth only).

26. **Parent:** "Simon says puff out both cheeks and hold."

(Parent puffs out cheeks and holds for 5 seconds.

27. **Parent:** "Simon says wave your tongue around."

(Parent sticks out tongue and waves it around).

28. **Parent:** "Simon says lick your lips this way."

(Parent licks lips in one direction).

29. **Parent:** "Simon says lick your bottom teeth."

(Parent licks bottom teeth in one direction).

30. **Parent:** "Simon says bite your lower lip."

(Parent bites lower lip bringing upper jaw over the lower one).

31. **Parent:** "Simon says scrunch up your nose."

(Parent scrunches up nose).

32. **Parent:** "Simon says stick out your tongue."

(Parent sticks out tongue).

33. **Parent:** "Simon says lick your lips."

(Parent licks lips starting at the top, going to the right and then the whole way around).

34. **Parent:** "Simon says lick your top teeth."

(Parent licks upper teeth in one direction).

35. **Parent:** "Simon says puff out one cheek only and hold."

(Parent puffs out one cheek and holds for 5 seconds).

36. **Parent:** "Simon says lick your teeth this way."

(Parent licks upper teeth in one direction).

37. **Parent:** "Simon says bite your upper lip."

(Parent bites upper lip, bringing lower jaw up over the upper one).

38. **Parent:** "Simon says lick all your teeth."

(Parent places tongue between the upper teeth and lips and moves it in one direction and continues onto the lower teeth until a circle has been completed).

39. **Parent:** "Simon says now smile with one side of the mouth only."

(Parents smiles with one side of the mouth only).

40. **Parent:** "Simon says lick your chin."

(Parent licks chin with tongue).

Part 3

41. **Parent:** "Simon says lick all your teeth."

(Parent places tongue between the upper teeth and lips and moves it in one direction and continues onto the lower teeth and across until a circle has been completed).

42. **Parent:** "Simon says puff out one only and hold."
(Parent puffs out one cheek and holds for 5 seconds – Ensure child has puffed out different cheek to previous).

43. **Parent:** "Simon says bite your upper lip."

(Parent bites upper lip, bringing lower jaw up over the upper one).

44. **Parent:** "Simon says try to reach your nose."

(Parent tips nose with tongue if she can or does her best).

45. Parent: "Simon says puff out both cheeks and hold."

(Parent puffs out cheeks and holds for 5 seconds).

46. **Parent:** "Simon says wave your tongue around."

(Parent sticks out tongue).

47. **Parent:** "Simon says smile."

(Parent smiles normally).

48. **Parent:** "Simon says lick your bottom teeth."

(Parent licks bottom teeth in one direction).

49. **Parent:** "Simon says scrunch up your nose."

(Parent scrunches up nose).

50. **Parent:** "Simon says lick your lips."

(Parent licks lips starting at the top and going the whole way around).

51. Parent: "Simon says lick your top teeth."

(Parent licks upper teeth in one direction).

52. **Parent:** "Simon says lick your teeth the other way."

(Parent licks upper teeth in the other direction).

53. **Parent:** "Simon says lick your chin."

(Parent licks chin with tongue).

54. **Parent:** "Simon says puff out one cheek only and hold."

(Parent puffs out one cheek and holds for 5 seconds).

55. **Parent:** "Simon says lick your lips this way."

(Parent licks lips in one direction).

56. **Parent:** "Simon says stick out your tongue."

(Parent sticks out tongue).

57. **Parent:** "Simon says lick your bottom teeth this way."

(Parent licks along the lower teeth in one direction).

58. **Parent:** "Simon says now smile with one side of the mouth only."

(Parent smiles with one side of the mouth only).

59. **Parent:** "Simon says smile with this side of your mouth only."

(Parent smiles with the other side of the mouth only).

60. **Parent:** "Simon says bite your lower lip."

(Parent bites lower lip bringing upper jaw over the lower one).

Estimated time: 4 minutes. Repeat if necessary to bring the time up to 10 minutes.

Face Games

Instructions

Party Food

Child sits in a chair with a high back so that the head is supported. Sit opposite him and ask him to keep **his head and body still**. Parent narrates the story and asks child to enact the facial movements as she is talking. For the first week Parent and child should do faces together while story is being narrated. All facial movements should be done slowly and should be held for at least 5 seconds. The child should only move the face muscles, not the head or body.

Do not worry if it takes time for your child to be able to do each face game.

Party Food

(Game 2 of 4)

1) **Parent narrates:** "Once there was a little girl called Suzie. It was Suzie's birthday and her mummy made lots of yummy food for Suzie's dessert. She made a big chocolate cake and lots of ice-cream and lots of sweets. They were all Suzie's favourite foods."

2) **Parent:** "Suzie ran into the kitchen. She could see all the lovely food on the table. She walked over to the chocolate cake. It looked SO good that she started to smile."

(Child should smile on one side of face only, hold for five seconds, then smile on the other side of the face only, hold for five seconds, then break into a proper full smile and hold for five seconds).

3) Parent: "Suzie wanted to smell the chocolate. She took a big sniff of the cake".

(Child should scrunch up the nose and hold for 5 seconds).

4) **Parent:** "Suzie thought the cake looked and smelled so good! She licked her lips."

(Child should lick the lips starting at the top, going in one direction to complete a circle. Then lick the lips in a circle in the other direction).

5) **Parent:** "Suzie picked up some cake and opened her mouth really wide **(child should open mouth, pretending to take a bite, child should not use hands).**
Suzie took such a big bite that it filled one side of her mouth **(child should puff out one cheek and hold it for 5 seconds).** Then she took another big bite and that filled the other side of her mouth **(child should puff other cheek and hold for 5 seconds).** Then Suzie took a REALLY big bite and her entire mouth was full of cake!" **(Child should puff out both cheeks and hold for 5 seconds then pretend to swallow).**

6) **Parent:** "Suzie could feel lots of bits of cake inside her mouth. She licked all around her mouth."

(Child should now lick the upper teeth, in one direction, then the other. Then along the bottom teeth, in one direction, then the other. Then he should place the tongue between the upper teeth and lips and move it in one direction then continue onto the lower teeth until a full circle is completed).

7) **Parent:** "Suzie knew she shouldn't eat ALL the cake. She wanted to save some for her mummy and daddy. But it was really hard not to eat more cake. She bit down on her lower lip to try to stop herself eating any more."

(Child should bite lower lip, bringing the upper jaw over the lower jaw, then he should bite upper lip, bringing lower jaw over upper jaw).

8) **Parent:** "Suzie knew there was cake all over her face. She stuck out her tongue and licked her chin and nose."

(Child should stick out tongue as far as possible, wave it around, reach down and try to lick the chin, then reach up and try to lick the nose).

9) **Parent:** "THEN Suzie saw the ice-cream. Suzie took a big sniff of the ice-cream."

(Child should scrunch up the nose and hold for 5 seconds.)

10) **Parent:** "Suzie thought the ice-cream looked and smelled so good! She licked her lips."

(Child should lick the lips starting at the top, going in one direction to complete a circle. Then lick the lips in a circle in the other direction).

11) **Parent:** "Suzie picked up a bowl of ice-cream and opened her mouth really wide **(child should open mouth, pretending to take a bite, child should not use hands).**

Suzie took such a big spoonful that it filled one side of her mouth **(child should puff out one cheek and hold it for 5 seconds).**

Then she took another big spoonful and that filled the other side of her mouth **(child should puff other cheek and hold for 5 seconds).**

Then Suzie took a REALLY big spoonful and her entire mouth was full of ice-cream! **(Child should puff out both cheeks and hold for 5 seconds then pretend to swallow).**

12) **Parent:** "Suzie could feel lots of ice-cream inside her mouth. She licked all around her mouth."

(Child should now lick the upper teeth, first in one direction, and then the other. Then along the bottom teeth, first in one direction and then the other. Then he should place the tongue between the upper teeth and lips and move it in one direction, then continue onto the lower teeth until a full circle is completed).

13) **Parent:** "Suzie knew she shouldn't eat ALL the ice-cream. She wanted to save some for her mummy and daddy. But it was really hard not to eat more ice-cream. She bit down on her lower lip to try to stop herself eating any more."

(Child should bite lower lip, bringing the upper jaw over the lower jaw, then he should bite upper lip, bringing lower jaw over upper jaw).

14) **Parent:** "Suzie knew there was ice-cream all over her face. She stuck out her tongue and licked her chin and nose."

(Child should stick out tongue as far as possible, wave it around, reach down and try to lick the chin, then reach up and try to lick the nose).

15) **Parent:** "THEN Suzie saw the sweets. Suzie took a big sniff of the sweets."

(Child should scrunch up the nose and hold for 5 seconds.)

16) **Parent:** "Suzie thought the sweets looked and smelled so good! She licked her lips."

(Child should lick the lips starting at the top, going in one direction to complete a circle. Then lick the lips in a circle in the other direction).

17) **Parent:** "Suzie picked up a handful of sweets and opened her mouth really wide **(child should open mouth, pretending to take a bite, child should not use hands).**
Suzie took so many sweets that they filled one side of her mouth **(child should puff out one cheek and hold it for 5 seconds).**
Then she took another big handful and that filled the other side of her mouth **(child should puff other cheek and hold for 5 seconds).**
Then Suzie took a REALLY big handful and her entire mouth was full of sweets! **(Child should puff out both cheeks and hold for 5 seconds then pretend to swallow).**

18) **Parent:** "Suzie could feel lots of bits of sweets inside her mouth. She licked all around her mouth."

(Child should now lick the upper teeth, first in one direction and then the other. Then along the bottom teeth, again in one direction and then the other. Then he should place the tongue between the upper teeth and lips and move it in one direction and then continue onto the lower teeth until a full circle is completed).

19) **Parent:** "Suzie knew she shouldn't eat ALL the sweets. She wanted to save some for her mummy and daddy. But it was really hard not to eat more sweets. She bit down on her lower lip to try to stop herself eating any more."

(Child should bite lower lip, bringing the upper jaw over the lower jaw, then he should bite upper lip, bringing lower jaw over upper jaw).

20) **Parent:** "Suzie knew there were bits of sweets all over her face. She stuck out her tongue and licked her chin and nose."

(Child should stick out tongue as far as possible, wave it around, reach down and try to lick the chin, then reach up and try to lick the nose).

Estimated time: 4 minutes. Repeat if necessary to bring the time up to 10 minutes.

THE END

Face Games

(Game 3 of 4)

Snap/Copy Cat

Child sits in a chair with a high back so that the head is supported. Sit opposite him and ask him to keep **his head and body** still. This game involves both Parent and child making faces at the same time with the Parent going through the specified list of movements and the child attempting to do the same face. The parent counts out loud '1,2,3' and then each does a face.

If the child guesses the next face correctly the Parent should ensure it is performed properly. If the child guesses the wrong face allow them to finish it correctly but then ensure they do the correct face as well. This ensures that they perform all the facial movements

Because of the nature of the game the child should be familiar with the types of movements in this treatment. The Parent should ensure that the child performs each face properly and slowly - All facial movements should be done slowly and should be held for at least 5 seconds.

Do not worry if it takes time for your child to be able to do each face game.

Snap/Copy Cat

Instructions

1) **Parent:** "Can you guess which face I am going to do?"

2) Encourage child to say yes and to make a face from the list. The Parent should make the specific face at the same time as the child.

3) If the child gets the face wrong say "oops" but allow them to continue finishing it then ensure they copy the correct face.

4) If the child gets the face right say "well done."

5) Ask child "What's Next?"

6) Continue until all faces have been pulled.

7) In the first few weeks if the child is finding it difficult to remember the various faces, you can show them (as in the three types of smiles, or three types of puffing cheeks), and then ask which of the three you are going to do.

8) The whole procedure should be completed three times - Sessions 2 and 3 are the same exercises in a different order.

Snap/Copy Cat

(Game 3 of 4)

Faces: Session 1.

Warm up:

Parent: "Can you guess which face I am going to do...something with my nose?"

Parent demonstrates: Scrunch up nose.

Parent: "Snap! Well done!" if the child is right, and **"oops! try again if the child gets it wrong".**

Parent: "Can you guess which face I am going to do...something to do with smiling?"

In the first few weeks if the child is finding it difficult to remember the various faces, you can list them, as below, and ask which of the three you are going to do.

1) Smile a big smile.

2) Smile this side.

3) Smile the other side.

Parent: "Can you guess which face I am going to do...something to do with licking lips?"

4) Lick lips one way in a circle.

5) Lick lips the other way in a circle.

Parent: "Can you guess which face I am going to do...something to do with cheeks?"

6) Puff this cheek.

7) Puff the other cheek.

8) Puff both cheeks.

Parent: "Can you guess which face I am going to do...something to do with my tongue?"

9) Stick out tongue.

10) Wave tongue.

Parent: "Can you guess which face I am going to do...something to do with biting lips?"

11) Bite bottom lip.

12) Bite top lip.

Parent: "Can you guess which way I am going to do a circle?"

13) Place tongue between the upper teeth and lips and move it in a circle in one direction, then continue onto the lower teeth until you come full circle.

14) Place tongue between the upper teeth and lips and move it in a circle in the **other direction**, then continue onto the lower teeth until you come full circle.

Parent: "Can you guess whether I am going to lick chin or nose?"

15) Lick chin.

16) Lick nose.

Parent: "Can you guess which way I am going to lick my teeth?"

17) Lick top teeth.

18) Lick bottom teeth.

19) Lick all teeth in a circle.

Faces: Session 2

Repeat the same movements in your own or the suggested order:

Parent: "Can you guess which face I am going to do...something with my nose?"

Scrunch up nose.

Parent: "Can you guess which way I am going to lick my teeth?"

1) Lick top teeth.

2) Lick bottom teeth.

3) Lick all teeth in a circle.

Parent: "Can you guess whether I am going to lick chin or nose?"

4) Lick chin.

5) Lick nose.

Parent: "Can you guess which way I am going to do a circle?"

6) Place tongue between the upper teeth and lips and move it in a circle in one direction, then continue onto the lower teeth until you come full circle.

7) Place tongue between the upper teeth and lips and move it in a circle in the **other direction**, then continue onto the lower teeth until you come full circle.

Parent: "Can you guess which face I am going to do...something to do with biting lips?"

8) Bite bottom lip.

9) Bite top lip.

Parent: "Can you guess which face I am going to do...something to do with my tongue?"

10) Stick out tongue.

11) Wave tongue.

Parent: "Can you guess which face I am going to do...something to do with cheeks?"

12) Puff this cheek.

13) Puff the other cheek.

14) Puff both cheeks.

Parent: "Can you guess which face I am going to do...something to do with licking lips?"

15) Lick lips one way in a circle.

16) Lick lips the other way in a circle.

Parent: "Can you guess which face I am going to do...something to do with smiling?"

17) Smile a big smile.

18) Smile this side.

19) Smile the other side.

Faces: Session 3

Repeat the same movements in your own or the suggested order:

Parent: "Can you guess whether I am going to lick chin or nose?"

1) Lick chin.

2) Lick nose.

Parent: "Can you guess which face I am going to do...something to do with smiling?"

3) Smile a big smile.

4) Smile this side.

5) Smile the other side.

Parent: "Can you guess which face I am going to do...something to do with biting lips?"

6) Bite bottom lip.

7) Bite top lip.

Parent: "Can you guess which face I am going to do...something to do with cheeks?"

8) Puff this cheek.

9) Puff the other cheek.

10) Puff both cheeks

Parent: "Can you guess which way I am going to do a circle?"

11) Place tongue between the upper teeth and lips and move it in a circle in one direction, then continue onto the lower teeth until you come full circle.

12) Place tongue between the upper teeth and lips and move it in a circle in the **other direction**, then continue onto the lower teeth until you come full circle.

Parent: "Can you guess which face I am going to do...something to do with licking lips?"

13) Lick lips one way in a circle.

14) Lick lips the other way in a circle.

Parent: "Can you guess which face I am going to do...something with my nose?"

14) Scrunch up nose.

Parent: "Can you guess which face I am going to do...something to do with my tongue?"

15) Stick out tongue.

16) Wave tongue.

Parent: "Can you guess which way I am going to lick my teeth?"

17) Lick top teeth.

18) Lick bottom teeth.

19) Lick all teeth in a circle.

Estimated time: 4 minutes. Repeat if necessary to bring the time up to 10 minutes.

THE END

Face Games

Instructions

Slow-Fast

Child sits in a high back chair so his head is supported. Sit opposite him and ask him to keep his **head and body still**. The object of the game is the parent gives a prompt as to whether or not the face should be done slowly or fast, again competing to see who can finish doing it last or first depending on the prompt. The parent gives the prompt as to which face to pull and then both child and parent compete to see who can do it fastest or slowest. It is important that the child complete all exercises properly. The "winning" element is only for entertainment purposes and the parent can let the child win if they like. These can be slow or fast as long as they are repeated a second time with each movement subsequently getting the opposite treatment (i.e. slow or fast). Each movement is done twice. The child winning is at the discretion of the parent but is encouraged. Congratulate them if they "win" and congratulate yourself if you "win."

Do not worry if it takes time for your child to be able to do each face game.

Slow-Fast

(Game 4 of 4)

Prompts are as follows, ask the child to hold each face for 5 seconds.

1) "Can you... scrunch your nose slower than me?"

2) "Can you.. smile (both sides) faster than me?"

3) Smile this side faster?

4) Smile the other faster?

5) "Can you.. puff this cheek slower than me?"

6) Puff the other cheek slower?

7) Puff both cheeks slower

8) "Can you...bite bottom lip faster than me?"

9) Bite top lip faster?

10) "Can you...put tongue out slower?"

11) Wave tongue slower?

12) "Can you...lick chin faster?"

13) Lick nose faster?

14) "Can you...lick lips this way slower?"

15) Lick lips the other way slower

16) "Can you...lick top teeth this way faster?"

17) Lick top teeth the other way faster?

18) "Can you...lick bottom teeth this way slower?"

19) Lick bottom teeth the other way slower?

20) Lick all teeth (in a circle) slower ?

and then try:

1) "Can you ...scrunch your nose faster than me?"

2) "Can you.. smile (both sides) slower' than me?"

3) Smile this side slower?

4) Smile the other slower?

5) "Can you... puff this cheek faster than me?"

6) Puff the other cheek faster?

7) Puff both cheeks faster?

8) "Can you...bite bottom lip slower than me?"

9) Bite top lip slower?

10) "Can you...put tongue out faster?

11) Wave tongue faster?

12) "Can you...lick chin slower?"

13) Lick nose slower?

14) "Can you...lick lips this way faster?"

15) Lick lips the other way faster?

16) "Can you...lick top teeth this way slower than me?"

17) Lick top teeth the other way slower

18) "Can you...lick bottom teeth this way faster than me?"

19) Lick bottom teeth the other way faster?

20) Lick all teeth (in a circle) faster?

Estimated time: 4 minutes. Repeat if necessary to bring the time up to 10 minutes.

THE END

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LOG BOOK & TIME-TABLE

Start Date: _____

Day	Exercise	Page	Tick
1	1	6	
2	1	6	
3	1	6	
4	2	12	
5	2	12	
6	2	12	
7	3	19	
8	3	19	
9	3	19	
10	4	26	
11	4	26	
12	4	26	
13	5	32	
14	5	32	
15	5	32	
16	6	43	
17	6	43	
18	6	43	
19	7	53	
20	7	53	
21	7	53	

Day	Exercise	Page	Tick
22	8	64	
23	8	64	
24	8	64	
25	4	26	
26	3	19	
27	6	43	
28	7	53	
29	1	6	
30	2	12	
31	3	19	
32	8	64	
33	1	6	
34	3	19	
35	8	64	
36	2	12	
37	7	53	
38	8	64	
39	1	6	
40	3	19	
41	1	6	
42	8	64	

Day	Exercise	Page	Tick
43	4	26	
44	7	53	
45	7	53	
46	8	64	
47	7	53	
48	8	64	
49	3	19	
50	6	43	
51	5	32	
52	1	6	
53	4	26	
54	2	12	
55	1	6	
56	7	53	
57	6	43	
58	5	32	
59	1	6	
60	6	43	
61	5	32	
62	6	43	
63	1	6	

Day	Exercise	Page	Tick
64	2	12	
65	1	6	
66	1	6	
67	4	26	
68	3	19	
69	6	43	
70	6	43	
71	7	53	
72	2	12	
73	1	6	
74	7	53	
75	7	53	
76	6	43	
77	5	32	
78	4	26	
79	3	19	
80	1	6	
81	2	12	
82	6	43	
83	7	53	
84	3	19	

Day	Exercise	Page	Tick
85	6	43	
86	1	6	
87	4	26	
88	4	26	
89	1	6	
90	2	12	
91	6	43	
92	4	26	
93	7	53	
94	5	32	
95	1	6	
96	6	43	
97	1	6	
98	7	53	
99	4	26	
100	6	43	
101	6	43	
102	8	64	
103	5	32	
104	4	26	
105	6	43	

Day	Exercise	Page	Tick
106	3	19	
107	5	32	
108	4	26	
109	6	43	
110	7	53	
111	5	32	
112	7	53	
113	2	12	
114	7	53	
115	6	43	
116	2	12	
117	4	26	
118	5	32	
119	7	53	
120	5	32	
121	7	53	
122	4	26	
123	7	53	
124	5	32	
125	7	53	
126	4	26	

Day	Exercise	Page	Tick
127	2	12	
128	2	12	
129	6	43	
130	7	53	
131	5	32	
132	4	26	
133	6	43	
134	5	32	
135	4	26	
136	2	12	
137	7	53	
138	1	6	
139	7	53	
140	3	19	
141	5	32	
142	6	43	
143	6	43	
144	8	64	
145	1	6	
146	6	43	
147	5	32	

Day	Exercise	Page	Tick
148	8	64	
149	2	12	
150	2	12	
151	8	64	
152	6	43	
153	1	6	
154	8	64	
155	1	6	
156	4	26	
157	1	6	
158	4	26	
159	8	64	
160	1	6	
161	3	19	
162	8	64	
163	2	12	
164	6	43	
165	1	6	
166	6	43	
167	5	32	
168	8	64	

Day	Exercise	Page	Tick
169	2	12	
170	1	6	
171	4	26	
172	3	19	
173	1	6	
174	4	26	
175	3	19	
176	4	26	
177	4	26	
178	7	53	
179	5	32	
180	1	6	
181	3	19	
182	3	19	
183	6	43	
184	6	43	
185	7	53	
186	1	6	
187	2	12	
188	3	19	
189	7	53	

Day	Exercise	Page	Tick
190	5	32	
191	7	43	
192	4	26	
193	7	53	
194	4	26	
195	6	43	
196	6	43	
197	3	19	
198	8	64	
199	3	19	
200	3	19	
201	5	32	
202	3	19	
203	6	43	
24	5	32	
205	8	64	
206	4	26	
207	7	53	
208	4	26	
209	4	26	
210	5	32	

Day	Exercise	Page	Tick
211	7	53	
212	3	19	
213	2	12	
214	5	32	
215	6	43	
216	5	32	
217	7	53	
218	3	19	
219	5	32	
220	8	64	
221	4	26	
222	6	43	
223	7	53	
224	4	26	
225	8	64	
226	4	26	
227	7	53	
228	2	12	
229	8	64	
230	7	53	
231	7	53	

Day	Exercise	Page	Tick
232	6	43	
233	3	19	
234	7	53	
235	6	43	
236	5	32	
237	6	43	
238	6	43	
239	5	32	
240	4	26	
241	3	19	
242	3	19	
243	4	26	
244	2	12	
245	4	26	
246	2	12	
247	6	43	
248	3	19	
249	2	12	
250	4	26	
251	8	64	
252	7	53	

Day	Exercise	Page	Tick
253	2	12	
254	6	43	
255	8	64	
256	4	26	
257	2	12	
258	4	26	
259	3	19	
260	5	32	
261	4	26	
262	4	26	
263	4	26	
264	7	53	
265	7	53	
266	5	32	
267	2	12	
268	8	64	
269	5	32	
270	7	53	
271	1	6	
272	3	19	
273	4	26	

Day	Exercise	Page	Tick
274	8	64	
275	6	43	
276	2	12	
277	7	53	
278	4	26	
279	3	19	
270	4	26	
281	3	19	
282	2	12	
283	7	53	
284	8	64	
285	5	32	
286	4	26	
287	1	6	
288	2	12	
289	5	32	
290	8	64	
291	5	32	
292	5	32	
293	3	19	
294	2	12	

Day	Exercise	Page	Tick
295	6	43	
296	1	6	
297	5	32	
298	3	19	
299	7	53	
300	3	19	
301	3	19	
302	8	64	
303	6	43	
304	8	64	
305	8	64	
306	8	64	
307	1	6	
308	6	43	
309	1	6	
310	5	32	
311	3	19	
312	1	6	
313	5	32	
314	7	53	
315	3	19	

Day	Exercise	Page	Tick
316	6	43	
317	5	32	
318	8	64	
319	5	32	
320	4	26	
321	3	19	
322	6	43	
323	5	32	
324	4	26	
325	4	26	
326	7	53	
327	8	64	
328	6	43	
329	7	53	
330	6	43	
331	1	6	
332	5	32	
333	7	53	
334	2	12	
335	8	64	
336	3	19	

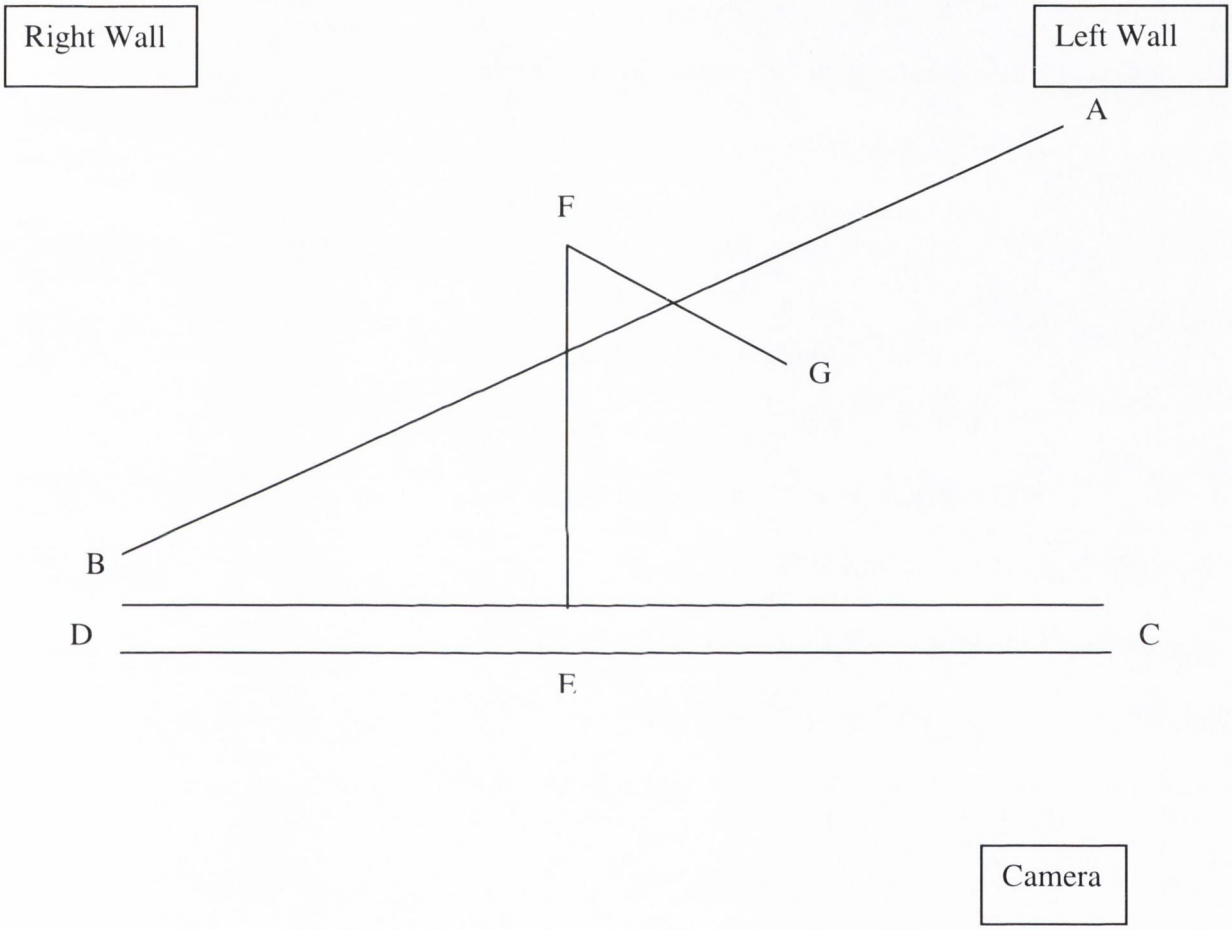
End Date: _____

Comments

Please feel free to comment on any problems encountered on any particular days of your programme. Please include the relevant date of your comment.

APPENDIX 6

Room Lay-out



The testing area had green sheets taped to the walls and floor to allow for easy detection of the child's limbs when scoring. The floor had a diagonal line of white masking tape running 6 feet across the floor (Line A-B). This measured 25 inches out from the left wall and 8 inches out from the right wall. It was 66 inches from the back wall on the left hand side and 4 feet from the back wall on the right hand side.

A 6 foot double line (4 inches wide) ran across the front of the floor, from the left to right wall (Line C-D). A line running perpendicular to this (Line E-F) extended for 28 inches from the centre of the line, just crossing the diagonal line. From "F" at an angle of 45 degrees, ran another line (Line F-G) for 22 inches.

Test positions were as follows:

1) Child stands at "F", facing front of room.

2) Child stands on line F-E.

3) Child walks line C-D.

4) Child walks line C-D.

5) Child walks line A-B.

6) Child stands at "F", facing front of room.

7) Child walks perimeter of room.

8) Child crawls B-A.

9) Child lies on back from E-F, head at F.

10) Child on all fours, from E-F, head at E.

11) Child on all fours, at F and facing right-hand wall.

12) Child stands at "F", facing forward.

13) Child stands at "F", facing "G".

14) Child stands at "F", facing left-hand wall.

APPENDIX 7

DYSLEXIA

Reading / Writing difficulties?

Your participation in an experiment is needed!

The Dyslexia Research Group, located in the Department of Psychology, Trinity College, Dublin, has launched a 1 year experimental study into dyslexia and its treatment.

We are contacting families with children aged 4-6 years, where older brothers/sisters or parents suffer from either dyslexia or reading and spelling problems. We wish to recruit **boys** between 4-6 years and **girls** who may already be presenting with dyslexic symptoms.

Our aim is to investigate early indicators of dyslexia and evaluate 3 different potential treatments for dyslexia, designed for pre-school or early-school children. These treatments (versions of some of them cost well over 1000 Euro) may alleviate the problems associated with dyslexia and our aim is to determine which, if any, of these treatments will be effective. It is of course possible that none of them may be.

Participation in the study may involve a regular commitment of about 10 minutes each day over a period of approximately one year.

Contact Details

For further information or involvement in the study please contact:

Ms. Erika Doyle at edoyle4@tcd.ie or
Ms. Elaine Smith at esmith@tcd.ie or
telephone us during normal office hours on 01-6082095.

APPENDIX 8

Dyslexia Research Group
01-608 2095

October, 2003.

Dear Principal

Re: Dyslexia Early Years Project

The Dyslexia Research Group within the Department of Psychology at Trinity College, Dublin has launched a year-long study into dyslexia. The study is to focus on the different motor treatments that are currently available, some of which are sold as commercial treatments without having been extensively scientifically tested.

For our research study we are contacting parents of young boys between the ages of three and six years who have someone in the family with dyslexia or chronic reading or spelling difficulty.

In order to do that we would be most grateful if you would pass on the enclosed advert and information sheet to the parents of dyslexic children or children with reading and spelling difficulties in your school. This way we may be able to access their younger male siblings. We are also being assisted by the Dyslexia Association of Ireland in contacting relevant families around the country. Interested parents can contact us directly for additional information.

Please refer to the website: **www.dysvideo.org** if you need to download additional copies of our advert and information sheet for your parents. If you have any queries, or need us to send on the information electronically, or simply wish to know more about the study please do not hesitate to contact us via the following:

Elaine Smith esmith@tcd.ie
Erika Doyle edoyle4@tcd.ie

or telephone us during normal office hours on **01-608 2095.**

Many thanks for your help.

Yours sincerely

Elaine Smith Erika Doyle

Erika Doyle
Elaine Smith
Dr. Ray Fuller - Supervisor

APPENDIX 9

Department of Psychology
University of Dublin, Trinity College
Dublin 2, Ireland

Tel: +353 1 608 1886
Fax: +353 1 671 2006

Head of Department: Professor Ruth Byrne

Department of Psychology
Ethics Committee

20 November 2002

Dear Erika Doyle and Elaine Smith,

The Department of Psychology Ethics Committee met recently to consider your application entitled 'Retained primary reflexes and motor development: a longitudinal study of their relationship and of the effects of a remedial programme to stimulate normal primary reflex processes' for approval. I am pleased to tell you that the Committee has decided to approve your application.

Yours sincerely,

Chairperson, Department of Psychology Ethics Committee

APPENDIX 10

QUESTIONNAIRE

Section 1: General

(1) Son's Details

(a) Surname: _____

(b) Forenames: _____

(c) Date of Birth (dd-mm-yyyy): | | | |

(d) Age: | | (Years) | | (Months)

(2) Parent's Details

(a) Surnames

Mother: _____

Father: _____

(b) Forenames

Mother: _____

Father: _____

(3) Contact Information

(a) Address: _____

_____

_____

(b) Phone no: _____

(c) E-mail *(if available):* _____

(d) Mobile *(if available):* _____

Section 2: Child participating

(4) **Child's Developmental History**

(a) The following are a list of the normal motor stages through which a child should pass.
Please tick the box if your child *left any of them out.*

Rolling (front to back) ☐

Rolling (back to front) ☐

Creeping on tummy ☐

Crawling on hands and knees ☐

Sitting alone ☐

Standing alone ☐

Walking ☐

Running ☐

Jumping ☐

(b) If he did not leave any of the previous stages out, what age, in months, was he when he began showing the following behaviours?

Creeping on tummy ☐

Crawling ☐

Sitting alone ☐

Walking ☐

(c) At what age, in months, was he when he began demonstrating the following language abilities?

Imitating sounds (e.g.ma, da): ☐

One word expressions: ☐

Babbling: ☐

Two word 'sentences:' ☐

Three or more word 'sentences:' ☐

(d) Does he know any nursery rhymes? ☐ Yes ☐ No

(e) Does he learn nursery rhymes easily? ☐ Yes ☐ No

(5) Child's Medical History

(a) Does he now or has he ever suffered from any of the following?
Please tick those that apply.

Feeding Problems: ☐

Sleeping Problems: ☐

Hearing Problems: ☐

Visual Problems: ☐

Speech Problems: ☐

Allergies: ☐

Asthma: ☐

Epilepsy: ☐

Other: ☐

Please give details of any of the above:

_____

_____

_____

_____

(6) Child's General Behaviour

(a) Does your child display any of the following behaviours?
Please tick those that apply.

Unusual fussiness ☐

Difficulty to control ☐

Little interest in exploring his environment ☐

Resistance to being held or cuddled ☐

Over-activity ☐

Under-activity ☐

Poor posture ☐

Poor attention ☐

Poor balance / Clumsiness ☐

Frequent falling ☐

Fidgeting ☐

Bed-wetting ☐

Other ☐

Are there any details you wish to add regarding any of the above?

_____

_____

_____

_____

Section 3: Family member with reading/spelling difficulties

(7) <u>Other Family Member</u>

(a) Please specify the other family member who suffers from some form of reading/spelling problem.

Please tick the box that applies.

☐ Mother ☐ Sister

☐ Father ☐ Brother

(b) Has their problem been formally diagnosed as dyslexia by an educational psychologist / agency?

☐ Yes ☐ No

(c) If yes, who was the psychologist / agency who diagnosed the dyslexia?

_____

_____

_____

_____

(d) If no, please specify what it is that suggests this person has a reading / writing problem? Please give details.

_____

_____

_____

_____

APPENDIX 11

Information Sheet for Parents of Participants

Title of the study: Dyslexia Early Years Project

Researchers: Elaine Smith, BSc (Psychology), Erika Doyle, BA (Psychology)
Research Supervisor: Ray Fuller, MA, PhD, FPsSI, FTCD, MESI

Dear Parent,
We are conducting a longitudinal study, designed to look for early indicators of dyslexia in very young children and to assess a number of remedial interventions. We are recruiting up to 400 children between the ages of 3 and 6 years to take part in a study over a one year period. As dyslexia is thought to be a genetic problem (i.e. one that runs in families) we are looking for children from families in which someone has been diagnosed as dyslexic or where there is at least one person with experience of a chronic difficulty with reading and or spelling. We are particularly interested in boys because the prevalence of dyslexia is about three times greater in boys than in girls, however we are interested in girls who are already presenting with problems.

Participant children will be initially assessed in order to determine whether or not they retain certain movement reflexes and to determine their balance skills. They will then be assigned to one of four groups. Three groups will perform different movement exercises with a parent for 10 minutes each evening. One group will be asked to defer implementing any intervention for about 12 months, by which time we should have evidence as to which intervention works best. The parents will then be instructed in that intervention, should they so wish.

As the parent/carer of the child you will be responsible for ensuring that a series of head, arm and leg movements or a balance programme is performed by your child during each session. In addition you will be asked to mark a record sheet, indicating that a session has taken place. We would like you to continue with your movement/balance programme for 12 months. However at 6 months your child will be re-assessed and for some children (perhaps even all) we may suggest that continuation of the programme will not be necessary. In any event, all participant children will be invited back at 12 months for a final assessment.

We appreciate that commitment to this study is a big decision. The main effort involved is the twelve months in which the exercise programmes are taking place. It is extremely important that these exercises are done every night for about 10 minutes during the study period. This information will be most valuable in increasing the knowledge we have of learning disabilities. Having said this, if you do get involved and subsequently feel unhappy being part of this study, you are, of course, at liberty to withdraw at any point that you wish.

If you do feel you would be interested in becoming involved in the study you would be making a great contribution to the knowledge of dyslexia in general as well as being of potential help to your own child's development.

This project satisfies the requirements of the Department of Psychology Ethics Committee and is consistent with Ethical Guidelines of the Psychological Society of Ireland. If you have any queries regarding this study please contact us or our supervisor, Dr. Ray Fuller, at the address below.

Yours sincerely,

Elaine Smith, BSc (Psychology)
Erika Doyle, BA (Psychology)
Department of Psychology
Trinity College, Dublin 2

Tel: 608 2095/608 2426
Fax: 671 2006

APPENDIX 12

Consent Form
Dyslexia Early Years Project

Dear Parent,

As part of our post-graduate doctoral degrees in psychology we (Ms. Elaine Smith and Ms. Erika Doyle) are conducting a long-term study of the motor (i.e. movement) development of children in relation to specific learning difficulties. We are inviting families to take part in our research who feel they are capable of making the commitment necessary for involvement in the study (see attached information sheet) and who meet some other necessary criteria.

The first part of this study will involve a forty minute assessment of your son's physical reflexes and balance skills. Your child will be videotaped whilst completing these elements, to enable later analysis.

If you wish to permit your son to take part in this phase of the study, please sign the consent form below and return to us as soon as possible. Our study satisfies the requirements of the Department of Psychology Ethics Committee and is consistent with Ethical Guidelines of the Psychological Society of Ireland.

Please note that if your child does become involved in the study he will remain anonymous in all reports and confidentiality of individual data is assured. Videotaped material will remain the property of the Department of Psychology and will not be released to any other researcher without your express permission.

Finally, you are at liberty to withdraw your child from the study at any point, should you so wish.

Elaine Smith, BSc (Psychology), Erika Doyle, BA (Psychology), Dr. Ray Fuller, MA, PhD, FPsSI, FTCD, MESI (Research Supervisor)
Department of Psychology
Trinity College
Dublin 2.
Tel: 608 2095/608 2426
Fax: 671 2006

I do wish my child to take part _____
I do not wish my child to take part _____
(Please tick one)

Signature of parent or guardian

_____

Date _____

APPENDIX 13

Dyslexia Early Years Project

This is to confirm that I agree to my child participating in the Dyslexia Early Years Project at his own risk and that I shall not hold Trinity College Dublin or any of its agents liable for any injury associated with that participation

Signed: _____

Date: _____

The Dyslexia Early Years Project satisfies the requirements of the Department of Psychology Ethics Committee and is consistent with Ethical Guidelines of the Psychological Society of Ireland.

APPENDIX 14

Dyslexia Early Years Project

Department of Psychology

Trinity College

Dublin 2.

This is to confirm that I understand that my child is participating in an experiment to evaluate a range of interventions which may help children at risk for dyslexia. However the effectiveness of these interventions cannot be predetermined.

My child is participating with my agreement not to reproduce, lend or otherwise distribute any of the project materials to a third party. I also agree not to instruct other people in the procedures I shall be using.

I note that the video and the handbook are the property of TCD and must be returned to the Dyslexia Research Group on completion of my child's programme.

Signed: _____

Date: _____

APPENDIX 15

DEST-2 Score Sheet for _____

Entering Scores

Having written the test scores (from the response sheet) into the second column of the 'DEST-2 Score Sheet', check the Score Key is for the right age, then place it carefully on top of the Score Sheet. The cutaway allows you to enter the 'At Risk Index' scores in the third column of the score table. Look up the corresponding index (--, -, o, +, ++) in the right hand columns of the Score Key, and enter it in the 'At Risk Index' column. e.g. for rapid naming for age 6:0 to 6:5, you would enter -- for a score of 70; - for a score of 60; o for a score of 55; + for a score of 32; and ++ for a score of 31. The norms on the Score Keys differ for each age, so make sure you use the right one. Note that some of the tests are at ceiling in the 'o' or '+' category, and therefore the higher category has the same score in brackets. If the same score appears in two columns, select the lower category. The score in this category will not be in brackets.

	Test Score	At Risk Index	--	-	o	+	++
1. Rapid Naming							
2. Bead Threading							
3. Phon. Discrimination							
4. Postural Stability							
5. Rhyme/First Letter							
6. Forwards Digit Span							
7. Digit Naming							
8. Letter Naming							
9. Sound Order							
10. Shape Copying							
11. Corsi Frog							
12. Vocabulary							

Overlay the Score Keys for the appropriate age

Interpretation

Work out the numbers of -- and - scores. If 4 or more are --, or 7 or more are either -- or -, the diagnosis is 'at risk'. For a quantitative measure, score 2 for --, 1 for -, 0 for the remainder, and add up the scores. The 'At Risk Quotient' (ARQ) is then the sum divided by 12. An ARQ of 0.9 or greater is strong evidence of being at risk, and an ARQ of 0.6-0.8 is mild evidence of being at risk. Also complete the profile chart. At risk areas such as rhyming suggest areas for possible remediation work. Digit span weakness suggests possible memory difficulties. Discrimination weakness may suggest hearing acuity or processing problems.

Full Name _____

Class _____

School _____

Child's date of birth _____

Age at testing _____

Family history of reading difficulties

(from permission form)_____

Test behaviour: concentration _____

anxiety _____

Other _____

Number with - -_____(A)

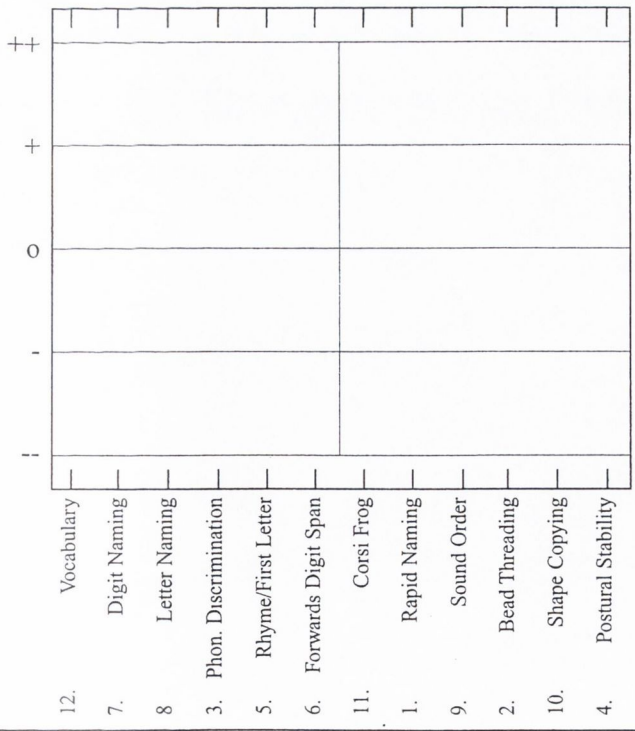
Number with - _____(B)

At risk score (2xA)+B_____(C)

At Risk Quotient (C)/12_____(D)

Screening diagnosis _____

Tester _____ Date _____



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DEST-2 Response Sheet for _____

1. Rapid Naming	2. Bead Threading	3. Phon. Discrimination	4. Postural Stability
Time (secs)	Total	bad dad	Arms by side
Errors	Less initial 3	cap cap	1
Time + (5 x errors)	Minus 1 if string dropped	day day	2
Add 10 if place card needed	more than once	pin thin	
		lip limp	Arms in front
		fat fat	1
		cub cup	2
		leg leg	
		fan van	
Total	Total	Total (max 9)	Total (max 24)

5. Rhyme	6. Forwards Digit Span	7. Digit Naming	8. Letter Naming
leg hen	5 1	4	t
pin win	3 9	8	s
cup pup	1 3 6	3	d
hop doll	4 9 5	9	e
toe toad	7 6 4 2	5	w
mud bud	5 8 4 7	7	o
peg pet	7 4 6 5 8	6	b
wing sing	8 6 1 3 9		q
First letter	7 5 4 3 1 6		n
dog	9 7 5 2 4 3		y
sack	1 3 4 7 9 6 2		
hat	2 1 3 4 8 7 5		
tap	1 7 2 9 6 3 5 8		
net	5 7 6 4 8 1 3 2		
Total (max 13)	Total (max 14)	Total (max 7)	Total (max 10)

9. Sound Order	10. Shape Copying	11. Corsi Frog	12. Vocabulary
mouse	Hand used	4 2	Seesaw
duck		5 1	Pineapple
mouse	ll	1 3 6	Camel
mouse	□	5 3 7	Lizard
duck	=	7 6 4 2	Volcano
mouse	O	6 1 3 4	Light bulb
duck	†	2 4 7 3 5	Autumn
duck	Δ	4 6 1 3 7	Canoe
duck	◇	7 5 4 3 1 6	Jellyfish
mouse		1 7 5 2 4 3	Habit
mouse		1 3 4 7 5 6 2	
duck		2 1 3 4 6 7 5	
duck		1 7 2 4 6 3 5 2	
mouse		5 7 6 4 2 1 3 2	
mouse			
duck			
Total (max 16)	Total (max 21)	Total (max 14)	Total (max 10)

Name: _____

Child ID: _____

Session no: _____

Date: _____Age: Y: ____M:___

SCORE SHEET

COMMENT BOX

See individual test description sheets for scoring procedure

5 = Unable

Oculo-Motor Functioning Tests:

Visual Tracking		**1 2 3 5**
Hand-Eye Tracking		**1 2 3 5**
Fixation with Eyes		**Yes 1 No 5**
Measure start of near Point Convergence and re-establishment of Binocular Vision (range 6-8 cms)		_____ cms
		Yes 1 Slow 4

Motor/Adiadochokinesia tests:

Fingers-to-nose	- eyes open	**1 2 3 4 5**
	- eyes closed	**1 2 3 4 5**
Heel on shin	- left	**1 2 3 4 5**
	- right	**1 2 3 4 5**

rapid alternate movement

Finger tapping (Birdie Song)	- left hand	**1 2 3 4 5**
	- right hand	**1 2 3 4 5**
Palm reversal		**1 2 3 4 5**

Name: _____

Child ID: _____

Session no: _____

Date: _____Age: Y: ____M:__

Finger to thumb (Incy Wincy spider) _____ rotations in ____ secs
Child should attempt up to 10 rotations **Able 1** **Unable 5**

Phonological Tests:

In School	**Yes**	**No**
Letterland Scheme	**Yes**	**No**
Letter Name Knowledge		**/ 26**
Phoneme Deletion		**/ 8**
Non-word Repetition		**/ 10**
		/ 10
		/ 30

Video Data

Balance tests:

Dominant Leg		Right	
		Left	
01	Standing on one leg ***(5 secs @ 4yrs, 6 secs @ 6 years)***	- right	____ secs **1 2 3 4 5**
		- left	____ secs **1 2 3 4 5**
02	(Mann Test) Advanced Romberg ***(min 8 secs)***	- eyes open	____ secs **1 2 3 4 5**
		- eyes closed	____ secs **1 2 3 4 5**
03	Tandem walk *(min 10 steps)*	- forwards	__ / 10 steps

Name: _____

Child ID: _____

Session no: _____

Date: _____ Age: Y: ____ M:___

			1 2 3 4 5
		5 yrs+ - backwards	__ / *10 steps*
			1 2 3 4 5 *
04	Walking on tiptoe *(min 10 steps)*	- forwards	__ / 10 steps
			1 2 3 4 5
		5 yrs+ - backwards	__ / *10 steps*
			1 2 3 4 5 *
05	Slalom Walk *(min 10 steps)*	- forwards	__ / 10 steps
			1 2 3 4 5
		6 yrs+ - backwards	__ / *10 steps*
			1 2 3 4 5 *
06	Hopping on the spot *(min 10 hops)*	- right	___ / 10 hops
			1 2 3 4 5
		- left	___ / 10 hops
			1 2 3 4 5
07	March and about turn		**1 2 3 4 5**
08	Crawling on all fours		**1 2 3 4 5**

Name: _____

Child ID: _____

Session no: _____

Date: _____Age: Y: ____M:__

Balance Score Matrix

Scores	x Amount	Total
1		
2		
3		
4		
5 = Unable		
	Total : - 5 yrs / 11 +5 yrs / 13 + 6 yrs / 14	
	Average	

If average 2 or less = Low Risk

Otherwise High Risk

Name: _____

Child ID: _____

Session no: _____

Date: _____Age: Y: ____M:___

Video Data

Reflex tests:

09 ATNR: Lying Down

Eyes open:	- left arm	**1 2 3 4 5**
	- left leg	**1 4**
	- right arm	**1 2 3 4 5**
	- right leg	**1 4**
Eyes closed:	- left arm	**1 2 3 4 5**
	- left leg	**1 4**
	- right arm	**1 2 3 4 5**
	- right leg	**1 4**

10 ATNR: (Ayres) Table position – front view

Eyes open:	- left arm	**1 2 3 4 5**
	- right arm	**1 2 3 4 5**
Eyes closed:	- left arm	**1 2 3 4 5**
	- right arm	**1 2 3 4 5**

11 STNR: Table position – side view

Eyes open:	- Trunk	**1 2 3 4 5**
	- Arms	**1 2 3 4 5**
Eyes closed:	-Trunk	**1 2 3 4 5**
	- Arms	**1 2 3 4 5**

12 ATNR: (Schilder) Standing position

Eyes open:	- left arm	**1 2 3 4 5**
	- right arm	**1 2 3 4 5**
Eyes closed:	- left arm	**1 2 3 4 5**
	- right arm	**1 2 3 4 5**

Name: _____

Child ID: _____

Session no: _____

Date: _____Age: Y: ___M:__

13 TLR: Standing

Eyes open:	Head down:	**1 2 3 4 5**
	Head back:	**1 2 3 4 5**
Eyes closed:	Head down:	**1 2 3 4 5**
	Head back:	**1 2 3 4 5**

14 MORO: Standing

Eyes open:	**1 2 3 4 5**
Eyes closed:	**1 2 3 4 5**

Reflex Score Matrix

Scores	**x Amount**	**Total**
1		
2		
3		
4		
5 = Unable		
	Total /26	/30
	Average	

If average 2 or less = Low Risk

Otherwise High Risk

Name: _____

Child ID: _____

Session no: _____

Date: _____Age: Y: ____M:___

Group: _____

APPENDIX 16

DST Record Form for _____

Entering Scores

Write the test scores in the 'Test Score' column of the score table below. Find the Score Key appropriate for the child's age. Place it carefully o top of this sheet. The cutaway allows you to enter the 'At Risk Index' scores in the third column of the record form. Look up the correspondin index (---, --, -, o, +) in the right hand columns of the Score Key (using the instructions at the top of the Score Key) and enter it in the 'At Ris column on this sheet. The score keys differ for each age, so make sure you use the right one.

	Test Score	'At Risk' Index	---	--	-	o	+
1. Rapid Naming							
2. Bead Threading							
3. One Minute Reading							
4. Postural Stability							
5. Phonetic Segment"							
6. Two Minute Spelling							
7. Backwards Span							
8. Nonsense Passage							
9. One Minute Writing							
10. Verbal Fluency							
11. Semantic Fluency							

Overlay the score keys for the appropriate age

Interpretation

Work out the numbers of ---, -- and - scores. If 4 or more are --- or --, or 7 or more are either ---, -- or -, the diagnosis is 'At Risk'. For a quantitative measure, score 3 for ---, 2 for --, 1 for -, 0 for the remainder, and add up the scores. The 'At Risk Quotient' is then the sum divided by 10. An ARQ of 1 or greater is strong evidence of being 'At Risk'. Also complete the profile graph. 'At Risk' areas such as Segmentation suggest possible remediation work. Digit Span weakness suggests possible memory difficulties. Areas of strength may provide the basis for a remedial strategy

Full Name _____

Class _____ School _____

Child's date of birth _____

Age at testing _____

Family history (permission form) ☐

Test behaviour: concentration _____

anxiety _____

Other info:

Number with --- _____ (A)

Number with -- _____ (B)

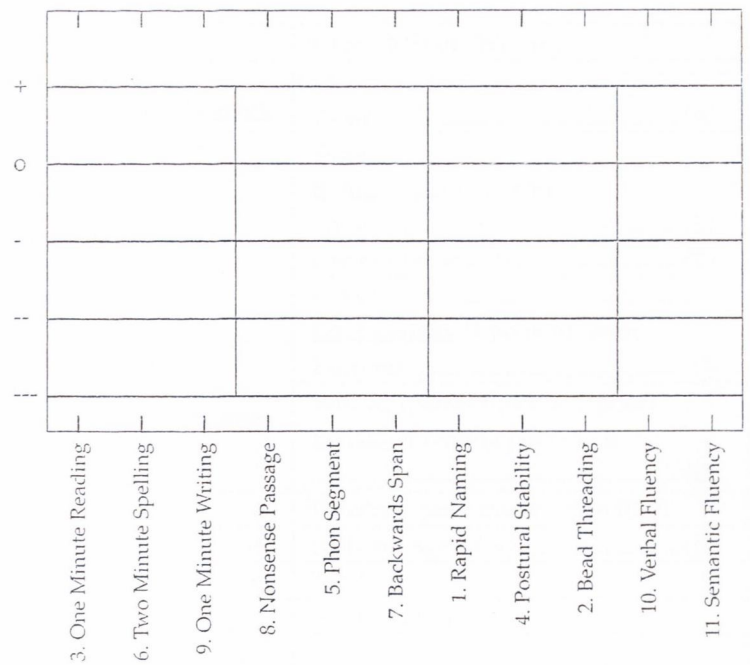
Number with - _____ (C)

'At Risk' score (3xA) + (2xB) + C _____ (D)

'At Risk' quotient (D)/10 _____ (E)

Screening diagnosis _____

Tester _____ Date _____



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DST Record Form for _____

1. Rapid Naming	2. Bead Threading	4. Postural Stability	10. Verbal Fluency	11. Semantic Fluency
Time (sec)	Total	Arms by side	S	Animals
Errors	Less initial 3	1		
		2		
Time + 5 x errors		Arms in front		
		1		
		2		
Total	Total	Total (max 24)	Total	Total

3. One Minute Reading	5. Phonemic Segmentation	6. Two Minute Spelling	7. Back Span
Form (A or B)	1. Rainbow (rain)	Hand used	2 4
Errors	2. Wigwam (wam)	Handwriting quality	6 9
Passes	3. Marmalade (malade)	(good/ave/poor)	8 3 5
Last word read	4. Dog (d)		1 7 6
Words attempted (A)	5. boat (oat)	Number completed	6 9 3 4
Errors plus passes (B)	6. stake (ache)	Number of errors	3 8 1 7
Score (A–B) (C)	7. stake (take)	Number correct	4 1 6 2 3
Time	8. stake (stay)	Add 8 if used only the	2 7 4 6 8
Bonus (1 pt. per sec. under 60)	9. snail (snay)	9.5+ spellings	8 7 1 5 6 9
_____ (D)	10. flag (lag)		4 1 5 2 7 8
Total (C+D)	11. glow (go)		3 8 6 4 1 7 5
	12. igloo (igoo)		5 8 2 3 9 6 1
			6 8 4 5 3 2 1 7
			4 1 5 3 8 7 6 2
Total	Score (max 12)	Total score	Total score

8. Nonsense Passage Reading		9. One Minute Writing
A. One day a mib fell into the feg. He was so sup and tid that he cried. He said "Chig". Then he ran back to his dom. She said "Oh stid "and gave him a reb lim drack		Words _____ (A)
B. One day a mib fell into the feg. So gruny was he that he cried "Ober, give me a throld!" He climbed out, shook the water off, and ran back to his dom. She said "Oh stid " and gave him a reb lim drack		Time Bonus (1 point for each 2s under 60) _____ (B) Lesser of B and 10 _____ (C)
C. Once upon a time a boy ran away from dring. He ran and ran until he fell into a loury greel. So gruny was he that he cried "Ober, give me a throld, remshill me, slossren the widdyfer. At last, he dragged himself out, shook the water off, and quickly went gobbistum.		Errors _____ Error penalty (1 point for each 2 errors) _____ (D)
D. In the olden days, a rennifer set out to craiberg an enormous dollitroy that threatened his country. It was a really gragwally illadonter and after killing it he was chingersomely tired. But the very next day he set out to Oligondervock to graffidanter his stettlenab. On his arrival, he met his bontuvildam at the station. They were married and lived happily ever after		Writing quality (good ave poor) Penalty if writing poor (1–3) _____ (E)
Real Words correct: (max: A = 28, B = 34, C = 42, D = 53)	(A)	Penalty if poor punctuation (0–2)
Nonsense words correct (max = 10)	(B)	(only for age 8:6 +) _____ (F)
Score = (A) + (2 x B)	(C)	Total:
Time		A + C – D – E – F
*Time Bonus (1 per 2 s less than 60 s) [Max 10] _____ *or* Time Penalty (1 per 2 s more than 60 s) [Max 60] _____ **Bonus should only be given if 15 or more is scored.*		
Score after penalty / bonus	(D)	
Half Score (= half of C)	(E)	
Total Score (greater of D and E)		Total Score

Name: _____

Child ID: _____

Session no: _____

Date: _____Age: Y: ____M:___

SCORE SHEET

COMMENT BOX

See individual test description sheets for scoring procedure

5 = Unable

Oculo-Motor Functioning Tests:

Visual Tracking		**1 2 3 5**
Hand-Eye Tracking		**1 2 3 5**
Fixation with Eyes		**Yes 1 No 5**
Measure start of near Point Convergence and re-establishment of Binocular Vision (range 6-8 cms)		_____ cms
		Yes 1 Slow 4

Motor/Adiadochokinesia tests:

Fingers-to-nose	- eyes open	**1 2 3 4 5**
	- eyes closed	**1 2 3 4 5**
Heel on shin	- left	**1 2 3 4 5**
	- right	**1 2 3 4 5**

rapid alternate movement

Finger tapping (Birdie Song)	- left hand	**1 2 3 4 5**
	- right hand	**1 2 3 4 5**
Palm reversal		**1 2 3 4 5**

Name: _____

Child ID: _____

Session no: _____

Date: _____Age: Y: ____M:__

Finger to thumb (Incy Wincy spider) _____ rotations in ____ secs
Child should attempt up to 10 rotations **Able 1** **Unable 5**

Phonological Tests:

In School	**Yes**	**No**
Letterland Scheme	**Yes**	**No**
Letter Name Knowledge		**/ 26**
Phoneme Deletion		**/ 8**
Non-word Repetition		**/ 10**
		/ 10
		/ 30

Video Data

Balance tests:

Dominant Leg — Right / Left

01	Standing on one leg ***(5 secs @ 4yrs, 6 secs @ 6 years)***	- right	____ secs **1 2 3 4 5**
		- left	____ secs **1 2 3 4 5**
02	(Mann Test) Advanced Romberg ***(min 8 secs)***	- eyes open	____ secs **1 2 3 4 5**
		- eyes closed	____ secs **1 2 3 4 5**
03	Tandem walk *(min 10 steps)*	- forwards	__ / 10 steps

Name: _____

Child ID: _____

Session no: _____

Date: _____Age: Y: ____M:___

1 2 3 4 5

5 yrs+ - backwards __ */ 10 steps*

1 2 3 4 5 *

04 Walking on tiptoe - forwards __ / 10 steps
(min 10 steps)

1 2 3 4 5

5 yrs+ - backwards __ */ 10 steps*

1 2 3 4 5 *

05 Slalom Walk - forwards __ / 10 steps
(min 10 steps)

1 2 3 4 5

6 yrs+ - backwards __ */ 10 steps*

1 2 3 4 5 *

06 Hopping on the spot - right ___ / 10 hops
(min 10 hops)

1 2 3 4 5

- left ___ / 10 hops

1 2 3 4 5

07 March and about turn **1 2 3 4 5**

08 Crawling on all fours **1 2 3 4 5**

Name: _____

Child ID: _____

Session no: _____

Date: _____Age: Y: ____M:___

SCORE SHEET

COMMENT BOX

See individual test description sheets for scoring procedure

5 = Unable

Oculo-Motor Functioning Tests:

Visual Tracking		**1 2 3 5**
Hand-Eye Tracking		**1 2 3 5**
Fixation with Eyes		**Yes 1 No 5**
Measure start of near Point Convergence and re-establishment of Binocular Vision (range 6-8 cms)		_____ cms
		Yes 1 Slow 4

Motor/Adiadochokinesia tests:

Fingers-to-nose	- eyes open	**1 2 3 4 5**
	- eyes closed	**1 2 3 4 5**
Heel on shin	- left	**1 2 3 4 5**
	- right	**1 2 3 4 5**

rapid alternate movement

Finger tapping (Birdie Song)	- left hand	**1 2 3 4 5**
	- right hand	**1 2 3 4 5**
Palm reversal		**1 2 3 4 5**

Name: _____

Child ID: _____

Session no: _____

Date: _____Age: Y: ____M:__

Finger to thumb (Incy Wincy spider) _____ rotations in ____ secs
Child should attempt up to 10 rotations **Able 1** **Unable 5**

Phonological Tests:

In School	**Yes**	**No**
Letterland Scheme	**Yes**	**No**
Letter Name Knowledge		**/ 26**
Phoneme Deletion		**/ 8**
Non-word Repetition		**/ 10**
		/ 10
		/ 30

Video Data

Balance tests:

Dominant Leg — Right / Left

01	Standing on one leg ***(5 secs @ 4yrs, 6 secs @ 6 years)***	- right	____ secs **1 2 3 4 5**
		- left	____ secs **1 2 3 4 5**
02	(Mann Test) Advanced Romberg ***(min 8 secs)***	- eyes open	____ secs **1 2 3 4 5**
		- eyes closed	____ secs **1 2 3 4 5**
03	Tandem walk *(min 10 steps)*	- forwards	__ / 10 steps

Name: _____

Child ID: _____

Session no: _____

Date: _____Age: Y: ____M:___

1 2 3 4 5

5 yrs+ - backwards __ / *10 steps*

1 2 3 4 5 *

04 Walking on tiptoe *(min 10 steps)* - forwards __ / 10 steps

1 2 3 4 5

5 yrs+ - backwards __ / *10 steps*

1 2 3 4 5 *

05 Slalom Walk *(min 10 steps)* - forwards __ / 10 steps

1 2 3 4 5

6 yrs+ - backwards __ / *10 steps*

1 2 3 4 5 *

06 Hopping on the spot *(min 10 hops)* - right ___ / 10 hops

1 2 3 4 5

- left ___ / 10 hops

1 2 3 4 5

07 March and about turn **1 2 3 4 5**

08 Crawling on all fours **1 2 3 4 5**

Name: _____

Child ID: _____

Session no: _____

Date: _____Age: Y: ____M:__

Balance Score Matrix

Scores	x Amount	Total
1		
2		
3		
4		
5 = Unable		
	Total : - 5 yrs / 11 +5 yrs / 13 + 6 yrs / 14	
	Average	

If average 2 or less = Low Risk

Otherwise High Risk

Name: _____

Child ID: _____

Session no: _____

Date: _____Age: Y: ____M:___

Video Data

Reflex tests:

09	ATNR: Lying Down	Eyes open:	- left arm	**1 2 3 4 5**
			- left leg	**1 4**
			- right arm	**1 2 3 4 5**
			- right leg	**1 4**
		Eyes closed:	- left arm	**1 2 3 4 5**
			- left leg	**1 4**
			- right arm	**1 2 3 4 5**
			- right leg	**1 4**
10	ATNR: (Ayres) Table position – front view			
		Eyes open:	- left arm	**1 2 3 4 5**
			- right arm	**1 2 3 4 5**
		Eyes closed:	- left arm	**1 2 3 4 5**
			- right arm	**1 2 3 4 5**
11	STNR: Table position – side view			
		Eyes open:	- Trunk	**1 2 3 4 5**
			- Arms	**1 2 3 4 5**
		Eyes closed:	-Trunk	**1 2 3 4 5**
			- Arms	**1 2 3 4 5**
12	ATNR: (Schilder) Standing position			
		Eyes open:	- left arm	**1 2 3 4 5**
			- right arm	**1 2 3 4 5**
		Eyes closed:	- left arm	**1 2 3 4 5**
			- right arm	**1 2 3 4 5**

Name: _____

Child ID: _____

Session no: _____

Date: _____Age: Y: ____M:__

13 TLR: Standing

Eyes open:	Head down:	**1 2 3 4 5**
	Head back:	**1 2 3 4 5**
Eyes closed:	Head down:	**1 2 3 4 5**
	Head back:	**1 2 3 4 5**

14 MORO: Standing

Eyes open:	**1 2 3 4 5**
Eyes closed:	**1 2 3 4 5**

Reflex Score Matrix

Scores	**x Amount**	**Total**
1		
2		
3		
4		
5 = Unable		
	Total /26	/30
	Average	

If average 2 or less = Low Risk

Otherwise High Risk

Name: _____

Child ID: _____

Session no: _____

Date: _____Age: Y: ____M:___

Group: _____

APPENDIX 17

Dyslexia Early Years Project

Results from the Dyslexia Early Screening Test

Name

10-06-04

The Dyslexia Early Screening Tests (DEST-2) is designed for children between the ages of 4:6 – 6:5 years. It consists of 12 different tests on which dyslexic children may perform poorly. Though it is not a diagnosis of dyslexia itself, the results provide an 'at risk' score for dyslexia and a profile of strengths and weaknesses which may be used as a guide for the child's academic support in school.

Screening Tests

1. Rapid naming: This involves correctly naming different pictures as quickly as possible.
2. Bead threading: This represents a test of fine motor skill requiring hand-eye coordination.
3. Phonological discrimination: This tests the child's ability to judge the sounds of a word.
4. Postural stability: This involves a gentle push in the back to determine whether the child has a problem with maintaining balance.
5. Rhyme: This assesses the child's ability to detect rhyme.
6. Forwards digit span: This is a test of working memory, requiring the child to listen to a series of numbers then repeat them back in the same order.
7. Digit naming: This tests the child's knowledge of numbers.
8. Letter naming: This tests the child's knowledge of letters.
9. Sound order: This gives an indication of whether the child may have difficulty detecting rapid changes in consonants by testing whether they can determine the order in which 2 different tones are presented.
10. Shape copying: This tests the ability to write neatly and fast.

11. The Frog test: This tests the child's spatial memory.
12. Vocabulary: This tests the child's word knowledge.

According to the scoring criteria, XXXX's scores were as follows.

TEST	**RESULT**
Rapid naming	Below average
Bead threading	Average
Phonological discrimination	Average
Postural stability	Well above average
Rhyme	Above average
Forwards digit span	Average
Digit naming	Average
Letter naming	Average
Sound order	Average
Shape copying	Well above average
Spatial memory	Average
Vocabulary	Well above average

An 'At Risk Quotient' of 0.9 or greater is strong evidence that a child is 'at risk' of developing dyslexia. However, XXXX's score was only 0.08, which suggests that the risk of him developing dyslexia is extremely low.

Having said this, and given that there is a family history of dyslexia, if you are ever concerned about XXXX's subsequent progress in school, and you suspect that the problem may be related to dyslexia, you can seek to have him assessed, either through the school with the National Educational Psychological Service (N.E.P.S.), or privately. Information regarding private assessment can be sought from the Dyslexia Association of Ireland (D.A.I.). Please be aware that there may be long waiting lists for such assessments regardless of whether you go through N.E.P.S. or privately. Furthermore, children are not usually suitable for testing before they are 6 years old.

APPENDIX 18

Dyslexia Early Years Project

Results from the Dyslexia Screening Test

Name

10-06-04

The Dyslexia Screening Tests is designed for children between the ages of 6:6 – 16:5 years and, though it is not a diagnosis of dyslexia itself, provides an indication (an 'At Risk' Quotient) of whether a further, full psycho-educational assessment should be sought. It includes tests of attainment (reading, writing & spelling) as well as screening tests for dyslexia which cover a range of skills usually problematic for dyslexic children.

Attainment Tests

1. Reading test
2. Spelling test
3. Writing test

Screening Tests

4. Rapid naming: This involves correctly naming different pictures as quickly as possible.
5. Bead threading: This represents a test of fine motor skill requiring hand-eye coordination.
6. Postural stability: This involves a gentle push in the back to determine whether the child has a problem with maintaining balance.
7. Phonetic segmentation: This assesses the child's phonological skill, that is, their ability to break down the sounds of a word, as well as assessing their short-term memory.
8. Backwards digit span: This is a test of working memory, requiring the child to listen to a series of numbers then repeat them back in reverse order.

9. Nonsense passage reading: This tests the child's ability to read words with which they are not familiar, such that they must break down the word into chunks to work out how it sounds.
10. Semantic & Verbal fluency: These tests measure the ability to generate a list of words from some category, for example, food (semantic fluency) and the ability to generate a list of words beginning with the same sound (verbal fluency).

According to the scoring criteria, XXXX's scores were as follows.

TEST	**RESULT**
Reading	Above average
Spelling	Above average
Writing	Average
Rapid naming	Average
Bead threading	Average
Postural stability	Above average
Phonetic segmentation	Below average
Backwards digit span	Average
Nonsense passage reading	Average
Semantic & Verbal fluency	Above average

An 'At Risk' Quotient of 1 or greater is strong evidence that a child is 'at risk' of developing dyslexia. However, XXXX's score was well under this risk quotient. Her score was only 0.1, which suggests that the risk of her developing dyslexia is extremely low.

Having said this, and given that there is a family history of dyslexia, if you are ever concerned about XXXX's subsequent progress in school and you suspect that the problems are related to dyslexia, you can seek to have her assessed, either through the school with the National Educational Psychological Service (N.E.P.S.), or privately. Information regarding private assessment can be sought from the Dyslexia Association of Ireland (D.A.I.). Please be aware that there may be long waiting lists for such assessments regardless of whether you go through N.E.P.S. or privately. Children are not usually suitable for testing before they are 6 years old.

APPENDIX 19

Child Name: _____ Date of Birth: _____

Section 1: Doing the Programme

Please answer the following questions as accurately and honestly as possible. There is no "right" answer; we are simply interested in how practical you found these programmes.

- Did your child participate in a movement programme?

  YES [ ] *Please continue this section*

  NO [ ] *Please skip to Section 2*

- On average, how many days per week did your child do the movements?

  Please indicate 1-7---------- [ ]

- Approximately, for how many minutes each day did your child engage in the movements?

  2-4 minutes [ ]

  4-6 minutes [ ]

  6-8 minutes [ ]

  8-10 minutes [ ]

- Regarding the quality of the movements, please tick the box which you feel applies most to the way you and your child did the movements.

  Usually rushed through them----------[ ]

  Sometimes rushed them / sometimes slow and controlled ----------[ ]

  Most of the time slow and controlled way, but occasionally rushed ------[ ]

  We usually did them in a slow and controlled way----------[ ]

- Child's motivation for the exercises?

  He / she was happy to do them----------[ ]

  He / she was not always happy about it----------[ ]

  It was difficult to get him / her to do them----------[ ]

- Were there any family or personal difficulties which interfered with participation in the programme? *Please state YES or NO; you may give reasons only if you wish to do so.*

- Were there any difficulties in learning / teaching the programme to the child? If so, what?

- Was the video helpful?

- Were the storylines helpful?

- Do you have any suggestions for making the programme more appealing to the child?

- How did you fit the movements into your child's daily routine?

Please tick if you wish to avail of another programme used in this study. **YES [ ]**
NO [ ]

Section 2: Other Activities

The following questions concern other activities your child might have been involved in over the last year. Please respond to those which apply.

- Sport: If your child has been involved in any sport (e.g. swimming, gymnastics, football, dancing), please list what they were and for how long they were involved in them.

Sport (e.g. tennis)	How long? (e.g. 1 month)	How often? (e.g. once a week)
1.		
2.		
3.		
4.		
5.		

- School / Academic Help: If your child was involved in any clubs or groups which focussed on school related work (e.g. remedial classes in school, private tutoring, language classes), please list what they were and for how long they were involved in them.

Group (e.g. remedial help)	How long (e.g. school year)	How often? (e.g. once a week)
1.		
2.		
3.		
4.		

- Other Specialised Help: If you child received any other help specifically to do with any developmental problems, please give details (e.g. Speech & Language therapy, DDAT, Primary Movement, Neuro-developmental Therapy (NDT)).

Help (e.g NDT)	How long (e.g. all year)	How often? (e.g. once a week)
1.		
2.		
3.		
4.		

- Other Extra-Curricular Activities: Please give details of any other *regular* extra-curricular activities your child might have engaged in (e.g. music lessons) and for how long they were involved in them.

Activity (e.g. music)	How long (e.g. all year)	How often? (e.g. once a week)
1.		
2.		
3.		
4.		

Section 3: School Attendance

- Has your child received any schooling, be it pre-school, primary school or otherwise?

No schooling at all yet----------[ ] ***Skip to Section 4***

Number of years child has spent in pre-school (e.g. Montessori)------[ ]

Number of years child has spent in primary school----------[ ]

- In the past year, how regular has your child's attendance in school been? *Please tick box which applies.*

  Missed 0-2 days----------[ ]

  Missed 3-5 days----------[ ]

  Missed 6-20 days----------[ ]

  Missed more than 20 days----------[ ]

Section 4: Medical & Family

- Have there been any medical problems since the study started (in addition to anything mentioned in the first questionnaire)?

  _____

- Have there been any diagnoses of any of the following?

  Dyslexia [ ] **Dyspraxia** [ ] **ADHD** [ ]

  ADD [ ] **Autism** [ ] **Other** [ ] *Please specify* _____

- Any other speech/hearing problems?

  _____

- Is the child taking 'EyeQ'?_____

- Is the child taking other medication or supplement? _____

- Please comment on whether you think your child has progressed over the last year in terms of reading, balance or attention.

_____

_____

_____

_____

Are there any other comments you would like to make?

_____

_____

_____

_____

_____

_____

- - - - - - - - - - - - - - - - - -END OF QUESTIONNAIRE-THANK YOU - - - - - - - - - - - - - -

OFFICE USE

Child ID:_____

Initial Group [ ]
End Group [ ]

If changed group, how much of initial programme did they do (in weeks)? [ ]

Age child started study:_____ Finished:_____

Manual returned:

Video returned:

Cert.:

T-shirt:

Report:

Further Programme:

R-Questionnaire entered:

APPENDIX 20

Conners' Parent Rating Scale - Revised (S)

by C. Keith Conners, Ph.D.

Child's Name:_____ **Gender: M F**

Birthdate:____/____/____ **Age:**_____ **School Grade:**_____
Month Day Year

Parent's Name:_____ **Today's Date:**____/____/____
Month Day Year

Instructions: Below are a number of common problems that children have. Please rate each item according to your child's behavior in the last month. For each item, ask yourself, "How much of a problem has this been in the last month?", and circle the best answer for each one. If none, not at all, seldom, or very infrequently, you would circle 0. If very much true, or it occurs very often or frequently, you would circle 3. You would circle 1 or 2 for ratings in between. Please respond to each item.

	NOT TRUE AT ALL (Never, Seldom)	JUST A LITTLE TRUE (Occasionally)	PRETTY MUCH TRUE (Often, Quite a Bit)	VERY MUCH TRUE (Very Often, Very Frequent)
1. Inattentive, easily distracted	0	1	2	3
2. Angry and resentful	0	1	2	3
3. Difficulty doing or completing homework	0	1	2	3
4. Is always "on the go" or acts as if driven by a motor	0	1	2	3
5. Short attention span	0	1	2	3
6. Argues with adults	0	1	2	3
7. Fidgets with hands or feet or squirms in seat	0	1	2	3
8. Fails to complete assignments	0	1	2	3
9. Hard to control in malls or while grocery shopping	0	1	2	3
10. Messy or disorganized at home or school	0	1	2	3
11. Loses temper	0	1	2	3
12. Needs close supervision to get through assignments	0	1	2	3
13. Only attends if it is something he/she is very interested in	0	1	2	3
14. Runs about or climbs excessively in situations where it is inappropriate	0	1	2	3
15. Distractibility or attention span a problem	0	1	2	3
16. Irritable	0	1	2	3
17. Avoids, expresses reluctance about, or has difficulties engaging in tasks that require sustained mental effort (such as schoolwork or homework)	0	1	2	3
18. Restless in the "squirmy" sense	0	1	2	3
19. Gets distracted when given instructions to do something	0	1	2	3
20. Actively defies or refuses to comply with adults' requests	0	1	2	3
21. Has trouble concentrating in class	0	1	2	3
22. Has difficulty waiting in lines or awaiting turn in games or group situations	0	1	2	3
23. Leaves seat in classroom or in other situations in which remaining seated is expected	0	1	2	3
24. Deliberately does things that annoy other people	0	1	2	3
25. Does not follow through on instructions and fails to finish schoolwork, chores or duties in the workplace (not due to oppositional behavior or failure to understand instructions)	0	1	2	3
26. Has difficulty playing or engaging in leisure activities quietly	0	1	2	3
27. Easily frustrated in efforts	0	1	2	3

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Conners' Parent Rating Scale - Revised (S)

by C. Keith Conners, Ph.D.

Child's Name:_____ **Gender: M F**

Birthdate:____/____/____ (Month / Day / Year) **Age:**_____ **School Grade:**_____

Parent's Name:_____ **Today's Date:**____/____/____ (Month / Day / Year)

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For each item, transfer the circled number into each of the white boxes across the row. Sum each column and write the totals in the boxes labelled "TOTALS" at the bottom.

A: Oppositional	B: Cognitive Problems/ Inattention	C: Hyper-activity	D: ADHD Index				
				0	1	2	3
				0	1	2	3
				0	1	2	3

Profile for Males: Conners' Parent Rating Scale - Revised (S)

Child's Name:_____ **Gender:** M F (Circle One)

Birthdate:____/____/____ (Month / Day / Year) **Age:**_____ **School Grade:**_____

Parent's Name:_____ **Today's Date:**____/____/____ (Month / Day / Year)

	A. Oppositional					B. Cognitive Problems/ Inattention					C. Hyperactivity					D. Conners' ADHD Index				
T	1	2	3	4	5	1	2	3	4	5	1	2	3	4	5	1	2	3	4	5
90	.	.	.	.	.	17	.	.	.	.	.	18	.	.	.	.	.	.	.	.
89	.	.	18	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	36
88	.	17	.	.	16	.	.	.	.	.	.	17	15	12	7	.	.	.	.	35
87	.	.	.	18	.	16	.	.	.	.	.	.	.	.	.	.	.	.	.	.
86	18	.	17	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	34
85	.	16	.	17	15	15	.	.	.	.	.	16	14	.	.	36	.	.	.	33
84	17	.	.	.	.	.	.	.	.	.	.	.	.	11	.	35	.	.	.	32
83	.	.	16	.	.	.	.	.	.	.	.	.	.	.	.	.	36	.	36	.
82	.	15	.	16	14	14	18	.	.	.	.	15	13	.	6	34	35	36	35	31
81	16	.	.	.	.	.	.	.	.	.	18	.	.	10	.	33	34	.	34	30
80	.	.	15	.	.	.	.	.	.	18	.	14	.	.	.	32	33	35	33	29
79	.	14	.	15	13	13	17	18	.	.	.	.	12	.	.	31	.	34	.	.
78	15	.	14	.	.	.	.	.	.	17	17	.	.	.	.	.	32	33	32	28
77	.	13	.	14	.	.	16	17	18	.	.	13	.	9	.	30	31	32	31	27
76	14	.	.	.	12	12	.	.	.	16	16	.	11	.	.	29	30	31	30	26
75	.	.	13	.	.	.	15	16	17	.	.	.	.	.	5	28	29	30	29	.
74	.	12	.	13	.	.	.	.	.	15	15	12	.	.	.	.	28	.	28	25
73	13	.	.	.	11	11	14	15	16	.	.	.	10	8	.	27	.	29	.	24
72	.	.	12	.	.	.	.	.	.	14	14	11	.	.	.	26	27	28	27	23
71	.	11	.	12	.	10	.	14	15	.	.	.	.	.	.	25	26	27	26	.
70	12	.	.	.	10	.	13	.	.	.	.	.	9	7	.	24	25	26	25	22
69	.	.	11	11	.	.	.	13	14	13	13	10	.	.	4	.	24	25	24	21
68	11	10	.	.	.	9	12	.	.	.	.	.	.	.	.	23	23	24	23	.
67	.	.	10	.	9	.	.	12	13	12	12	.	8	.	.	22	.	23	22	20
66	.	.	.	10	.	.	11	.	.	.	.	9	.	6	.	21	22	.	.	19
65	10	9	.	.	.	8	.	11	12	11	11	.	.	.	.	.	21	22	21	18
64	.	.	9	9	8	.	10	.	.	.	.	8	7	.	.	20	20	21	20	.
63	.	.	.	.	.	.	.	10	11	10	.	.	.	5	3	19	19	20	19	17
62	9	8	.	.	.	7	9	.	.	.	10	.	.	.	.	18	18	19	18	16
61	.	.	8	8	7	.	.	9	10	9	.	7	6	.	.	17	.	18	17	15
60	8	.	.	.	.	.	.	.	.	.	9	.	.	.	.	.	17	17	.	.
59	.	7	7	.	.	6	8	.	9	8	.	.	5	4	.	16	16	16	16	14
58	.	.	.	7	6	.	.	8	.	.	8	6	.	.	.	15	15	.	15	13
57	7	6	.	.	.	.	7	.	8	7	.	.	.	.	2	14	14	15	14	12
56	.	.	6	6	.	5	.	7	.	.	7	5	4	3	.	.	13	14	13	.
55	.	.	.	.	5	.	6	.	7	.	.	.	.	.	.	13	.	13	12	11
54	6	5	.	.	.	4	.	6	.	6	.	.	.	.	.	12	12	12	11	10
53	.	.	5	5	.	.	5	.	6	.	6	4	3	.	.	11	11	11	.	9
52	5	.	.	.	4	.	.	5	.	5	.	.	.	2	.	10	10	10	10	.
51	.	4	.	.	.	3	.	.	5	.	5	.	.	.	1	.	9	.	9	8
50	.	.	4	4	.	.	4	4	.	4	.	3	2	.	.	9	.	9	8	7
49	4	.	.	.	3	.	.	.	4	.	4	.	.	.	.	8	8	8	7	6
48	.	3	3	3	.	2	3	3	3	3	.	2	.	1	.	7	7	7	6	.
47	3	.	.	.	.	.	.	.	.	.	.	.	1	.	.	6	6	6	5	5
46	.	.	.	.	2	.	2	2	2	2	3	.	.	.	.	.	5	5	.	4
45	.	2	2	2	.	1	.	.	.	.	.	1	.	0	0	5	4	4	4	.
44	2	.	.	.	.	.	1	1	1	1	2	.	0	.	.	4	.	3	3	3
43	.	.	.	1	1	.	.	.	.	.	.	.	.	.	.	3	3	.	2	2
42	.	1	1	.	.	0	.	0	0	0	1	0	.	.	.	.	2	2	1	1
41	1	.	.	.	.	.	0	.	.	.	.	.	.	.	.	2	1	1	0	.
40	.	0	.	0	0	.	.	.	.	.	0	.	.	.	.	1	0	0	.	0
39	0	.	0	.	.	.	.	.	.	.	.	.	.	.	.	0	.	.	.	.
38	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.

Note:
For age-groups:

Column 1: ages 3 to 5

Column 2: ages 6 to 8

Column 3: ages 9 to 11

Column 4: ages 12 to 14

Column 5: ages 15 to 17

Please see back of scoring sheet for Scale Descriptions

Please see reverse for CPRS-R Female Profile

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Profile for Females: Conners' Parent Rating Scale - Revised (S)

Child's Name:_____ **Gender: M F** (Circle One)

Birthdate:____/____/____ (Month / Day / Year) **Age:**_____ **School Grade:**_____

Parent's Name:_____ **Today's Date:**____/____/____ (Month / Day / Year)

	A. Oppositional					B. Cognitive Problems/ Inattention					C. Hyperactivity					D. Conners' ADHD Index				
T	1	2	3	4	5	1	2	3	4	5	1	2	3	4	5	1	2	3	4	5
90	.	.	17	16	17	.	.	16	.	.	.	15	.	.	.	27	33	30	.	.
89	.	16	.	.	.	12	15	.	16	.	.	.	9	.	.	.	32	.	28	29
88	.	.	.	.	.	.	.	15	.	16	.	.	.	.	.	26	.	29	27	28
87	14	.	16	15	16	.	.	.	15	.	12	14	.	.	6	.	31	28	.	.
86	.	15	.	.	.	.	14	.	.	.	.	.	.	6	.	25	30	.	26	27
85	.	.	.	.	.	11	.	14	.	15	.	.	.	.	.	.	.	27	.	.
84	13	.	15	14	15	.	.	.	14	.	.	13	8	.	.	24	29	26	25	26
83	.	14	.	.	.	.	13	.	.	14	11	.	.	.	.	.	28	.	24	25
82	.	.	.	.	.	.	.	13	.	.	.	.	.	.	.	23	.	25	.	.
81	.	.	14	13	14	10	.	.	13	.	.	12	.	.	.	22	27	24	23	24
80	12	13	.	.	.	.	12	.	.	13	.	.	.	5	5	.	26	.	.	23
79	.	.	.	.	.	.	.	12	.	.	10	.	7	.	.	21	.	23	22	.
78	.	.	13	12	13	.	.	.	12	.	.	11	.	.	.	.	25	.	.	22
77	11	12	.	.	.	9	11	.	.	12	.	.	.	.	.	20	24	22	21	.
76	.	.	12	.	12	.	.	11	.	.	9	.	.	.	.	.	23	21	20	21
75	.	.	.	11	.	.	.	.	11	.	.	10	.	.	.	19	.	.	.	20
74	.	11	.	.	.	8	10	.	.	11	.	.	6	.	.	.	22	20	19	.
73	10	.	11	.	11	.	.	10	.	.	.	.	.	4	4	18	21	19	.	19
72	.	.	.	.	.	.	.	.	10	.	8	9	.	.	.	.	.	.	18	.
71	.	10	.	10	.	.	9	.	.	10	.	.	.	.	.	17	20	18	17	18
70	9	.	10	.	10	7	.	9	9	.	.	.	.	.	.	.	19	.	.	17
69	.	.	.	.	.	.	.	.	.	9	.	8	5	.	.	16	.	17	16	.
68	.	9	.	9	.	.	8	.	.	.	7	.	.	.	.	15	18	16	.	16
67	.	.	9	.	9	.	.	8	8	.	.	.	.	.	.	.	17	.	15	.
66	8	.	.	.	.	6	.	.	.	8	.	7	.	3	3	14	.	15	14	15
65	.	.	.	8	.	.	7	.	.	.	.	.	.	.	.	.	16	14	.	14
64	.	8	8	.	8	.	.	7	7	.	6	.	4	.	.	13	15	.	13	.
63	7	.	.	.	.	5	.	.	.	7	.	6	.	.	.	.	.	13	.	13
62	.	.	.	7	.	.	6	.	.	.	.	.	.	.	.	12	14	.	12	12
61	.	7	7	.	7	.	.	6	6	.	.	.	.	.	.	.	13	12	11	.
60	.	.	.	.	.	.	.	.	.	6	5	5	3	.	.	11	.	11	.	11
59	6	.	.	6	.	4	5	5	.	.	.	.	.	2	2	.	12	.	10	.
58	.	6	6	.	6	.	.	.	5	5	.	.	.	.	.	10	11	10	.	10
57	.	.	.	.	.	.	.	.	.	.	4	.	.	.	.	.	.	9	9	9
56	5	.	.	5	.	.	4	4	4	.	.	4	.	.	.	9	10	.	8	.
55	.	5	5	.	5	3	.	.	.	4	.	.	2	.	.	8	9	8	.	8
54	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	8	.	7	.
53	.	.	.	4	.	.	3	3	3	.	3	3	.	.	.	7	.	7	.	7
52	4	4	4	.	4	.	.	.	.	3	.	.	.	1	1	.	7	6	6	6
51	.	.	.	.	.	2	.	.	.	.	.	.	.	.	.	6	6	.	5	.
50	.	.	3	3	.	.	2	2	2	.	.	2	1	.	.	.	.	5	.	5
49	3	3	.	.	3	.	.	.	.	2	2	.	.	.	.	5	5	4	4	4
48	.	.	.	.	.	1	.	.	.	.	.	.	.	.	.	.	4	.	.	.
47	.	.	2	2	.	.	1	1	1	1	.	1	.	.	.	4	.	3	3	3
46	.	2	.	.	2	.	.	.	.	.	.	.	.	0	.	.	3	.	2	.
45	2	.	.	.	.	.	.	.	.	.	1	.	0	.	0	3	2	2	.	2
44	.	.	1	.	.	0	0	0	0	0	.	0	.	.	.	.	.	1	1	1
43	.	1	.	1	1	.	.	.	.	.	.	.	.	.	.	2	1	.	.	.
42	1	.	.	.	.	.	.	.	.	.	0	.	.	.	.	1	0	0	0	0
41	.	.	0	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
40	.	0	.	0	0	.	.	.	.	.	.	.	.	.	.	0	.	.	.	.
39	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
38	0	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.

Note: For age-groups:

Column 1: ages 3 to 5

Column 2: ages 6 to 8

Column 3: ages 9 to 11

Column 4: ages 12 to 14

Column 5: ages 15 to 17

Please see back of scoring sheet for Scale Descriptions

Please see reverse for CPRS-R Male Profile

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APPENDIX 21



Early Years Project

This is to confirm that _____

took part in the ***Dyslexia Early Years Project***

Signed _____ *Date*_____

Dyslexia Research Group
Department of Psychology
Trinity College
Dublin 2.

APPENDIX 22

	Number of minutes done			
Group	*2-4*	*4-6*	*6-8*	*8-10*
Reflex	11.4%	48.6%	17.1%	22.9%
Balance	3.7%	29.6%	33.3%	33.3%
Placebo	0%	18.8%	25%	56.3%

Table g: Average (estimated) time spent per day on respective programmes, expressed as a percentage of group.

	Quality of programme			
Group	*Rushed*	*Sometimes rushed*	*Usually controlled*	*Controlled*
Reflex	11.4%	48.6%	17.1%	22.9%
Balance	3.7%	29.6%	33.3%	33.3%
Placebo	0%	18.8%	25%	56.3%

Table h: Estimated quality of programme done per day, expressed as a percentage of group.

	Engaged in sport?	
Group	*Yes*	*No*
Reflex	85.7%	14.3%
Balance	96.3%	3.7%
placebo	100%	0%

Table i: Percentages of groups engaged in sport over the year of programme participation.

	Engaged in Academic help?	
Group	*Yes*	*No*
Reflex	42.9%	57.1%
Balance	59.3%	40.7%
placebo	50%	50%

Table j: Percentages of group availing of academic help over the year of programme participation.

	Engaged in specialist help?	
Group	*Yes*	*No*
Reflex	28.6%	71.4%
Balance	33.3%	66.7%
placebo	25%	75%

Table k: Percentages of group engaged in specialist help over the year of programme participation.

	Engaged in other activities?	
Group	*Yes*	*No*
Reflex	51.4%	48.6%
Balance	22.2%	77.8%
placebo	37.5%	62.5%

Table l: Percentages of group engaged in other activities over the year of programme participation.

APPENDIX 23

Minutes

	Number of minutes done			
Dyslexia Status	*2-4 mins*	*4-6 mins*	*6-8 mins*	*8-10 mins*
At risk	12%	28%	32%	28%
Not at risk	4.1%	36.7%	24.5%	34.7%

Table a: Average (estimated) time spent per day on respective programmes, expressed as a percentage of group, where groups refer to Dyslexia status and the higher band of Dyslexia status is used (i.e., risk >= 0.9 on the DEST).

	Number of minutes done			
Dyslexia Status	*2-4 mins*	*4-6 mins*	*6-8 mins*	*8-10 mins*
At risk	9.7%	29%	32.3%	29%
Not at risk	4.7%	37.2%	23.3%	34.9%

Table b: Average (estimated) time spent per day on respective programmes, expressed as a percentage of group, where groups refer to Dyslexia status and the wider band of Dyslexia risk is used (i.e., risk >= 0.6 on the DEST).

	Number of minutes done			
ADHD Status	*2-4 mins*	*4-6 mins*	*6-8 mins*	*8-10 mins*
At risk	21.1%	26.3%	21.1%	31.6%
Not at risk	1.8%	36.4%	29.1%	32.7%

Table c: Average (estimated) time spent per day on respective programmes, expressed as a percentage of group, where groups refer to ADHD status.

Performance

	Quality of programme			
Dyslexia Status	*Usually rushed*	*Often rushed*	*Often controlled*	*Usually controlled*
At risk	8%	32%	48%	12%
Not at risk	4.1%	34.7%	40.8%	20.4%

Table d: Self-judged quality of programme performance, expressed as a percentage of group, where groups refer to Dyslexia status and the higher band of Dyslexia status is used (i.e., risk >= 0.9 on the DEST).

	Quality of programme			
Dyslexia Status	*Usually rushed*	*Often rushed*	*Often controlled*	*Usually controlled*
At risk	9.7%	29%	48.4%	12.9%
Not at risk	2.3%	37.2%	39.5%	20.9%

Table e: Self-judged quality of programme performance, expressed as a percentage of group, where groups refer to Dyslexia status and the wider band of Dyslexia risk is used (i.e., risk >= 0.6 on the DEST).

	Quality of programme			
ADHD Status	*Usually rushed*	*Often rushed*	*Often controlled*	*Usually controlled*
At risk	21.1%	47.4%	21.1%	10.5%
Not at risk	0%	29.1%	50.9%	20%

Table f: Self-judged quality of programme performance, expressed as a percentage of group, where groups refer to ADHD status.

APPENDIX 24 / 25

		120 DAYS SAMPLE						FULL SAMPLE						
		Full 120 days (n=118)	*120 days-private 5 (n=113)*	*Private 5 only group 1 (n=88)*	*120 days–ADHD (n=89)*	*ADHD only (n=29)*	*120-ADHD-private 5 (n=84)*	*Entire full sample (n-136)*	*Entire full sample-private 5 (n=131)*	*Private 5 only group 1 (n=102)*	*Entire full sample-ADHD (n=105)*	*ADHD only (n=31)*	*Entire full sample-ADHD-private 5 (n=100)*	*Entire sample with Reading variable as outcome (n=65)*
MANOVA		P < .05	P < .05	P < .05	P < .05	P < .01	P < .05	P < .01	P < .05	P < .05	P < .01	P < .05	P < .05	
ANOVA	*Dyslexia*	P < .05	P < .05	P < .05	P < .05		P < .05	P < .05	**P = .065**	**P = .086**	P < .05			P < .01 (N.P.)
	Reflex													
	Balance													
	VM													
	FM													
	Phon													
	ADHD													
POST-HOC	*Ref v Bal*	P < .05			P < .05			P < .05			**P = .058**			P < .01 (N.P.)
	Ref v Placebo													P < .05 (N.P.)
	Ref v Control													
	Bal v Placebo													
	Bal v Control	P < .05	P < .05	**P =.07**										P < .05 (N.P.)
	Placebo v Control													
Chi-squared*	"High" cut-off	P < .05	P < .05	**N.A.**	P < .05	**N.A.**	**P = .06**	**P = .07**		**N.A.**	P < .05	**N.A.**		
	"Low" cut off	**P = .06**	**P = .06**	**N.A.**	**P = .06**	**N.A.**				**N.A.**	**P = .087**	**N.A.**		

Treatment-efficacy: Summary of significant findings.* Group and Dyslexia status (i.e. "at risk" or "not at risk"). N.A. = Not Applicable. N.P. = Non Parametric (i.e. Kruskal Wallis or Mann-Whitney U tests)

		Full 120 days (n=118)	*120 days-private 5 (n=113)*	*Private 5 only group 1 (n=88)*	*120 days–ADHD (n=89)*	*ADHD only (n=29)*	*120-ADHD-private 5 (n=84)*	*Entire full sample (n-136)*	*Entire full sample-private 5 (n=131)*	*Private 5 only group 1 (n=102)*	*Entire full sample-ADHD (n=105)*	*ADHD only (n=31)*	*Entire full sample-ADHD-private 5 (n=100)*	*Entire sample with Reading variable as outcome (n=65)*
ANCOVA	*Dyslexia*	P < .05	P < .05	P < .05	P < .05		P < .05	P < .05	**P < .05**	**P < .05**	P < .05		**P = .06**	P < .01
	Reflex													
	Balance													
	VM													
	FM													
	Phon													
	ADHD													
POST-HOC (Sidak adjustment)	*Ref v Bal*	P < .05	**P = .06**	**P = .07**	P < .05		**P < .05**	P < .05	**P < .05**		**P < .05**			**P < .05**
	Ref v Placebo													
	Ref v Control													
	Bal v Placebo													
	Bal v Control	P < .05	P < .05											P < .05 (N.P.)
	Placebo v Control													

Treatment-efficacy examined using ANCOVA rather than ANOVA where age is the controlled variable. Changes from the MANOVA analyses are printed in bold.

	MULTIPLE REGRESSION								LOGISTIC REGRESSION							
	Model steps		*Predictor variables*						*Model steps*		*Predictor variables*					
	Step 1	Step 2	*Ref*	*Schil*	*Bal*	*VM*	*FM*	*Phon*	Step 1	Step 2	*Ref*	*Schil*	*Bal*	*VM*	*FM*	*Phon*
1ST step variable / Outcome variable																
Full Reflex / DEST & DST (n=98)				**N.A.**								**N.A.**				
Schilder / DEST & DST (n=98)			**N.A.**							p<.001	**N.A.**		p<.05			
Full Reflex / Reading only (n=54)		p<.01	p<.05	**N.A.**			p<.05	p<.01		p<.001	p<.01	**N.A.**	p<.05		p<.01	p<.01
Schilder / Reading only (n=54)		p<.05	**N.A.**					p<.01	p<.05	p<.001	**N.A.**	p<.05		p<.05		p<.01
Full Reflex / Mild Dyslexia (n=98)	**N.A.**	**N.A.**	**N.A.**	**N.A.**	**N.A.**	**N.A.**	**N.A.**	**N.A.**		p<.001		**N.A.**		p<.05	p<.05	p<.05
Schilder / Mild Dyslexia (n=98)	**N.A.**	**N.A.**	**N.A.**	**N.A.**	**N.A.**	**N.A.**	**N.A.**	**N.A.**		p<.001	**N.A.**			p<.05		p<.001
All entered / ADHD	**N.A.**	**N.A.**	**N.A.**	**N.A.**	**N.A.**	**N.A.**	**N.A.**	**N.A.**		**N.A.**						
Full Reflex / Very poor reading										p<.05						p<.01

Predictive-efficacy: Summary of significant predictor variables for all outcomes.
N.A. = Not Applicable

APPENDIX 26

	GROUPS											
	Reflex			Balance			Placebo			Control		
TIME	**1**	**2**	**3**	**1**	**2**	**3**	**1**	**2**	**3**	**1**	**2**	**3**
Reflex	1.84 (.35)	1.77 (.28)	1.81 (.3)	1.92 (.4)	1.99 (.35)	1.9 (.29)	1.79 (.42)	1.85 (.36)	1.83 (.3)	1.86 (.45)	1.88 (.38)	1.89 (.38)
Balance	2.47 (.67)	2.34 (.7)	2.26 (.61)	2.8 (.7)	2.6 (.62)	2.35 (.5)	2.8 (.73)	2.5 (.67)	2.37 (.7)	2.5 (.74)	2.4 (.74)	2.21 (.69)
Visual-motor	9.54 (4.08)	8.8 (3.97)	8.51 (4)	8.5 (3.56)	10 (3.6)	9.7 (3.5)	8.53 (3.19)	8.8 (3.4)	8.95 (3.8)	9.03 (3.56)	8.37 (4.22)	8.57 (4.01)
Fine-motor	15.33 (4.37)	12.77 (3.25)	12.33 (3.12)	16.47 (5.4)	15.45 (4.45)	15.8 (4.56)	15.16 (5.16)	14 (4.71)	13.7 (4.3)	16.3 (5.6)	14.6 (4.8)	14 (4.7)
Phonological	60.38 (15.44)	64.77 (17.17)	70.03 (13.43)	50.45 (15.65)	58 (15.19)	64.87 (12.88)	56.32 (16.01)	63.53 (17.14)	67.6 (14.95)	55.88 (16.34)	61.8 (17.93)	68.77 (11.29)
DEST / DST			.59 (.39)			.89 (.55)			.82 (.66)			.65 (.45)
ADHD			54.72 (11.58)			54.55 (10.32)			57.53 (10.68)			55.6 (11.45)

Mean (S.D.)

Means of the pre and post tests for Reflex, Balance, Visual-motor, Fine-motor and Phonological tests, plus the means of the Dyslexia and ADHD screening tests administered at the end of the study.