DEFICITS IN MOTOR CONTROL: CAUSES OR CORRELATES OF READING DISABILITY?

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June 2006

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Doctor of Philosophy
2006

Reading disability or developmental dyslexia is a specific disorder of reading that is associated with inconsistent evidence of deficits in motor control. Balance measures have revealed the most convincing evidence and several dyslexia screening batteries include tests of postural stability. A prominent causal theory of dyslexia links reading and balance difficulties through mild impairment of the cerebellum. Cerebellar abnormalities are associated with other developmental disorders including Attention Deficit Hyperactivity Disorder (ADHD), which co-occurs with both dyslexia and motor deficits (Chapter 1). A meta-analysis of dyslexia and balance studies (Chapter 3) yielded a robust (d = .64) combined between-groups effect size, however heterogeneity was revealed across studies. The strongest predictor of postural stability was whether samples had been screened for ADHD. Inconsistencies in sampling, procedure and measurement across studies were also revealed. The thesis aimed to introduce an objective, quantitative approach to the study of balance in dyslexia using theory and techniques from postural and balance research (Chapters 2 & 4). Unselected samples of adults and children from the dyslexia and normal reading populations were assessed on psychometric, literacy and behavioural measures. Postural stability was measured using digital optical motion capture (Qualisys Systems), obtained during a perturbation of balance caused by the release of a suspended weight equivalent to 5% body mass. The paradigm replicated the hitherto most frequently used balance measure with adults (Chapters 5 & 6) and children (Chapters 7 & 8). The findings suggest that balance difficulties in the dyslexia and general populations are correlates of reading difficulties but are related through hyperactivity or combined inattention and hyperactivity ratings. Tests of postural stability are therefore more likely to be useful predictors of risk of more general developmental disorder than specifically of reading failure. Postural control is therefore placed within the wider context of co-occurring developmental disorders (Chapter 9).

KEYWORDS: DYSLEXIA; ADHD; BALANCE; POSTURAL STABILITY; COMORBIDITY.

To my family

Yvonne

Renzo, Liz and Mario

for their love and support.

Acknowledgements

First and foremost I would like to express my sincere and eternal thanks to my supervisor Joel Talcott, for always expecting more than my best.

Thanks also to:

Ian Richards for advice on psychometric and literacy measures.
Jon Wood for technical assistance.
Alan Wing, Kristen Hollands, Huiya Chen and the Birmingham University Posture and Balance Group for advice on reading and analysis.
Alison Fisher for mentoring and advice on formatting.
The parents, teachers, children, undergraduates and colleagues who participated.

And finally, thanks to:

Peter Reddy and the Aston University Psychology Department for re-awakening my love of learning.

Steffi, Nia, Jenny, Avgis and the Neurosciences postgraduate and post-doctoral community for empathy, fun and support throughout.

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1 Deficits in motor control and the comorbidity of developmental disorders

1.1 Developmental dyslexia: an overview

Developmental dyslexia, hereafter dyslexia, is a specific learning disorder characterised by inconsistent achievement between literacy skills and other cognitive abilities. Also known as specific reading disability (Shaywitz & Shaywitz, 2003), it is typically diagnosed when measured reading ability is significantly below the level of ability predicted for a given age or level of general intellectual ability. Although it is considered to be of constitutional origin, dyslexia occurs in the absence of obvious physiological or neurological impairment and when motivation to read and reading tuition appear to be adequate (Habib, 2000; Snowling, 2000; Stein, 2001). It is a culture-specific disorder and is prominent in the developed world due to our contemporary expectation of global literacy achievement (Pennington, 2003). Typically, more males than females are affected (2:3 to 4:5), although this gender inequality has also been attributed to selection bias (Habib, 2000; Shaywitz & Shaywitz, 2003). Prevalence of RD is estimated at 5-17% of the population (American Psychiatric Association, 2000; Habib, 2000; Shaywitz & Shaywitz, 2003). Dyslexia appears frequently in families, suggesting a strong, heritable component with loci on chromosomes 2, 3, 6, 15 and 18 implicated over several genetic linkage studies (e.g., Cardon et al., 1994; 1995; Fisher & Smith, 2001; Pennington, 2003; Willcutt & Pennington, 2000; Willcutt et al., 2005). According to Shaywitz and Shaywitz (2003) up to 50% of children of parents with dyslexia, 50% of siblings of children with dyslexia and 50% of parents of children with dyslexia may demonstrate symptoms of the disorder. While dyslexia is considered to be a dysfunction of the neural systems involved in the acquisition of reading skills, its precise neurological origin remains elusive in spite of abundant research evidence from several areas of neuroscience and a number of plausible theoretical frameworks.

Traditionally, a formal diagnosis of dyslexia was given when reading ability was measured to be at least 18 months behind the level expected for a child's chronological age. This 'cut-off' method has more recently, at least in the UK, been replaced by a regression method, whereby there must be a significant discrepancy (1.5 standard deviations) between the level of reading ability and the level of ability

predicted by a child's Full-Scale intelligence quotient (FSIQ). This method of diagnosis is subject to criticism however, particularly in the United States (US), where research and educational bodies argue that non-discrepant or 'garden variety' poor readers (i.e., with IQ in the below-average to average range) demonstrate similar phonological deficits to discrepant 'dyslexic' poor readers (i.e., those with IQ within the average to exceptionally high range) (e.g., Vellutino et al. 2004). This argument has implications for both research and remediation. Although behavioural outcomes may be similar for both discrepant and non-discrepant groups, their underlying causes may well be different. Psychometric intelligence, for example, has been shown to account for a substantial percentage of the shared variance between reading and visual and auditory processing (Hulslander et al., 2004). Inclusion of 'garden variety' readers in experimental designs that aim to address research questions about dyslexia may therefore obscure research findings as dyslexia is a specific rather than a general learning difficulty. It may be difficult to discriminate the unique variance of the variable under investigation from that attributable to IQ. Furthermore, the choice of adopting either exclusionary or inclusionary criteria for diagnosis of dyslexia impacts directly upon meeting the child's individual needs. Children of intellectual ability within the high average, high or exceptionally high range, as is often the case in children diagnosed with dyslexia, are more likely to be frustrated by their learning difficulties than less gifted children and consequently they may be even more vulnerable to developing the negative psychological characteristics associated with dyslexia.

It is widely acknowledged that dyslexia arises from cognitive deficits in the acquisition and usage of reading component skills, primarily either phonological or orthographic or both (Bradley & Bryant, 1978; Olson et al., 1989; Snowling, 2001; Stein, 2001). However, dyslexia is also associated with a broad spectrum of symptoms, both proximally and distally related to language and literacy skills. Proximal deficits (directly associated with reading processes) include: the acquisition of oral language (Bishop & Adams, 1990), speech articulation (Fawcett & Nicolson, 2002), visual processing (Stein & Walsh, 1997), handwriting (dysgraphia), spelling, mathematical skills (dyscalculia) (Miles, 1993), reading of music (dysmusia) (Gordon, 2000), rapid processing of non-linguistic stimuli (Denckla & Rudel, 1976)

and timing (dyschronia) (Llinas, 1993; Wolff, Michel, Ovrut & Drake, 1990). Distal deficits (not directly associated with reading processes) include: lateral confusion and organisational skills (Stein, 2001), gross and fine motor incoordination (dyscoordia), inaccuracy of movement (dysmetria) and postural instability (Nicolson & Fawcett, 1990).

Behavioural measures of gross and fine motor skills, such as postural stability and bead threading, are included in several dyslexia screening batteries (for example, the Dyslexia Screening Test Junior [DST-J] [Fawcett & Nicolson, 2004a] and the Preschool Early Screening Test [PREST] [Fawcett, Nicolson & Lee, 2001]) as non-linguistic predictors of reading difficulties. Research has shown that success of remediation techniques is strongly related to early identification of the potential for literacy failure and prompt onset of support (Bradley, 1988). Early assessment of motor skills, therefore, may provide a useful method for prediction of developmental disorder of literacy skills. Such indicators of being at risk of dyslexia would facilitate early intervention and subsequent prevention of the negative social and self-esteem factors frequently associated with dyslexia in adolescence (e.g., Casey, Levey, Brown & Brooks-Gunn 1992; Kirk & Reid, 2001; Maughan, Grey & Rutter, 1985).

However, the evidence for balance and motor skills deficits in the dyslexia population is inconclusive. For example, some studies have reported a high incidence of balance deficits in both children and adults with dyslexia (e.g., Fawcett & Nicolson, 1999; Nicolson & Fawcett, 1997), others have found little evidence for this (e.g., Ramus, Pidgeon & Frith, 2003; Ramus et al. 2003). Furthermore, the theoretical aetiological frameworks for dyslexia have yet to provide substantiated accounts of the link between reading difficulties and deficits in motor control. The co-occurrence of Attention Deficit Hyperactivity Disorder (ADHD) with dyslexia is substantial (15% to 40%) and approximately 50% of ADHD cases also present symptoms characteristic of Developmental Coordination Disorder (DCD) (Barkley 1990; Diamond, 2000; Gillberg, 2003; Hartsough & Lambert, 1985; Kadesjö & Gillberg, 2001; Kaplan, Dewey, Crawford & Wilson, 1998; Piek, Pitcher & Hay, 1999; Willcutt et al., 2005). The link between reading and motor skills therefore,

may not be as straight forward as previous dyslexia research has claimed. Defining the nature of this link may be central to refining not only the broader phenotype of dyslexia but also that of co-existing developmental disorders including ADHD and DCD. Children with symptoms of more than one developmental disorder are likely to need support in several areas of home and school life. Differential diagnoses of developmental disorder are therefore essential for the onset of disorder specific interventions and support.

The focal question of the thesis is whether motor control deficits are related to reading difficulties by means of a direct causal mechanism, or whether they are a correlate by means of association with other developmental disorders. The first aim is to quantify and evaluate the evidence for motor skills deficits in dyslexia to date. The second aim is to improve upon previous methodology to obtain new data. The third aim is to inform theory and practice not only of the efficacy of assessing motor skills to identify specific reading difficulties but also of the potential implications of the findings for reading interventions based upon motor skills training. Before examining the evidence for motor control deficits in dyslexia, however, the causal frameworks for reading difficulties in dyslexia are summarised and their accounts of the nature of the relationship between reading and motor skills are examined. The evidence for the co-occurrence of dyslexia with ADHD and DCD is discussed within the context of contemporary frameworks for the study of developmental disorders. Finally, the most appropriate motor skill is selected for study, from the wide range of skills that have been assessed within the dyslexia literature.

1.2 Causal theories of deficits in reading and motor control in dyslexia

Frith (1997) categorised the three most prominent theoretical frameworks of developmental dyslexia: the phonological deficit hypothesis, the cerebellar deficit hypothesis and the rapid temporal processing deficit hypothesis, within a model with three levels of explanation. This framework is claimed to conceptualise the range of skills and deficits encountered in developmental disorders including dyslexia (Frith,

1997; 2001). The three levels of description are: biological: including genetic influences and neuroanatomical irregularities; cognitive: neurofunctional anomalies underlying orthographic and phonological reading component skills, executive function and memory; and behavioural: including reading, spelling, writing and motor skills. Frith's model is shown in Figure 1:1. Each level is subject to environmental influence. The model is currently the most influential framework for the study of developmental disorders, at least within the UK. Each of the major causal accounts of dyslexia is presented below, with reference to their particular explanation of co-existing motor deficits described within the Frith framework. It quickly becomes apparent however, that this framework does not adequately demonstrate how each causal theory explains the interrelationship between co-occurring symptoms such as reading and motor control difficulties. Evidence for the causal theories described below is drawn not only from the respective authors but also from recent literature reviews including that by Habib (2000), who presented a comprehensive review of the range of evidence for the neurological basis of dyslexia obtained from a variety of neuroscientific techniques.

Figure 1:1 Three levels framework for understanding developmental disorders (taken from Frith, 2001).

1.2.1 The phonological deficit hypothesis

The phonological deficit hypothesis predicts that disordered processes within the cognitive level of Frith's model underlie reading difficulties at the behavioural level (see Figure 1:2). Stanovich (1988) first proposed that the cause of developmental dyslexia is a core phonological deficit. Reading depends upon the ability of the novice reader to segment spoken word sounds into syllables or their single phoneme components, then to learn, recognise and manipulate these sounds in their abstract

form in order to map them to their corresponding written form or grapheme. This phase of learning to read, termed logographic, is a requirement for the competent acquisition of the alphabetic principle and the subsequent transition of the reader into the alphabetic stage of reading. This leads into the final orthographic stage, whereby words can be recognised by their orthographic form and read fluently (Frith, 1985). Phonological awareness is a prerequisite for reading and this ability is typically impaired in discrepant and non-discrepant poor readers (e.g., Bradley & Bryant, 1978; Snowling, 1981, 2000; Stanovich, 1988; Vellutino et al., 2004). This inability to segment words into component units persists even after a prolonged period of learning to read and write (Bradley & Bryant, 1983). Phonological representations are the means by which the sound composites of speech and written language are neurally encoded. This hypothesis proposes that phonological representations are weakly specified in dyslexia and that this deficit underlies not only difficulties in phonological learning and the acquisition of the alphabetic principle, but also all other deficits associated with the disorder (Snowling & Hulme, 1994). These include: verbal short term memory, (e.g., Hulme, 1981; Snowling, Nation, Moxham, Gallagher & Frith, 1997), verbal naming deficits (Denckla & Rudel, 1976), Rapid Automatised Naming (RAN) (Wolf, 1986), verbal repetition (Snowling, 1981), non-word repetition (Gathercole & Baddeley, 1989), subtle speech perception (Brady, 1997) and spoken word identification (Metsala, 1997) (for review see Snowling, 2000).

Figure 1:2 The phonological deficit hypothesis (taken from Frith, 1997).

A cognitive deficit at the level of phonological representations in dyslexia has been supported by means of a large body of neuroanatomical and functional evidence of

perisylvian region abnormality, which is encapsulated in Frith's biological level of explanation (for review see Habib, 2000). This includes evidence of increased activation of Broca's area during a single letter rhyme and a nonword rhyme task, which was interpreted as evidence of more effortful phonological processing in dyslexia (Shaywitz et al., 1998). Increased activity of this area is also suggestive of motor-articulation difficulties in dyslexia (Heilman, Voeller & Alexander, 1996). Ivry and Justus (2001) also draw attention to the close interrelationship between phonology and articulation from a motor perspective in the context of the motor theory of speech perception (Liberman & Mattingly, 1985). This is discussed further in section 1.2.2. Apart from Heilman's identification of oromotor difficulties in speech articulation in dyslexia, proponents of this hypothesis maintain that motor deficits do not arise in cases of 'pure' dyslexia and only occur in the presence of other developmental disorders (e.g., Ramus, 2003). Whilst Frith's model illustrates the biological, cognitive and behavioural characteristics associated with the phonological deficit hypothesis of dyslexia, it does not provide an adequate explanation for the links between behavioural symptoms for example, how poor reading is associated with poor memory. Neither does it account for the range of non-linguistic difficulties that are associated with dyslexia.

1.2.2 The cerebellar deficit hypothesis

Nicolson and Fawcett (1999) presented a review of over ten years of research from the Sheffield Research Programme, which culminated in the proposal of the cerebellar deficit hypothesis of dyslexia. Initial studies investigated dyslexia within a skill learning framework, leading to the prediction of a universal deficit in the automatisation of all skills including gross and fine motor ability and those that impact directly upon reading. Achieving automaticity in the mapping of phonemes to graphemes and the blending of reading elements is an essential requirement for the attainment of reading fluency (Frith, 1985). The "Automatisation Deficit Hypothesis" (ADH) (Nicolson & Fawcett, 1990) predicts difficulties during the initial stages of the acquisition of any learned skill. Nicolson and Fawcett (1990) tested this hypothesis on children with dyslexia and controls across a variety of fine and gross motor skills. The most notable results were found on measures of balance, particularly when subjects were required to complete a task secondary to the initial

balance task. The secondary task involved counting or a choice reaction task; either would require a degree of higher order cognitive function. Whereas most control subjects improved on the secondary tasks, presumably through practice effects (Nicolson & Fawcett, 1999), 22/23 children with dyslexia showed balance difficulties. The authors interpreted these findings as reflecting evidence of an automatisation deficit that is revealed when a secondary task imposes load on the postural control system causing the "conscious compensation" process, a strategy that they suggest is typically employed in dyslexia to achieve normal performance, to break down (Nicolson & Fawcett, 1990, p. 3).

The next phase of this research programme examined the performance of three groups of children with dyslexia aged 8 years, 12 years and 16 years on measures of phonological ability, working memory, information processing speed and primitive motor skills. Performance was compared with that of chronological age and reading age control children. This second level of comparison was undertaken to establish whether between-groups differences could be explained by simple developmental delay (by comparison with a chronological age matched control group) or by developmental deviance (by comparison with younger children at a similar level of reading ability). Impairment in comparison with the latter would indicate a functional deviance rather than developmental lag (e.g., Bryant & Goswami, 1986). The authors concluded that children with dyslexia presented a general automatisation deficit across all primitive skills, that was not observed in their reading level peer group. This suggested that the difficulties were associated with a general performance deviance rather than a delay in normal development. This was most likely due to a fundamental problem underpinning all skill learning, including acquisition and execution stages, which lead the authors to speculate about the possibility of cerebellar impairment in dyslexia (Nicolson & Fawcett 1995; 1999).

The third phase of this programme examined evidence for a neurological deficit in dyslexia using clinical measures of cerebellar function (Fawcett, Nicolson & Dean, 1996; Fawcett & Nicolson, 1999). A battery of 14 measures was used (described in Dow & Morruzzi, 1958) and these can be partitioned into three types of test: 1)

posture and muscle tone assessment in quiet standing or in response to internally or externally generated provocation of balance; 2) tests of hypotonia (reduced muscle resistance to stretch) in the upper limbs in response to internally or externally motivated movement; 3) tests of the execution of complex voluntary movement. The children with dyslexia achieved a significantly lower level of performance than their chronological age peers on all 14 measures and on 11 measures in comparison with their reading level peers. The authors concluded that this profile of performance corresponded with that predicted by an impairment of cerebellar function (Fawcett, Nicolson & Dean, 1996). They then proposed a causal model relating cerebellar impairment to phonological deficits and ultimately reading difficulties, albeit indirectly, through the ontogenetic course of development (see Figure 1:3).

Figure 1:3 Ontogenetic causal model of developmental dyslexia from birth to 8 years (taken from Fawcett and Nicolson, 2004b)

The main causal influence of dyslexia, within the ontogenetic model, is weak implicit learning as a direct consequence of cerebellar impairment (Nicolson, Fawcett & Dean, 2001). The model directly relates cerebellar abnormality with motor impairment and consequently writing skills. It also directly relates cerebellar impairment with constrained articulatory skill. This constraint has two indirect effects: 1) more conscious resources are required for articulation therefore fewer remain for processing sensory feedback from articulation; 2) speed of articulation is reduced. These indirect effects lead to difficulties not only in language acquisition and dexterity but also lowered sensitivity to the temporal and spatial structure of

language. Nicolson, Fawcett and Dean (2001) claim that in dyslexia, direct and indirect effects of cerebellar abnormality on articulation can explain the core phonological deficit. They also claim that the cerebellar deficit hypothesis effectively subsumes all three causal theories of dyslexia. Nicolson and Fawcett (1999) cite evidence (Leiner, Leiner & Dow, 1989) of neural connections between the cerebellum and Broca's area to substantiate this claim. They also draw upon evidence from Passingham (1975) to argue that articulation is the main contributor to the structural development of the neocerebellum in *homo sapiens*.

Although it was assumed previously that the role of the cerebellum was limited primarily to the coordination and control of motor behaviour (Holmes, 1939), contemporary research has highlighted the contributing role of this structure to various non-motor functions including: stimulus sequencing (Ivry & Spencer, 2004), skill automatisation (Jenkins, Brooks, Nixon, Frackowiak, & Passingham, 1994; Lang & Bastian, 2002), speech perception (Mathiak, Hertrich, Grodd & Ackermann, 2002), receptive language and language dexterity (Justus, 2004; Fiez & Raichle, 1997). Deficits in these abilities are associated with reading impairments, so it is plausible that a neurological deficit of cerebellar origin could account for many of the wide range of symptoms associated with dyslexia. Attention has therefore returned to the motor theory of speech perception linking phonological representation to motor articulation (Liberman & Mattingly, 1985; Ivry & Justus, 2001). Furthermore, the cerebellum helps to control eye movements during reading. It has been shown to regulate eye movement responses to visual motion signals in maintaining fixation and adjusting eye movements during reading saccades (Stein, 2001). Right side cerebellar lesions in children have also been associated with language and literacy problems (Scott et al, 2001) although speech and literacy deficits are infrequently observed in acquired cerebellar lesions in adults (Ivry & Justus, 2001). Direct evidence for cerebellar abnormalities in dyslexia has been demonstrated in several neuroimaging studies for measures of brain biochemistry (Rae et al., 1998), neural morphology (Rae et al., 2002) and functional activity related to motor learning (Nicolson et al., 1999). Cerebellar contribution to motor control is further discussed in Chapter 2.

Deficits in motor control: causes or correlates of reading disability?

Although the cerebellar deficit hypothesis provides a parsimonious account of the reading and motor control deficits encountered in the broad phenotype of dyslexia, it is by no means accepted in all neurophysiological or neuropsychological areas of dyslexia research. For example, Zeffiro and Eden (2001) accept that development of cerebellar function may be impaired in dyslexia. However, they suggest that this may be a consequence of neuropathology elsewhere within the cerebellar-cortical neural networks that give rise to a simulation of cerebellar disease. They cite evidence of circuitry between the cerebellum, the thalamus and the perisylvian neocortical regions associated with phonological dysfunction and the cortical-pontine projections to the cerebellum via the pontine tegmentum (e.g., Eden & Zeffiro, 1998). They also speculate that impaired sensory information would also impair sensorimotor integration and cerebellar function. This prediction is supported by evidence from a structural MRI study that identified abnormalities within the cerebellar- frontal cortical circuitry that were associated with the double-deficit of phonological and temporal processing subtype of dyslexia (Eckert et al., 2003) (see section 1.2.3). Ivry and Justus (2001) further suggest that cerebellar impairment may be only one of numerous neural anomalies that underpin the range of behavioural deficits observed in dyslexia. Moreover, Bishop (2002) argues that cerebellar impairment may equally arise from avoidance of the tasks that typically aid cerebellar development. For instance, a child who frequently experiences failure in ball skills during team games is more likely to avoid further failure by selecting a different activity in which she or he may perform well. Cerebellar impairment may therefore arise as a consequence rather than a cause of motor difficulty because typical developmental improvement through practice is avoided.

The cerebellar deficit hypothesis appears to provide both cognitive (automaticity) and biological (cerebellar dysfunction) accounts of the multiple behavioural manifestations associated with the broader phenotype of dyslexia. Conceptualisation of the cerebellar deficit hypothesis within Frith's (2001) framework however, does not demonstrate how symptoms of cerebellar dysfunction interrelate, unless they are viewed as discrete symptoms of comorbid disorders with a shared biological cause as demonstrated in Figure 1:4.

Figure 1:4 The cerebellar deficit hypothesis (taken from Frith, 1997)

1.2.3 The temporal processing sensory deficit hypothesis

All events are temporally defined and multimodal sensory information is distributed over time. Perceptual and cognitive meaning is derived from the correct interpretation of temporal structure via the sensory modalities in all behaviour, including reading. Temporal structure is the organisation of a series of elements which can be tones, phonemes, dots, and printed words, changes in joint angle position or muscle tension and so on. Two basic examples of temporal structure are music and language. For language, written and oral communications convey meaning through rhythmic time structure. Understanding of meaning depends not only upon symbol (letter and word) recognition but also upon decoding mechanisms for temporal structure. One important means of decoding structures of rhythmic time is by relative timing, where each element in the temporal sequence is determined relative to all of the other elements along the time continuum, whether or not they are spatially adjacent (Lewkowicz, 2000). This is the basis of sequencing, another time-based function which is frequently impaired in developmental dyslexia (for review see Hari and Renvall, 2001).

According to Fraisse (1982), from an early stage of development children are predisposed to see the temporal world in terms of chunks. Fraisse suggested that

adults have a propensity for this even when sequences are isochronous. In dyslexia this is often a problem area that is demonstrated by difficulty in remembering telephone numbers (British Dyslexia Association, 2005). Individuals with dyslexia also demonstrate difficulty with developing temporal organisational skills (Miles, 1993), difficulties in time estimation (Nicolson, Fawcett & Dean, 1995), rhythm tapping (Wolff, Michel, Ovrut and Drake, 1990), temporal order judgement (Bakker and Schroots, 1981) and detecting complex timing patterns (Kujala et al., 2000). Dyslexia is also associated with slower naming speed for simple symbols, typically measured by tasks such as the Rapid Automatised Naming (RAN) task (Denckla & Rudel, 1976) or the Phonological Assessment Battery (PhAB) Naming Speed Pictures test (Frederickson, Frith & Reason, 1997). Wolf and Bowers (1999) proposed that there are three subtypes of dyslexia: a phonological deficit, a temporal processing deficit and a combination involving deficits in both phonological and temporal processing.

Impairment of the neurophysiological systems that underpin the gathering and processing of information about egocentric and exocentric events is very likely to impinge upon all temporal aspects of cognitive and behavioural function. In reading, this may lead to temporal blurring and confusion during either visual word and grapheme recognition or auditory phoneme distinction, or both. This is the way the rapid temporal processing deficit hypothesis accounts for visual-orthographic and auditory-phonological reading difficulties and, more speculatively, motor control deficits in dyslexia. In recent years, the biological theoretical basis of the rapid temporal processing deficit hypothesis within the UK has evolved predominantly from the Oxford University Laboratory of Physiology. The primary causal framework provided by this laboratory, the magnocellular deficit hypothesis of dyslexia (Stein & Walsh, 1997; Stein & Talcott, 1999) and reviewed by Stein (2001) provides a neurophysiological account of the visual deficits observed in cases of dyslexia. It also offers an explanation of the observed difficulties in the visual guidance of movement in dyslexia.

Deficits in motor control: causes or correlates of reading disability?

Stein (2001) summarised the human visual processing system that functions by means of two distinct pathways: the dorsal stream and the ventral stream, which are specialised for the detection of visual motion and visual form respectively (Ungerleider & Mishkin, 1982). The dorsal stream is responsible for the visual control of eye and limb movements and is dominated by sensory input from the magnocellular system, which is essential for timing visual events and tracking moving objects in the visual scene (Milner & Goodale, 1995). Visual motion sensitivity has been found to correlate strongly with orthographic skills for typical and atypical readers among children (Talcott et al., 2000a). Furthermore, readers with dyslexia have also been shown to demonstrate reduced sensitivity to sound amplitude and frequency changes and require significantly larger changes within a speech signal to detect transients (Stein & McAnally, 1996; Witton et al., 1997, 1998; Talcott et al., 1999, Talcott, Hansen, Elikem & Stein, 2000b). Lower frequencies are used for phoneme detection and FM sensitivity has been shown to predict phonological ability, as measured by assessment of non-word reading ability, in both adult and child samples (Witton et al., 1998; Talcott et al., 1999, 2000a). The rapid temporal processing hypothesis, therefore, provides a plausible account for both orthographic and phonological reading deficits in dyslexia. It also appears to provide an explanation of impaired visual guidance of movement essential for achieving accuracy in gross and fine motor skills, such as in catching a ball or handwriting, that have been reported as areas of weakness in dyslexia (e.g., Haslum, 1989; Miles, 1993).

Stein and Walsh (1997) proposed that magnocellular-type neurones might exhibit reduced sensitivity across modalities. Stein (2001) extends this hypothesis to the motor system and in addition, emphasises the close interrelationship between magnocellular and cerebellar structure and function, effectively subsuming the cerebellar deficit hypothesis. The temporal processing deficit hypothesis is another persuasive and parsimonious account, although it is speculative in its attempts to explain the more distal deficits associated with dyslexia. Moreover, because the cerebellum contributes to the spatial accuracy and temporal coordination of movement it is likely to be difficult to conclusively separate the role of the cerebellum from the effects of sensory processing in the assessment of motor

function in dyslexia by means of behavioural studies. Interestingly, Habib (2000) separates the visual magnocellular hypothesis from his review of the temporal processing deficit hypothesis opting instead to include the cerebellar deficit hypothesis. In Stein's view the two frameworks are compatible because the cerebellum is the head ganglion of the visual magnocellular system (Stein, 2001). This also demonstrates how, more recently, the Stein laboratory has extended its standpoint beyond the view of dyslexia as a visual deficit to one of a pan-modal temporal processing deficit. Once again, however, although the range of biological, cognitive and behavioural deficits predicted by this hypothesis can be placed within the Frith (2001) framework shown in Figure 1:5, it is unclear how such co-occurring symptoms interrelate within each level. For example, are the abnormalities of the language system and the magnocellular system associated by the same or different single or multiple genes? Whilst this model adequately demonstrates the phonological deficit hypothesis of dyslexia, it is not as effective in conceptualising the alternative theories.

Figure 1:5 The temporal processing sensory deficit hypothesis (taken from Frith, 1997).

1.3 Linking deficits in motor control to reading difficulties

While it is widely accepted that at least some children with dyslexia demonstrate some degree of motor control difficulty, as evidenced by the frequent occurrence of delay in achieving the motor milestones of crawling, walking, running, hopping and

skipping (Denckla, 1985; Gessel, 1946; Haslum, 1989; Miles, 1993) motor deficits are by no means ubiquitous and research findings are inconsistent (e.g., Fawcett & Nicolson, 1997; Ramus et al., 2003). The British Dyslexia Association (BDA) website lists several motor deficit indications of dyslexia including persistent difficulties in: dressing competently (including tying shoe laces, ties or ribbons), catching, throwing or kicking a ball, hopping and skipping, as well as a predisposition for bumping into objects, tripping and falling. The International Dyslexia Association (IDA) website, however, cites difficulty with motor skills (planning and coordination of body movements and in coordinating the movement of facial muscles to articulate speech) as one dimension of a number of other associated learning disorders that include dysgraphia, dyscalculia, ADD/ADHD and organisational difficulties. The role of motor deficits as indicators of reading difficulties is therefore viewed differently by each international organisation and this reflects the diversity of the different theoretical perspectives of dyslexia research.

Ramus (2003) among others (e.g., Snowling, 2001) draws a distinction between 'pure' phonological dyslexia and reading difficulties associated with sensory motor dysfunction. The central issue in linking motor control deficits to reading difficulty appears, therefore, to be one of theoretical viewpoint: whether dyslexia should be defined as a specific disorder of reading with a unique phonological aetiology, independent of associated cognitive and behavioural difficulties, including motor deficits. In my view, this issue effectively separates the phonological deficit hypothesis of dyslexia, from the cerebellar deficit and temporal processing deficit hypotheses, and may well account for the seemingly inconclusive evidence for motor deficits in dyslexia. The theoretical perspective of the research group would no doubt affect their inclusionary or exclusionary sampling criteria for a given study. The definition of dyslexia, therefore, has implications for both diagnosis and research.

1.3.1 The comorbidity of developmental disorders

Denckla, Rudel, Chapman and Krieger (1985) called for all future studies of dyslexia and motor control to provide a profile of attention and motor skill scores for all participants. Whether dyslexia should be regarded as a central disorder of reading,

independent of associated characteristics however, is not only an issue fundamental to dyslexia research but also one currently central to the study of developmental disorders in general (e.g., Frith, 2001; Gilger & Kaplan, 2001; Kadesjö & Gillberg, 2001). This issue has consequences not only for the theoretical framework within which dyslexia and other developmental disorders should be studied but also for diagnosis, research and intervention. The heterogeneity of individual patterns of symptoms reported in dyslexia causes problems in differential diagnosis and raises questions about the aetiology and overlap of various disorders (Gilger, Pennington & DeFries, 1992). For example, Frith (2001) would view motor deficits in dyslexia as the manifestation of a co-existing disorder categorised at the behavioural level. Gilger and Kaplan (2001) however, regard such characteristics as the variable expression of a single underlying impairment that elicits patterns of characteristics according to individual brain development.

Frith (2001) promotes using the concept of 'comorbidity' to describe the manifestation of co-existing developmental disorders. Kaplan, Dewey, Crawford and Wilson (2001) however, question the value of using the term 'comorbidity' in relation to developmental disorders, as it is a medical term used to describe distinct pathological syndromes. These authors contend that motor deficits and reading difficulties do not occur as separate, categorical constituents. Figure 1:6 illustrates Frith's categorical view of the comorbidity of attention deficits with dyslexia. The vertical linear relationship between the biological, cognitive and behavioural characteristics of each disorder is illustrated however, apart from the influence of the environment there are no other horizontal connections within the model. For example, it is not apparent how a temporal processing deficit is associated with impulse control.

Figure 1:6 Comorbidity within the three level model of developmental disorders (taken from Frith, 2001).

The co-existence of dyslexia with attention deficits is well evidenced (e.g., Willcutt & Pennington, 2000; Willcutt et al., 2005) as is the co-existence of motor problems with ADHD (Diamond, 2000; Gillberg, 2003; Hartsough & Lambert, 1982; Kadesjö & Gillberg, 2001; Piek, Pitcher & Hay, 1999; Kaplan, Wilson, Dewey & Crawford, 1998; Barkley, 1990). The relationship between dyslexia and DCD is relatively unexplored however, certainly within the UK, although O'Hare and Khalid (2002) have suggested that children with DCD should be assessed for phonological difficulties due to the high risk of reading and writing delay within this population. Prevalence of DCD is estimated at 6% of the population, a similar proportion to that of dyslexia and ADHD (Kaplan, Dewey, Crawford & Wilson, 2001). Furthermore, Dewey and Kaplan (1994) have found that different subtypes of DCD are associated with different academic, language and motor profiles of skills and deficits.

Kaplan et al. (2001) analysed the overlap of developmental disorders from the perspective of reading difficulties. They revealed that 65 children with RD from a sample of 126 (51.6%) also met the DSM-IV criteria for ADHD, which left a remaining 48.4% of 'pure' cases of dyslexia. A further significant overlap was found with DCD for both disorders and 29 children from the entire sample of 170 (17.1%) met the criteria for DCD in combination with either reading disability or ADHD. Furthermore, of 116 children who met the diagnostic criteria for ADHD, only 21 (19.6%) were classified as 'pure' ADHD leaving 80.4% presenting symptoms of at

least one other disorder, 63 (54%) also met the criteria for reading disability (Kaplan et al., 2001). A longitudinal study of 409 Swedish primary school children also revealed that the rates of comorbidity with reading and writing disorder were high in both ADHD (40%) and subthreshold ADHD (29%) and rates of comorbidity with DCD were 47% for both groups (Kadesjö & Gillberg, 2001). The evidence for an overlap of symptoms between developmental disorders is therefore convincing. The debate as to which framework (categorical or continuum) to use for understanding developmental disorders, however, continues. As Lyytinen (2001) points out, dyslexia may be the exemplar for developmental disorder research. Cases of pure disorders, as described by diagnostic tools such as The Diagnostic and Statistical Manual of Mental Disorders, text revision, (DSM-IV-*TR*) (American Psychiatric Association, 2000) may be the exception rather than the rule.

One further complication is that ADHD is defined along three dimensions: predominantly-inattentive (ADHD-PI), hyperactivity-impulsivity (ADHD-HI) and combined (ADHD-C). Each dimension should be examined separately as a child could present symptoms of more than one (Bjorklund, 1995). Manifestation of the predominantly-inattentive type includes daydreaming, inability to sustain focus of attention on a task and distractibility. Manifestation of the hyperactivity/impulsivity subtype includes restlessness, fidgeting and excessive talking (Biederman, 2005). Moreover, each dimension of ADHD has been shown to be associated with different types of motor deficit. For example, ADHD-PI is more strongly associated with poor fine motor skills, whereas ADHD-C is more strongly associated with gross motor deficits (Piek, Pitcher & Hay, 1999), although Pitcher, Piek and Hay (2003) failed to find any difference between subtypes, including ADHD-HI, on measures of gross motor skills. Piek, Pitcher and Hay (1999) called for motor assessment to form a mandatory part of assessment for ADHD and later the same team called for reciprocal assessment of attention deficits in DCD (Pitcher, Piek & Hay, 2003). Approximately 50% of children with ADHD may be experiencing the same problems attributable to DCD but without the appropriate intervention or support. The long term outcome for these disorders when they appear in combination, in terms of independence, social behaviour and substance abuse, is significantly worse than that for single disorders (Rasmussen & Gillberg, 2000). Longitudinal studies have

revealed that the most severe perceptual motor deficits manifested during childhood tend to persist into adulthood in DCD (Losse et al., 1991; Cantell, Smyth & Ahonen, 2003). Perceptual-motor deficits have also been identified in dyslexia and ADHD (e.g., Gillberg, 2003; Stein, 2001; Stein & Walsh, 1997). The prognosis for adolescents with dyslexia and symptoms of ADHD, DCD or both must, therefore, be assumed to be at risk of negative affect and potential social maladjustment. Consensus upon the framework within which developmental disorders are studied and diagnosed may therefore be of the greatest importance to these children with multiple deficits.

1.3.2 The genetic basis of comorbidity

Pennington (2003) suggested that dyslexia arises in part from a genetically influenced change in brain development that mainly affects phonological processing but which may equally affect other functional systems. An early change in brain development is unlikely to affect just one system, hence "comorbidity is likely" (p.19). Large sample genetic linkage studies have identified five replicated locations for risk alleles influencing dyslexia, these are 2p, 3p-q, 6p, 15q and 18p, with indicative findings on 1p, 21q and Xq (Fisher & DeFries, 2002). Location 6p is the best replicated risk locus and this site also influences development of ADHD (Willcutt et al. 2002). Evidence has shown that reading difficulties and ADHD co-occur more frequently than would be expected by chance in both clinical samples and non-referred community samples (Semrud-Clikeman et al., 1992; Willcutt, Pennington & DeFries, 2000). Willcutt and Pennington (2000) predict therefore that the comorbid association between dyslexia and ADHD arises from common genetic aetiological factors. This hypothesis is supported by a growing body of evidence from twin studies, which suggests that dyslexia with ADHD is highly heritable and potentially associated with more than one gene (e.g., Light et al., 1995; Willcutt, Pennington & DeFries, 2000; Willcutt et al., 2003).

Ongoing genetic linkage studies are looking for chromosomal loci that may contain genes that increase the probability for the co-occurrence of dyslexia and ADHD. To date chromosomal regions have been identified on chromosome 6, 16 and 17 (Loo et

al, 2004; Willcutt et al., 2002, 2003). Genes that have an effect on more than one phenotype are named pleiotropic. A recent collaboration between the universities of Oxford, UK, Colorado, Denver and Nebraska, US, identified a new locus at chromosome 14q and supporting evidence for loci at 13q that have a pleiotropic impact on dyslexia and ADHD. A locus at chromosome 20q was also indicated (Gayán et al., 2005). Although research is ongoing, the case for a common genetic aetiology of comorbid dyslexia and ADHD is promising. However, the precise mechanism by which these genes influence neurocognitive development is unknown, although it is accepted that a single gene can have multiple effects on cognitive functions (Willcutt et al., 2005). A further complication identified by Willcutt et al. (2003) is that linkage to chromosome 6 was stronger for dyslexia with hyperactivity-impulsivity than for dyslexia with inattention, as measured on the DSM-IV symptom dimensions, indicating that further research is warranted and that identification of comorbid subtypes is likely to be complex. Ultimately, identification of a common neurocognitive marker that reflects a common genetic risk for comorbid dyslexia and ADHD is required and 'slow and variable temporal processing' is proving to be a promising link because it is the most frequently occurring common cognitive and behavioural characteristic in dyslexia with ADHD (Willcutt et al., 2005, p.71). The first genetic study of DCD has recently revealed a potential shared genetic aetiology with ADHD (Martin, Piek & Hay, 2006). Future genetic studies of co-occurring dyslexia with ADHD and/or DCD are certainly required. However, as Pennington (2003, 2005) pointed out, genetic influences are only part of the story of dyslexia, which is a complex and multifaceted disorder, environmental and cultural factors also have a role to play.

1.3.3 ADHD: Theories

Given the high prevalence of co-occurring ADHD with dyslexia, causal theories of ADHD and evidence for the neural basis of ADHD are of particular relevance to the thesis. Briefly summarised, three single core deficit causal theories of ADHD have been proposed: response inhibition (Barkley, 1997), delay aversion (Sonuga-Barke, 2002) and regulation of arousal/activation (Sergeant, Oosterlan & Van den Meere, 1999). No single theory has adequately accounted for the heterogeneity of symptoms associated with ADHD and much of the evidence presented for either one is

contentious. One reason for this may be that most studies are descriptive comparisons between ADHD and control samples. The majority have not differentiated between classification subtypes and most have recruited opportunity samples of combined type ADHD (Castellanos & Tannock, 2002). The ratio of cases that manifest either a single or multiple neuropsychological deficits, either cognitive or motivational, is as yet unknown (Pennington, 2005).

1.3.3.1 Response inhibition

Barkley's unified theory of ADHD (Barkley, 1997) is a simple model of cognitive dysfunction whereby behavioural symptoms of the disorder are caused by reduced inhibitory control, which is essential to the regulation of all behaviour and executive function. Executive functions (EFs) are variably classified as psychological constructs that include response inhibition, working memory, planning, organisation, fluency, abstraction and facets of attention (Lyon & Krasnegor, 1996). Nigg (2000; 2001) suggested that executive inhibition could be further defined according to whether the responses are principally cognitive, motor or related to response conflicts. Tests of inhibitory processes are typically either executive (conscious and deliberate), motivational (driven by fear of punishment) or automatic (Nigg, 2000). Response inhibition is the aptitude to inhibit an inappropriate response for a more appropriate alternative (Sonuga-Barke, 2005). Slowed and variable response inhibition has been replicated by several research groups and has been proposed as a candidate endophenotype for ADHD (Castellanos & Tannock, 2002; Crosbie & Schachar, 2001; Nigg, 2001). EF deficits however, are not ubiquitous in ADHD (Doyle et al., 2005). Furthermore, inattention and not hyperactivity/impulsivity subtype has been found to be the strongest predictor of slowed response inhibition (Chhabildas, Pennington & Wilcutt, 2001). Slowed and variable response inhibition on measures of temporal processing has also been revealed in dyslexia and this may be one candidate endophenotype for co-occurring ADHD with dyslexia (See section 1.3.2 , [Willcutt et al., 2005]).

1.3.3.2 Delay aversion

Delay aversion is a negative affective reaction to imposed delay that can manifest as a preference for a small immediate reward over a longer wait for a larger reward (Sonuga-Barke, 2002; 2005). According to this theory, the effect of future rewards on behaviour is reduced because of fundamental abnormalities in the signalling of neural reward mechanisms (Schultz, 2001). This leads to a shortened delay gradient whereby there is a faster decline in the effectiveness of the reward as the delay between the behaviour and the reinforcement is increased. Furthermore, according to this theory, hyperactivity is a primary behavioural manifestation of compensatory responses arising from delay aversion (Sonuga-Barke, 2002).

In summarising a large collaborative study, Solanto et al. (2001) revealed that when children were tested on measures of response inhibition and delay aversion, differences between ADHD and control participants were larger in the case of delay aversion. Furthermore, there was no correlation between the differences within subjects leading Sonuga-Barke (2002, 2005) to propose a multiple developmental pathways model to account for the heterogeneity of characteristics found in the classification subtypes of ADHD. Sonuga-Barke (2005) proposed that disruption of the frontal striatal (prefrontal-dorsolateral neostratum) underpins executive function deficits whereas delay aversion deficits are underpinned by disruption of the orbitofrontal-ventral striatum.

1.3.3.3 Regulation of arousal/activation

Sergeant, Oosterlan & Van den Meere (1999) proposed that the main underlying deficit in ADHD is dysfunction in the regulation of arousal and activation. Sergeant (2005a) reviews this 'cognitive-energetic' model of ADHD in which he describes effort, arousal and activation in terms of three energy pools. Effort is the energy necessary to meet task demands and is influenced by such factors as cognitive load. Effort is required when the current neurophysiological state of the individual does not match that required to perform a particular task in context. Effort is hypothesised to depend upon the hippocampus to excite and inhibit arousal and activation. Arousal is the phasic (periodic) responding to a stimulus during temporal processing. Time

locking to the stimulus is influenced by salience, novelty and signal intensity. The arousal pool is hypothesised to emanate from the mesencephalic reticular formation and the amygdala. Activation is the tonic (background) physiological readiness to respond to a stimulus. It is influenced by task and individual factors such as preparation, time of day, time spent on task and alertness. The activation pool is hypothesised to arise from the basal ganglia and striatum. State dysregulation arising from the relationship between these energy pools is therefore hypothesised to be the cause of cognitive and behavioural symptoms of ADHD. Activation and effort pools in particular are assumed to be closely linked and have a significant effect on motor control (Sergeant et al., 1999).

1.3.4 The neural basis of ADHD

Earliest theories of the neural basis of ADHD proposed disorders of neurotransmission leading to chemical imbalance in the brain. First suggested was the dopamine hypothesis that subsequently stimulated a large body of genetic research (Castellanos & Tannock, 2002). Dopamine, noradrenaline and adrenaline are catecholamine neurotransmitters involved in the regulation of movement, mood, attention and visceral function (Bear, Connors & Paradiso, 2001). Dopaminergic neurons are involved in motivation and noradrenergic neurons are involved in selective attention (Volkow, Wang, Fowler & Ding, 2005). Candidate genes for ADHD have been linked to dopaminergic and noradrenergic pathways (Castellanos & Tannock, 2002). Highest concentrations of dopamine are typically found in the striatum. In ADHD abnormalities in the volume of the caudate nucleus have been found although inconsistent findings have been attributed to laterality/asymmetry differences. The dopamine transporter (DAT) is the main mechanism by which the dopamine terminal removes dopamine that has been released in response to a salient stimulus. DAT regulates the concentration of DA in the synapse and hence regulates both the magnitude and the duration of the dopaminergic signal (Pliszka, 2005). Striatal DAT density is elevated in adults with ADHD (Dougherty et al., 1999; Krause, Dresel, Krause, Kung & Tatsch, 2000). Elevated DAT density leads to increased re-uptake of DA and consequently reduced magnitude and duration of the signal.

Methylphenidate hydrochloride (MPH) is a psychostimulant medication prescribed for ADHD. It increases the level of dopamine in the synapse and extracellular space by blockading both DAT and noradrenaline transporters (Dougherty et al., 1998; Krause et al., 2000; Solanto, 1998). Dysfunction of the regulation of dopaminergic and noradrenergic systems plays a critical role in the pathogenesis of ADHD (Volkow et al., 2005). Dysfunction of the neurotransmission of dopamine leads to the dysregulation of the circuits modulated by dopamine, namely the frontal (prefrontal, motor frontal, cingulate gyrus); subcortical (striatum, mediodorsal thalamus) and the limbic (nucleus accumbens, amygdale, hippocampus). Volkow et al. (2005) suggest that one hypothesis for the therapeutic effect of MPH on symptoms of ADHD is that it simplifies the saliency of a stimulus that drives interest and attention. Dopamine is also converted to noradrenaline following uptake by noradrenergic neurons. Balanced interaction between the dopamine and noradrenaline systems is therefore essential for the several functions that manifest as symptoms of ADHD.

According to a review by Krain and Castellanos (2006) the key brain abnormality in ADHD involves structural and functional anomalies in the frontostriatal circuitry (Castellanos et al., 1997; Faraone & Biederman, 1998). The prefrontal cortex (PFC) has been found to be significantly smaller in ADHD (Castellanos et al., 1996; Filipek et al., 1994; Kates et al., 2002; Mostofsky et al., 2002). Symmetry differences in the PFC have also been found with the right PFC significantly smaller than normal (Castellanos et al., 1996; Hynd, Semrud-Clikeman, Loveys, Novey & Eliopulos, 1990; Reiss et al., 1996; Shaywitz, Shaywitz, Byrne, Cohen & Rothman, 1983; Yeo et al., 2003). Sowell et al. (2003) measured the cortical surfaces of adults and children with ADHD by analysing the distance between the centre of the brain and the cortical surface. They found that this distance was smaller bilaterally in ADHD. Volumetric and asymmetry differences have been observed in the basal ganglia in ADHD but the evidence is inconsistent (Krain & Castellanos, 2006). Volume of the caudate nucleus is smaller (e.g., Aylward et al., 1996; Castellanos et al., 2002; Hill et al., 2003). Volume of the left caudate has been shown to be larger than the right (Castellanos et al., 2001; Filipek et al., 1997; Giedd et al., 1999). The caudate nucleus, like the cerebellum also participates in the development of both motor and cognitive function (Diamond, 2000). Differences in the putamen that may contribute

to the motor control symptoms of ADHD however, have been inconsistent and ambiguous (Krain & Castellanos, 2006).

In terms of relevance to the thesis, the key area of brain abnormality in ADHD is the cerebellum. Smaller cerebellar volumes have been observed in children with ADHD and this has been shown to be sustained throughout adolescence (Berquin et al., 1998; Durston et al., 2004; Hill et al., 2003). Castellanos et al. (2002) compared 152 children and adolescents with ADHD with 139 matched controls and found volumetric reductions in all brain regions measured. When they adjusted for total cerebral volume (TCV) only the cerebellar volume remained significant. Also, cerebellar volumes were significantly and negatively correlated with ratings of attentional problems. Berquin et al. (1998) also found that vermal volume was significantly smaller in ADHD than controls even when TCV was controlled for. Furthermore, one of the most consistent findings in structural MRI studies is a smaller volume of the cerebellar vermis in ADHD in comparison with control participants, in particular lobules VIII –X of the posterior inferior vermis region (PIV) (Berquin et al., 1998; Bussing et al., 2002; Castellanos et al., 2001; Hill et al., 2003; Mostofsky et al., 1998). This region has been shown to regulate and synchronize balance, locomotion, eye movements, postural and vascular tone (Anderson, Lowen & Renshaw, 2006; Ghez & Thach, 2000; Morton & Bastian, 2004; Voogd, 2003; Voogd & Glickstein, 1998). All but one study failed to identify reduced volume in lobules I-VII (Hill et al., 2003). In support of these studies of boys, Castellanos et al. (2003) found significantly smaller TCV in 50 girls with ADHD than 50 girl controls. Adjustment for TCV revealed significantly smaller volumes in the PIV region. No other regions, even those revealed in boys, were found to be significantly smaller after covariance. The PIV is therefore the most robustly replicated finding in MRI studies of ADHD (Castellanos & Tannock, 2002; Anderson et al., 2006). Balance impairments have been frequently revealed in ADHD (Piek, Pitcher & Hay, 1999). For example, Raberger & Wimmer (2003) found that children with ADHD demonstrated weaker balance control than children with ADHD and dyslexia, who in turn were weaker than children with 'pure' dyslexia.

PIV lobules VIII-X differ from the remaining hemispheres and vermis in selectively containing dopamine transporter-type immunoreactive axons (Melchitzky & Lewis, 2000). The cerebellar vermis also shows evidence of psychostimulant sensitivity (Anderson et al., 2002; Ernst et al., 1997; Volkow et al., 2005). Furthermore, Glaser et al. (2006) suggested that DAT in the posterior inferior vermis is concerned with the regulation of noradrenaline. Castellanos and Tannock (2002) speculate that the putatively dopaminergic fibres in the PIV may form part of a cerebellar circuit that influences the ventral tegmental area and the locus coerulus. The ventral tegmental, a midbrain nucleus, is the main supplier of dopamine to the cortex. The locus coerulus, a nucleus of the brain stem, is the main supplier of noradrenaline to the cortex (Pliszka, 2005). Pliszka (2005) suggests that although dopamine and noradrenaline are implicated in the pathophysiology of ADHD the main contribution may not be the total level of either produced but the amount produced relative to a particular task. If the amount released by the ventral tegmental area or locus coerulus is too small, it could lead to symptoms of inattention and underactivity. Alternatively, if too much is released it could lead to overstimulation and disordered executive function (Pliszka, 2005). Given the hypothesised role for catecholamine dysregulation in the pathophysiology of ADHD (Castellanos & Tannock, 2002; Krain & Castellanos, 2005) these studies provide supporting evidence for the involvement of lobules VIII – X in ADHD. The role of this region in balance is discussed in Chapter 2. Reported balance impairment or postural instability in dyslexia may therefore arise from cerebellar deficits in co-occurring dyslexia with ADHD. Whether such impairment is associated with inattention or hyperactivity symptoms however, is as yet unexplored. Evidence of cerebellar dysfunction in subtypes of DCD has also been revealed (Ivry, 2003; O'Hare & Khalid, 2002). Research into the neural basis of DCD using neuroscience methods to date is sparse.

1.3.5 Developmental influences on deficits in motor control

Ivry and Justus (2001) cite neuroimaging evidence of cerebellar abnormality in autism (Courchesne, 1997), schizophrenia (Nopoulous, Ceilley, Gailis & Andreason, 1999) and ADHD (Berquin et al., 1998) in their discussion of the cerebellar deficit hypothesis in dyslexia. Given the high rates of prevalence of overlapping symptoms of motor deficits in developmental disorders described in section 1.3.1, why is there

so much variability in the patterns of deficits manifested within the population? If anomalous structure and function of the cerebellum is the underlying cause of developmental disorder we would predict that motor deficits would be manifest in all cases. Diamond (2000) suggests however, that characteristics of developmental disorders do not always covary due to the multiple neural connections within the cerebellar-cortical circuits. In particular she explains how the multitude of neural connections between the dorsolateral pre-frontal cortex, basal ganglia and the cerebellum can be influenced by both genetic and environmental factors. According to this view, cerebellar impairment may arise as a consequence of pathology located elsewhere within the neural circuitry of the cerebellar-cortical loop as Zeffiro and Eden (2001) postulate.

One attractive and recent framework, the neuronal group selection theory (NGST) (Sporns & Edelman, 1993), effectively underpins the epigenetic process of neurogenesis that Diamond (2000) describes and Zeffiro & Eden (2001) propose. Hadders-Algra (2000a) suggested that this theory could lead to a clearer perception of the mechanisms which underpin motor deficits in developmental disorders. According to NGST, the Central Nervous System (CNS) selects groups of multitudes of interconnected neurons which are dynamically organised into functional networks either, for example, for processing information from a particular sensory modality, or for performing a particular movement. Motor development proceeds through two stages of primary and secondary variability. In the primary variability stage, repertoires of multiple neuronal groups determined by evolution produce variable motor behaviour which elicits self-generated, variable afferent information. This information, the result of behaviour and experience, is used to select a motor behaviour broadly but not specifically suitable for a particular situation. After selection is achieved, behavioural variability is reduced but returns with exposure to new experience. Afferent information leads to modification of synaptic connections between and within neuronal groups leading to the secondary or adaptive variability stage, whereby movements are refined precisely to meet task and environmental demands. Within this framework, neurogenesis proceeds from genetic predisposition via environmental and experiential influences that shape and define uniquely specified functional networks (Edelman, 1989; Spons & Edelman, 1993).

Karmiloff-Smith (1998) also emphasises the importance of the dynamic developmental process itself to our understanding of developmental disorders. In this neuroconstructivist framework initial genetically predisposed deficits lead to differential phenotypic outcomes because of the effects of the environment and the developmental timecourse on neurogenesis. She emphasises the influence of dynamic interaction between genes, environment and temporal progress on the developing neural systems in the complex unfolding of typical and atypical development in children. This approach provides a neurocomputational model that supports Pennington's (2003) view and conceptualises Willcutt et al.'s (2005) prediction that a single gene can have multiple effects on cognitive functions. This would also extend to motor function at a behavioural level. Scerif and Karmiloff-Smith (2005) further caution against simply mapping genotype to phenotype without giving adequate consideration to developmental processes and relationships between areas of relative cognitive strength and weakness. Furthermore, Karmiloff-Smith (1998) highlights the flaw in the unidirectional vertical linearity in the Frith (2001) causal framework for developmental disorders that fails to capture the differential phenotypic outcomes observed in co-existing developmental disorders. If we extend this dynamic view of developmental disorders to the domain of motor function we can begin to better understand the heterogeneity and overlap of comorbid symptoms in developmental dyslexia. This non-linear systems approach to development and motor control will be a theme that runs throughout the thesis motor control in dyslexia is studied in terms of contemporary theory and practice.

1.4 Motor control deficits in dyslexia: defining the central issue

Having identified the issues of differential diagnosis of dyslexia and the co-occurrence of developmental disorders with dyslexia, I will now begin to review the empirical evidence for motor control deficits in the dyslexia population. The range of gross and fine motor skills tested in the dyslexia literature ranges from walking backwards to bead threading. The most frequently tested motor skill is balance, or postural stability and it is on measures of this function that the most notable results have been reported (e.g., Nicolson & Fawcett, 1995). Postural stability is the maintenance of equilibrium or balance and this is typically impaired with cerebellar

dysfunction (Ghez & Thach, 2000) or reduced sensitivity within the sensory systems (Horak, Nashner & Diener, 1990). Balance depends upon the successful temporal and spatial integration of perceptual information, adequate muscle tone, and the accurate sequencing and timing of muscle synergies to correct the alignment of body segments. According to Gibson (1966) the maintenance of postural stability is fundamental to and inseparable from the successful achievement of all movement. Early difficulties with competent balance function might therefore be expected to impede the typical development of all gross and fine motor skills. Postural instability is acknowledged to be one of the 'soft neurological signs' associated with developmental disorders (Denckla & Rudel, 1976; Touwen, 1979). Several dyslexia screening batteries include tests of postural stability. In order to place the thesis within the wider context of postural and balance research and introduce a more objective approach to the study of balance in dyslexia, Chapter 2 describes contemporary theory and practice in the study of postural stability.

2 Postural stability

2.1 Posture and balance

Posture is the alignment of body segments and can be described in terms of three coordinate systems: 1) the egocentric system describes the relative position of body segments with respect to one another, 2) the exocentric system describes the orientation of the body with respect to the environment, 3) the geocentric system describes the orientation of the body with respect to the constraints imposed by gravity (Balasubramaniam & Wing, 2002; Melvill Jones, 2000). Postural control is the ability to orient body segments relative to one another and to the environment without loss of equilibrium. The terms balance, postural stability and equilibrium are interchangeable in postural research and are used alternately in the thesis (Shumway-Cook & Woollacott, 2001; Winter, 1995, 2004).

To maintain stability, the CNS has to accurately perceive sensory information about constant (e.g., gravity) or variable (e.g., external events or self-generated actions) forces upon a body then generate and coordinate muscle activity (kinematic adjustments) to create the forces needed to return body segments to alignment (kinetic adjustments). Balance therefore, is an active process (Balasubramaniam & Wing, 2002). In principle, there is no difference between the processes that underpin balance and any other movement (Melvill Jones, 2000). When segments are returned to alignment, a state of equilibrium exists, in which all forces acting on the body are balanced, so that the body remains in either an intended position (static equilibrium) or is able to generate a volitional movement without losing balance (dynamic equilibrium) (Melvill Jones, 2000). In this state, the Centre of Mass (COM) and its vertical projection, the Centre of Gravity (COG), rest within the base of support formed by the feet. The COM is the point at which the whole body's mass is equally balanced and the CNS continuously monitors its position (Winter, 1995; 2004; Woollacott & Shumway-Cook, 2002). The systems that underlie postural stability therefore, have to maintain steady stance, anticipate the effects of goal directed movements and be capable of adaptive learning (Melvill-Jones, 2000). The cerebellum may be involved in every stage of this process (Morton & Bastian, 2004).

Deficits in motor control: causes or correlates of reading disability?

In maintaining steady stance, the cerebellum modulates postural control by regulating muscle tone, eye movements and balance. Vestibular neurons inform the CNS about linear or angular acceleration of the head. When lateral imbalance is detected by the semicircular canals, a reflexive eye movement is generated in the plane of the canal that has generated the signal. This is known as the Vestibular Ocular Reflex (VOR). Its primary role is to control the position of the eye during dynamic head movements in order to secure a stable retinal image. It also arises from linear acceleration signals emitted by the otolith organs albeit to a lesser extent (Shephard & Telian, 1996). In addition, Optokinetic Nystagmus (OKN) uses a combination of smooth pursuit and saccadic eye movements to maintain clear visual images during sustained movement of the head. The vestibular system interacts directly with the optokinetic response via the velocity storage generator that is hypothesised to be located in the vestibular nuclei and the vestibulocerebellum described below (Kubo, Igarashi & Wright, 1981).

The vestibulocerebellum comprises the nodulus (lobule X) and part of the uvula (lobule IX) in the posterior inferior region of the vermis and also the flocculus and ventral paraflocculus, the most caudal regions of the hemisphere (Voogd & Glickstein, 1998). The vestibulocerebellum is closely linked to the vestibular system and specifically involved in the control of balance and eye movements. It receives inputs from the visual and vestibular systems and sends outputs via the medial vestibular nucleus to control eye movements and to coordinate motion of the head and eyes to stabilize the head in the external world (Ghez & Thach, 2000). It has also been shown to regulate eye movement responses to visual motion signals in maintaining fixation (Leigh & Zee, 1991). Suppression of eye movements has been demonstrated to improve balance (e.g., Jahn et al., 2002). The vestibulocerebellum also sends outputs via the lateral vestibular nucleus to regulate the postural tone of the axial and limb extensors that are activated to withstand gravity and to control the COM (Ghez & Thach, 2000; Morton & Bastian, 2004). This region of the cerebellum is central to the maintenance of postural stability and it has provided the most robust evidence of brain abnormality in ADHD (see section 1.3.4).

Deficits in motor control: causes or correlates of reading disability?

When perturbations to balance are unexpected, coordinated postural reflexes return the body to a state of equilibrium. The postural reflex latency (0.1s) is shorter than the latency for a volitional movement (0.2s) but considerably longer than that of a spinal reflex (0.03s). This indicates that supraspinal circuits, possibly the sensorimotor cortex, are recruited in addition to low-level spinal circuits (Balasubramaniam & Wing, 2002). Postural reflexes may be innate but they may also be adjusted through learning to adapt to contextual changes (Horak, 1996). They then become automatic and are adopted whenever equilibrium is threatened within a similar context. Postural adjustments in response to unexpected perturbations therefore, depend upon feed-back control (Melvill-Jones, 2000). Anticipatory responses that precede volitional movement however, depend upon feed-forward control. When one body segment moves in a given direction the COM follows, therefore to maintain the COG within the base of support another segment must move in compensation. Even respiration needs to be countered by muscle activation (Melvill Jones, 2000) particularly when heightened in a state of arousal (Maki & McIlroy, 1996). All volitional movement therefore, is preceded by an anticipatory postural readjustment based upon a prediction of the outcome threat to balance imposed by that movement.

The cerebellum compares the motor cortex command to the spinal cord (efference copy) with feedback from the postural systems that underpin anticipatory postural control (reafference). It then processes this information and outputs it to the motor cortex and other systems in the brain stem to adapt postural responses to the perceived threat to stability. This is the process whereby the cerebellum anticipates volitional movement (feed-forward) and monitors the COM (feed-back) in postural control (Ghez & Thach, 2000; Morton & Bastian, 2004). Internal representation of posture is essential to postural control (Gurfinkel, 1994). The cerebellum is also involved in the construction of internal models of motor control (Miall & Wolpert, 1996; Miall & Reckess, 2001). Miall (1998) further suggests that the cerebellum generates a feed-forward model based upon the efference copy of the instruction for the volitional movement. This model is used to adjust the movement online in advance of sensory feedback. In addition, the cerebellum may be the central locus for all motor coordination, which would include postural control (Miall, 1998). The

generation of a precise representation of posture therefore depends upon intact cerebellar function.

Knowledge of the contribution of the cerebellum to postural control is mainly derived from clinical studies of cerebellar lesions. For example, increased postural sway is specific to the area of cerebellar lesion: vestibulocerebellar lesions have been shown to evoke postural sway in quiet standing on both anterior-posterior (A/P) and medial-lateral (M/L) axes. Anterior lobe lesions however, evoke sway on the A/P axis only (Morton & Bastian, 2004). Furthermore, Horak and Diener (1994) found that cerebellar patients were unable to learn to use predictive feed-forward control to adapt postural responses to anticipated perturbations to balance. Adaptation in postural control therefore, requires an intact cerebellum. Structural or functional anomalies of the cerebellum in developmental disorders would likely lead to balance difficulties (see section 1.3.3). Tests of postural stability therefore, would potentially be valid indicators of developmental disorders associated with cerebellar dysfunction.

Postural sway can also be evoked by stimulation of visual (e.g., Dijkstra, Shöner & Gielen, 1994; Lee & Aronson, 1974; Lee & Lishman, 1975), vestibular (e.g., Day et al., 1997) or somatosensory (muscle proprioceptors, joint receptors, Golgi receptors, cutaneous and tactile receptors) information (e.g., Jeka et al., 1997; Inglis, Horak, Shupert & Jones-Rycewicz, 1994). The visual system provides cues to vertical orientation and motion of the body relative to the environment. The vestibular system provides cues to the linear and angular displacement and acceleration of the head. Together these systems aim to stabilize the body within the environment and are dominant in the postural control systems of children under 3 years (Lee & Aronson, 1974). The primary cues to orientation in the mature postural control system however, are the joint receptors that signal changes in the ankle, knee and hip angles. Information about the ankle angle is the most dominant (Shepard and Telian, 1996). Dorsiflexion (acute ankle angle) and flexion (obtuse ankle angle) in response to unexpected perturbation stimulate the innervation and inhibition of different sequences of muscle activation (e.g., gastrocnemius, hamstring, paraspinal muscles)

that underlie successful use of an ankle strategy. This strategy is typically employed in each similar context-dependent situation. However, in another situation where the perturbation force is perceived to be too great for successful use of this strategy, posture is corrected by the simultaneous contraction of the quadriceps femoris and abdominal muscles. This gives rise to a rotational movement of the trunk around the hip joint. This use of a hip strategy requires greater expenditure of energy and signifies reduced postural control (Horak, Nashner & Diener, 1990). Responses to joint angle changes are mediated through afferent pathways involving the spinal cord, brainstem and the cerebellum. These pathways project to the motor cortices from which efferent responses to the musculoskeletal system are projected. Coordination of activity in the muscle spindles consequently returns the body to a state of equilibrium (Shepard and Telian, 1996).

2.2 Theories of postural control

2.2.1 Facilitation or reflex-hierarchy theory

According to the reflex-based neurofacilitation approach to motor control, postural stability and movement typically develop from a sequence of reflexes that are organized hierarchically within the CNS. Incoming sensory information stimulates reflexive motor responses. Over the typical developmental time course, lower level spinal reflexes are gradually suppressed as movements become organized at higher levels, first within the intermediate level brain stem centres and the cerebellum, then within the higher level cerebral cortex. Motor control therefore becomes "top down" and its developmental progress is referred to as corticalisation (Shumway-Cook and Woollacott, 2001). Magnus (1926) first described a prototypical series of righting reactions, which correct the orientation of the body in respect to the environment in the development of postural control. This progression reflects the maturation of cortical structures, which inhibit and integrate lower level CNS activity, until structures critical for more functional voluntary movement have evolved. Neural pathways between the association areas of the neocortex, basal ganglia, motor, premotor and supplementary motor cortices, brain stem and spinal cord gradually mature as connections are strengthened or attenuated. This enables the development

of the strategies, tactics and execution of goal directed movement (Bear, Connors & Paradiso, 2001; Shumway-Cook & Woollacott, 2001).

In this framework, developmental motor disorders are considered to arise from the failure to inhibit and integrate reflex mechanisms. When the corticalisation process is interrupted movement is dominated by primary reflexes. Failure to inhibit and integrate primary reflexes has been demonstrated in children and adults with general learning difficulties (Freides, 2001). More recently, persistence of primary reflexes has been associated with reading difficulties in a large study of schools in Northern Ireland (McPhillips and Sheehy, 2004). In acquired disorders, deficits arise as a direct result of the lesion itself after corticalisation, in adults at least. This approach makes two critical assumptions. First, once atypical movement patterns are inhibited and typical movement patterns have been facilitated, functional abilities will improve in developmental disorders or return in acquired disorders. Second, continuous practice and repetition of typical movement patterns will automatically transfer to cognitive and motor functional abilities (Shumway-Cook and Woollacott, 2001). Therapeutic interventions based upon this approach were developed during the 1950s, '60s and '70s. For example, the popularity of perceptual-motor/sensory integration therapy (Ayres, 1972) for the remediation of learning difficulties gave rise to a large number of studies that were reviewed using meta-analysis by Kavale and Mattson (1985). The review, however, found little evidence for the efficacy of treatments based on this approach upon academic, cognitive or perceptual-motor function. Perceptual-motor/sensory integration training also forms the basis of a highly-publicised and controversial commercial regime for improving reading skills which is currently expanding within the UK, USA and Australia (see Reynolds, Hambly and Nicolson, 2003). Reflex-hierarchy theories are still applied in physical therapy today, although there is less emphasis placed upon reflex inhibition and more prominence is given to functional training in the motor domain (Shumway-Cook and Woollacott, 2001).

2.2.2 Control or systems theory

More recent theories can be subsumed under the umbrella term 'dynamic systems theories', in which motor control emerges from the complex interaction of maturing neural networks and musculoskeletal structures as the CNS attempts to map and refine patterns of internal neural oscillations to the temporal and spatial patterns provided by the environment. Thelen and Smith (1994) questioned the reflex-hierarchical account of the emergence and suppression of reflexes, such as the stepping reflex in new born infants. Instead, they described these changes in behaviour in terms of heterarchical sensorimotor responses to contextual changes. In their view, behavioural manifestation is completely context dependent and development evolves within an ontogenetic state-space landscape. Motor equivalence, or ways of organising underlying systems in order to achieve a goal, emerges from the ability to map sensory input to motor response output within the constraints of the individual level of neural network maturation, the nature of task and the constraints of the environment (Woollacott and Shumway-Cook, 1990).

According to this framework, genetic and environmental constraints upon individual development begin at the moment of fertilisation. Environmental and genetic influences continue to constrain development throughout each pre-, peri- and post-natal stage as the neural networks form unique patterns of connections that generate the foundation of the sensory and motor systems. During the process of self-organization, coherent structure and pattern arise from the cooperation of many smaller neural components. This process, which takes place within parameters specified by time, is the basis of non-linear dynamic systems theory, which is founded upon a large body of research evidence from motor control and developmental research (for reviews see Kelso, 1999; Lewis, 2000; Lewkowicz, 2000; Thelen and Smith, 1994). Shumway-Cook and Woollacott (2001) suggest that postural control develops according to cephalocaudal and proximodistal developmental principles and the simultaneous maturation of postural, locomotor and manipulative systems is fundamental to the emergence and refinement of gross and fine motor skills. Reflexes are only one of many influences on the development of postural control and movement. According to reflex-hierarchy theories, visuo-motor coordination develops at 2m due to the appearance of the optical righting reaction.

Dynamic systems theories, however, suggest that mapping vision to posture is present at birth and with experience more refined rules for mapping sensory information to action emerge (see Table 2:1).

Mapping between the visual and vestibular systems emerges at around 2.5m. The visual system has a predominant role in the earlier emergence of sensory motor mapping but in certain age groups postural response synergies appear better organised without vision. For example, Shumway-Cook and Woollacott (1985) found that children under 7 years were unable to maintain stability efficiently using only vestibular cues. Foudriat, DiFabio and Anderson (1993) however, found that predominance of visual control gives way to somatosensory control by 3 years although adult-like postural responses are not apparent until age 7 years.

Table 1:1 Systems model showing the emergence of critical stages in the development of postural control (adapted from Shumway-Cook & Woollacott, 2001).

Within the systems framework, sensory information has a much more prominent role than that of its stimulus-response reflexive function within the facilitation model. It is essential in guiding the predictive and adaptive control of movement. Atypical movement patterns emerge not only from the impairment of one of the contributing systems but also from the adaptive involvement of all the other systems in compensation. In a typically developed postural control system there is wide variability of sensory information available to the individual with respect to egocentric, exocentric or geocentric coordinate systems (Melvill Jones, 2000). In maintaining postural stability, the individual uses this redundancy of information to select the best adaptive motor responses from the available degrees of freedom in order to control stability. The greater the sensory redundancy available, the less variability produced in the adaptive response. Where sensory redundancy is lowered in one system other systems compensate, but stability becomes more effortful because compensatory strategies are not always efficient (Shumway-Cook and Woollacott, 2001). Figure 2:1, illustrates the numerous systems that potentially contribute to postural control.

Figure 2:1 Graphic illustration of the systems contributing to postural control (adapted from Shumway-Cook and Woollacott, 2001).
The systems that underpin postural control. Deficits within any one system would influence postural control, as would the adaptive contribution of the remaining intact systems.

In clinical applications, control or systems theory takes a task-oriented approach. Compensatory strategies are targeted for improvement of motor skills. Specific tasks are identified and practiced. The key assumption that sets this approach apart from

the facilitation/reflex-hierarchy theory is that movement repetition is task specific, rather than simply a repetition of generalized typical movements. Development of strategies is therefore, also task specific and the individual learns a number of ways to achieve a behavioural goal within the available degrees of freedom of action. Ability to adapt to dynamic changes within the environment is also an essential part of function training and strategy development (Shumway-Cook & Woollacott, 2001). Hadders-Algra (2000b) further suggested that intervention treatments for developmental motor disorders should focus on a wide range of sensory and motor experiences and involve active practice of motor skills. The control model is currently the most widely supported framework in the sphere of postural and balance research and it is within this framework that I have attempted to advance the study of balance in dyslexia.

2.3 The role of attention in postural control

Facilitation/hierarchical reflex theory suggests a gradual reduction in the role of attention throughout development. This role increases and decreases as each stage of postural control is mastered, until it takes on a planning and monitoring role once postural stability has become mature. Control/dynamic systems theory however, predicts an ongoing role for attention according to the perception-action-cognition cycle (see Figure 2.2) within the constraints of the individual, the task or the environment. When variables are stable, postural stability is normal because internal and external states are balanced, hence minimal attention is required. However, when one or more variables destabilize the cycle, for example, when postural stability is perturbed due to an externally generated force, the continuum of attention used is organised according to the degree of effort required to re-establish stability (Shumway-Cook and Woollacott, 2001). Control theory predicts that postural stability emerges from the temporal coupling of the body within the environment and of body segments within the body. The review by Balasubramanian and Wing (2002) provides evidence of a strong perception-action relationship between postural stability and changes in the environment, with perception being the psychological meaning applied to incoming visual, vestibular, cutaneous and proprioceptive sensory information; action being the pre-programmed or task-specific assembly of

predictive or reactive muscle synergies. Cognition interacts with these processes and it is the effect of cognition on perception and action that is typically measured in dual task paradigms. This cycle of mutually influenced and influencing variables is illustrated in Figure 2:2.

Figure 2:2 Graphic representation of the perception-action-cognition cycle in postural control (adapted from Shumway-Cook & Woollacott, 2001).
Each element within the system influences and is influenced by the other elements. In this way, the mapping of sensory input to motor output influences and is influenced by available cognitive resources.

The review by Woollacott and Shumway-Cook (2002) suggests that attentional requirements for postural control vary according to the postural task, age of the individual and their ability to sense and adapt to environmental stimuli. Figure 2:3 illustrates the mutually influencing relationship between the individual, task and environment in postural control.

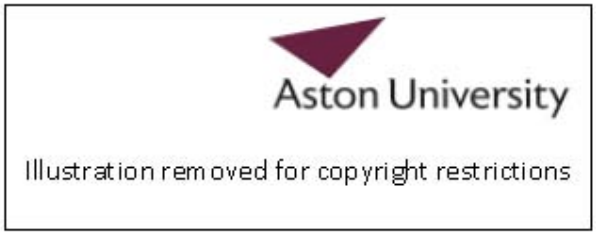

Figure 2:3 Graphic illustration of the individual-task-environment cycle in postural control (adapted from Shumway-Cook & Woollacott, 2001).
The individual, the task and the environment interact to influence postural control.

Horak, Nashner and Diener (1990) showed that the CNS weights and selects each sensory frame of reference according to the variability of the available information. They used the Sensory Organization Test (SOT) to assess postural sway in quiet stance with eyes open, blindfold and whilst wearing a visual surround to remove visual cues to orientation, standing both on a firm support surface and on a compliant foam surface. As predicted by systems theories of postural control, where there is greater redundancy of sensory information available for selection, fewer attentional resources are required to maintain stability. If visual, vestibular, tactual or proprioceptive input is distorted due to individual or environmental constraints, however, increased attentional demands are placed on central resources to compensate for loss of at least one part of the CNS frame of reference hierarchy. Woollacott and Shumway-Cook (2002) suggest that this provides evidence against postural control being a wholly automatic or reflex control task because low level postural responses would continue to control stability irrespective of the availability of sensory information. Likewise, more attention is required in open-loop tasks, for example when the support surface is continuously unpredictable when standing on a moving platform. Closed-loop tasks, however, such as during quiet stance with feet together on a firm support surface, require lower level processing and fewer attentional demands. Removal of only one source of sensory information, such as when participants are blindfold, creates attentional demands, even when low-level automaticity of skills remains intact (DiFabio & Badke, 1991).

Postural stability can therefore be threatened under experimental control by either the manipulation of the task through cognitive load, as determined by task difficulty in dual task paradigms, or by the manipulation of the environment through perceptual load, as determined by the removal, distortion or transience of available sensory information. Figures 2:4 and 2:5 illustrate the potential means of manipulating postural stability within a dynamic systems framework. In dyslexia, the cerebellar deficit hypothesis predicts that manipulation of the task draws attention away from postural control and towards the task in "conscious compensation" (Nicolson & Fawcett, 1990, p.3). It also predicts that failure to automate postural responses would threaten stability when sensory cues to orientation are manipulated. Co-existing

attention deficits would be predicted to impair postural control irrespective of automaticity.

Figure 2:4 Graphic illustration of potential methods for the manipulation of postural stability (adapted from Shumway-Cook & Woollacott, 2001).
The red arrow indicates how dual task paradigms can manipulate postural stability. The blue arrow indicates how paradigms that modulate sensory perception of the external word can manipulate postural stability under experimental control.

Figure 2:5 Graphic illustration of the influence of dual task and sensory modulation paradigms on postural stability (adapted from Shumway-Cook & Woollacott, 2001).
The red arrow indicates how dual task paradigms can manipulate postural stability. The blue arrow indicates how paradigms that manipulate sensory cues to orientation can manipulate postural stability under experimental control.

2.4 The way forward

In Chapter 1, it was suggested that the evidence for deficits in balance in dyslexia is inconsistent and identified issues of diagnosis and the co-occurrence of developmental disorders with dyslexia. It was also proposed that the thesis would be placed within the wider context of postural and balance research and that a more objective approach to the study of balance in dyslexia would be introduced. In this chapter current theories of postural control have been examined and methods of applying these to manipulate postural stability under experimental control have been identified. The next step is to introduce a quantitative approach to the studies of balance in dyslexia. Tests of postural stability are included in the range of dyslexia screening batteries devised by the Sheffield Research Group (e.g., Fawcett & Nicolson, 1998; Fawcett & Nicolson, 2004a; Fawcett, Nicolson & Lee, 2001). In reviewing the literature two questions need to be addressed. First, is balance related to reading specifically or by association with other developmental disorders? Second, can balance measures be used to predict reading failure? The quantitative review in Chapter 3 examines the reported findings of empirical studies of balance function in the dyslexia population.

3 A meta-analysis of dyslexia and balance studies

3.1 Introduction

A meta-analysis of the past 20 years' findings of balance studies in dyslexia was conducted in order to bring a more rigorous and objective approach to the existing body of evidence. The paper arising from this chapter is currently in the press (see appendix 1). Meta-analysis is not simply a statistical procedure; it is a rigorous and objective method of systematically combining data from a number of studies. It involves generating hypotheses, conducting a meticulous search of the literature, setting exclusion and inclusion criteria for studies, documenting, amalgamating and examining data across studies, looking for common characteristics that may act as moderator variables to explain relevant effects and, finally, reporting results. Meta-analysis is a particularly useful tool where a body of evidence is equivocal, such as in the area of dyslexia and balance, as it provides a quantitative as opposed to a narrative summary of available research findings (Rosenthal and DiMatteo, 2001).

The effect size is the focal statistic of the meta-analysis. This provides an estimate of the effect of interest by generating a standardized mean difference independent of statistical significance. This is important because significance is influenced by the size of the study sample. The general relationship is as follows (Rosenthal, 1991):

$$\text{Test of significance} = \text{size of effect} \times \text{size of study}$$

Studies with small samples that report non-significant effects may actually produce large effects that would reach significance with the optimum sample size. One of the main benefits of this method is to bring to light the variability or stability of an effect across studies. Another is that it enables the meta-analyst to identify potential moderator variables from which the relative strength of effect can be predicted. Finally, by examination of the characteristics shared by studies that generate similar findings it may be possible to identify participant group profiles and investigate trends within the factors that commonly occur with balance difficulties. To my

knowledge this is the first meta-analysis of research in the area of dyslexia and balance.

At the close of Chapter One, two key issues to be addressed were identified: First, is balance related to reading specifically or by association with other developmental disorders? Second, can balance measures be used to predict reading failure? In order to address these issues the following research questions were devised and the guidelines for meta-analysis prescribed by Rosenthal (1991; 1995) were followed:

1. Do people with developmental dyslexia differ from normal readers on measures of balance?
2. Do effect sizes diminish when samples are known to be ADHD or DCD exclusive?
3. Do balance difficulties persist into adolescence and beyond?
4. Does the method of classification of dyslexia groups moderate effect size?
5. Do groups which vary on tests of balance also vary on measures of FSIQ?
6. Does reading disability predict postural instability?
7. Do dual task paradigms yield greater effect sizes than single tasks?
8. Do eyes closed or blindfolded tasks engender larger effect sizes than when eyes are open?
9. Does the level of data collected predict balance effect size?

3.2 Method

3.2.1 Criteria for inclusion

Data were collected from studies that reported comparisons of participants with dyslexia and control participants on measures of balance. To locate studies electronic database searches (*Pubmed, Psych Articles, PsychInfo*) were conducted using the terms: developmental dyslexia, motor impairment, motor deficits, motor skills, motor control, balance and postural stability. Archives of journals known to publish articles

on dyslexia *(e.g., Dyslexia, Annals of Dyslexia, Journal of Child Psychology and Psychiatry)* were also searched as were the reference lists of acquired journal articles. The following criteria for inclusion were used:

1. Publication date post 1984 as Denckla et al. (1985) had pronounced the *status quo* at that time and effectively prepared the way forward for scientific studies of dyslexia.
2. Research question with focus on dyslexia and balance.
3. Group means, standard deviations and sample size provided to enable estimation of effect size.
4. Independence of samples.

Seventeen peer-reviewed journal articles describing 16 studies were identified, of these, 15 provided sufficient information for the meta-analytic procedures. The criterion for independence was that participants had taken part in only one study. There were seven independent studies each with one or more balance dependent variable. Non-independent studies from the Sheffield and Salzburg Research Groups that stated that participants had been repeatedly recruited from the same sample pool were treated as one study with several dependent variables per research group (Rosenthal, 1991). This resulted in nine independent studies which yielded 70 effect sizes for balance.

3.2.2 Study characteristics

Study characteristics were recorded and coded wherever possible. These included: sample (size, age, gender, education, volunteer status, handedness, recruitment source and inclusion/exclusion criteria), design, test paradigm (self-generated or external perturbation, sensory manipulation, single or dual task), psychometric and literacy variables and level of data recorded for balance measurement.

3.2.3 Predictor variables

Potential categorical and continuous moderator variables were identified. Subject factors included the age of participants, the amount their reading ability deviated from their control group, their level of general intelligence, and the proportion of effects yielded from samples screened for ADHD for exclusion. Surprisingly, only two studies (Ramus et al., 2003; Ramus, Pidgeon and Frith, 2003) specifically referred to DCD so the contribution of this variable to balance heterogeneity was not examined beyond generating further hypotheses. Stimulus factors arising from the balance test paradigm included the proportion of effects yielded from dual task paradigms and eyes closed or blindfolded paradigms.

3.2.4 Meta-analytic procedures

Rosenthal (1991) describes six types of meta-analytic procedures which he divides into categories of significance testing and effect size estimation each with three levels of analysis. The first two levels involve comparing studies through diffuse tests, which assess whether studies differ statistically, and focused tests, which assess whether they differ in a theoretically predictable way. The third level of analysis involves combining studies in order to assess the overall level of significance and average size of the effect. In this meta-analysis the outcome of the selected studies was standardized by extracting the means, standard deviations and sample sizes provided. Effect sizes for all balance measures within each study were then computed, using Cohen's *d* pooled variance method (Cohen, 1988) shown in Equation 3:1:

Equation 3:1 Calculation of *d* effect size.

$$d = \frac{\text{dyslexia group mean - control group mean}}{\sigma}$$

In this method, σ equals the sum of squares around each mean divided by the total number of participants, which takes into account disparity between group sizes (Rosenthal, 1991). Cohen's *d* effect sizes, for each of the seventy effects, were then transformed into correlation coefficients (r_{effect}) using Equation 3:2 below for unequal

sample sizes, where p is the proportion of participants in the dyslexia group and q is the proportion in the control group:

Equation 3:2 Transformation of *d* effect size to r_{effect}, unequal sample sizes.

$$r_{effect} = \frac{d}{\sqrt{\left(d^2 + 1 \div (p \times q)\right)}}$$

For equal sample sizes Equation 3:3 was used (Rosenthal, 1991).

Equation 3:3 Transformation of *d* effect size to r_{effect}, equal sample sizes.

$$r_{effect} = \frac{d}{\sqrt{d^2 + 4}}$$

Each r_{effect} value was then converted to the Fisher transformation Zr to provide 3 common metrics, d, r_{effect} *and* Zr for each effect size.

3.2.5 Data synthesis

The mean Zr was computed in order to provide a single effect size for each study across all balance measures. For non-independent studies, the mean sample size per effect size was also calculated (Rosenthal, 1991). This generated nine independent effect sizes and nine independent sample populations. The effect sizes were then combined using the weighted Stouffer et al. (1949) method shown in Equation 3:4.

Equation 3:4 Stouffer et al. (1949) method for effect size combination.

$$\text{Fisher } Z = \frac{\sum wjZr}{\sum wj}$$

In this formula wj represents the sample sizes of the studies, Zr is the Fisher transformation of the correlation coefficient of effect size. This provided a single

combined effect size for the cohort of studies in the meta-analysis. This, however, yields a fixed effect with typical limitation of generalisability beyond studies within the meta-analysis. In order to assess generalisability to other studies from the same population a one-sample *t* test was also used as a random effects test of significance using Equation 3:5:

Equation 3:5 One-sample *t* test.

$$t = \frac{mean\ Zr}{\sqrt{\frac{Zr^2}{k}}}$$

The normal standardised z values associated with the d effect size were next computed using Equation 3:6:

Equation 3:6 Calculation of normal standardised *z* values.

$$z = \frac{d\sqrt{N}}{\sqrt{d^2 + \frac{1}{p \times q}}}$$

A combined probability statistic from the standardized z values was then calculated using Equation 3:7:

Equation 3:7 Calculation of combined probability statistic.

$$\text{Combined } z = \frac{\sum(Njzj)}{\sqrt{\sum(Nj)^2}}$$

In this formula N = number of participants in study j, (j = 1-9), z = normal standardized deviate for study j.

The reported probability levels for each study were converted to normal standardized z values and combined to produce a summarised reported probability statistic for comparison. A z value of 0 was assigned where studies reported non-significance (Rosenthal, 1991). The heterogeneity of effects within the meta-analysis was next assessed. The weighted mean of the associated Zr value was first computed using the method shown in Equation 3:8:

Equation 3:8 Assessment of heterogeneity of effect sizes across studies

$$\text{weighted mean } Zr = \frac{\sum\left((Nj-3)r_{effect\ j}\right)}{\sum Nj-3}$$

In this formula N = number of participants in study j, (j = 1-9), r_{effect} = effect size for study j.

Heterogeneity of probability was next computed using Equation 3:9:

Equation 3:9 Assessment of heterogeneity of probability across studies

$$\chi^2(k-1) = \sum\left((Nj-3)\left(r_{effect\ j}{}^2\right)\right)$$

In this formula k = 9, N = number of participants in study j, (j = 1-9), r_{effect} = effect size for study j.

Finally, the strength of the association between the identified predictor variables and the obtained effect sizes was approximated using equation 3:10:

Equation 3:10 Contrast analysis

$$z = \frac{\sum \lambda j FisherZ}{\sqrt{\sum \frac{\left(\lambda j^2\right)}{(Nj-3)}}}$$

In this formula λ = weight ascribed to study j, (j = 1-9), N = number of participants in study j.

3.3 Results

3.3.1 Study characteristics

Table 3:1 shows a summary of the characteristics of the studies included in the meta-analysis. There were 491 participants in total, calculated on the basis of the mean sample size for the Sheffield and Salzburg Research Groups. 48% were classified with dyslexia. Not all studies provided sufficient information for the calculation of gender ratios or to make predictions about the influence of education, volunteer status, handedness or recruitment source. Nicolson and Fawcett (1997) was a validation study for the Dyslexia Adult Screening Test (DAST) (Fawcett and Nicolson, 1998). The others in the set were empirical studies.

3.3.2 Effect sizes and significance levels

Table 3:2 summarises the d, r_{effect} and Zr effect sizes and z and p significance levels for each independent study. The d effect sizes for balance ranged from -.07 (Ramus et al., 2003) to 1.42 (Moe-Nilssen et al., 2003) with only one negative effect, indicating that eight out of nine studies found that participants with dyslexia performed worse than their control peers on tests of balance. The overall combined effect size for balance was d =.64 (*CI* .44 - .78), which is equivalent to a correlation coefficient r_{effect} = .31 and a Fisher Z =.32, a medium effect (less than .8, greater than .3) (Cohen, 1988). The combined, fixed-effect probability statistic for the cohort of studies was significant ($z = 4.81$, $p < 0.001$). This is a highly significant result.

Table 3:1 Descriptive statistics for the studies included in the meta-analysis

Authors	Class.[a] of dyslexia	N	% N dyslexia	Age months		FSIQ		%[b] reading deviance	No. *d* effect sizes[c]	*d* effects[d] % *N*-ADHD	*d* effects[e] % dual task	*d* effects[f] no vision
				dyslexia	control	dyslexia	control					
1. Brown et al. (1985)	A	38	42	131.9	135.8	104.7	113.7	49.6	8	100	100	50
2. *Sheffield Research Group*												
Fawcett & Nicolson (1992)	B	44	53	155.0	152.5	107.6	105.7	17.6	12	0	67	0
Nicolson & Fawcett (1995)	B	63	50	144.3	147.3	109.9	111.2	24.3	5	0	20	40
Fawcett, Nicolson & Dean (1996)	C	55	53	173.7	176.0	108.4	110.3	25.2	6	100	0	100
Fawcett & Nicolson (1999)	C	116	47	144.5	141.3	107.5	124.2	39.9	4	0	0	100
Fawcett, Nicolson & MacLagan (2001)	C	27	26	99.36	101.76	103.3	119.3	41.6	1	0	0	100
3. Nicolson & Fawcett (1997)	D	165	9	students		-	-	91.5[g]	1	0	0	100
4. Ramus et al. (2003)	E	32	50	253.0	261.0	122.7	124.8	9.0	1	100	25	25
5. Ramus, Pidgeon and Frith (2003)	E	42	48	117.0	116.0	103.6	102.0	22.7	1	0	0	100
6. Yap and van der Leij (1994)	F	28	50	121.0	125.0	-	-	74.6	4	0[h]	50	0
7. van Daal and van der Leij (1999)	F	75	45	150.0	150.0	113.7	109.8	26.6	8	25[h]	38	25
8. *Salzburg Research Group*												
Wimmer, Mayringer & Landerl (1998)	G	47	43	100.0	99.0	105.7	109.7	69.6	3	100	33	33
Wimmer, Mayringer & Raberger (1999)	G	60	48	109.3	109.3	109.0	-	-	6	50	33	33
Raberger & Wimmer (2003)	G	30	50	124.2	123.3	109.1	109.3	55.3	4	50	50	0

Note. Dashes indicate information not reported.

a Classification of dyslexia: A. Myklebust (1968); B. IQ 90+ reading 18m below age; C. WISC-WORD discrepancy (Wechsler, 1991; 1993); D. Adult Dyslexia Index (Fawcett & Nicolson, 1993); E. Educational Psychologist; F. Dutch school system; G. Salzburg Reading Test (Landerl, Wimmer & Moser, 1997); H. Reading 1.5 SD below age norm.

bDifference between dyslexia and control groups expressed as a percentage of control group average score.

cEffect sizes calculated using the pooled variance across all tested participants.

dPercentage of effects drawn from samples screened for ADHD.

ePercentage of effects obtained from dual-task paradigms.

fPercentage of effects obtained from no vision paradigms.

gDAST One Minute Reading Scores (Fawcett & Nicolson, 1998).

hWeighting applied. Study 7 authors report that study 6 dyslexia group likely included more participants with potential ADHD than study 7.

A one-sample t-test (t [8] = 3.30; $p < 0.001$) indicated that the effect was generalizable to the population beyond our meta-analysis. The critical number of "file drawer" studies required to bring the combined probability level for nine studies below significance is 55. The number of future or hidden studies required to reduce the findings of our meta-analysis to non-significance is 684.

Table 3:2 Effect sizes and significance levels for the set of studies.

Authors	*N*	Effect sizes[a]	*d*	r_{effect}	*Zr*	Probability statistic *z*	*p*
Brown et al. (1985)	38	8	0.05	0.03	0.03	0.16	0.436
Sheffield Group (1992-2001)	30[b]	28	1.17	0.57	0.65	2.77	0.003
Nicolson & Fawcett (1997)	165	1	0.92	0.42	0.44	3.26	0.001
Ramus et al. (2003)	32	1	-0.07	-0.04	-0.04	-0.20	0.421
Ramus, Pidgeon & Frith (2003)	42	1	0.83	0.38	0.40	2.49	0.006
Yap & van der Leij (1994)	28	4	0.75	0.35	0.36	1.85	0.032
van Daal & van der Leij (1999)	75	8	0.13	0.06	0.06	0.54	0.295
Salzburg Group (1998-2003)	41[b]	13	0.18	0.09	0.09	0.56	0.288
Moe-Nilssen et al. (2003)	40	6	1.42	0.58	0.66	3.65	0.000

[a] Number of effect sizes from which the mean *Zr* was obtained.
[b] Mean sample size per effect size.

3.3.3 Heterogeneity

A test of homogeneity revealed significant variability among the *d* effect sizes of the set of studies (χ^2 [8] = 23.7; $p < 0.005$). Four studies yielded *z* scores greater than 1.96 falling within the top 2.5% of the normal distribution for probability. Removal of the two largest effect sizes (Sheffield Research Group 1992 to 2001; Moe-Nilssen et al., 2003) rendered the remaining effects homogeneous. Neither removal of the two smallest (Brown et al., 1985; Ramus et al., 2003) nor the largest and smallest effect sizes (Moe-Nilssen et al., 2003; Ramus et al., 2003) influenced heterogeneity.

3.3.4 Strength of association between effect size and moderator variables

Table 3:3 shows the measures of association and contrasts between potential moderator variables and *d* effect size.

Table 3:3 Potential moderator variables of *d* effect size on measures of balance.

	measures of association			contrasts	
Moderator variable	k^a	r	r_s	z	p
Age	9	-0.19	-0.15	0.74	0.229
FSIQ RD group	7[b]	-0.76*	-0.71	2.42	0.008
% reading score deviance[c]	8[b]	0.27	0.41	0.57	0.284
% effects *N*-ADHD	9	-0.79**	-0.80**	3.46	0.000
% effects dual task	9	-0.28	-0.24	0.99	0.161
% effects no vision	9	0.14	0.15	0.91	0.184

[a] Number of studies reporting statistic
[b] Missing values excluded pairwise
[c] See Table 1 legend
* $p<0.05$
** $p<0.01$

3.3.4.1 Co-morbidity

Table 3:3 illustrates the association between *d* effect size and continuous hypothesised moderator variables. Proportion of effect sizes derived from samples known to be ADHD exclusive was a significant predictor of balance effect size (z = 3.46; $p < 0.001$). There was also a strong significant negative association between this moderator variable and *d* effect size (r_s = -.80; $p < 0.01$). The studies which pre-screened and excluded participants with ADHD symptoms tended to produce the smallest effect sizes.

3.3.4.2 Chronological age

Chronological age did not predict balance effect size (z = .74; p = 0.23). The association between *d* effect size and age of the sample was negligible (r_s = -.15; p = 0.70).

3.3.4.3 Classification of dyslexia

The method of classifying participants for inclusion within the dyslexia group varied among studies (as shown in Table 1) but this categorical moderator variable did not predict effect size (z = .55; $p = 0.29$).

3.3.4.4 Psychometric intelligence

Dyslexia group FSIQ was also a significant predictor of balance effect size ($z = 2.42$; $p < 0.01$). There were moderate negative associations between *d* effect size and dyslexia group FSIQ scores ($r = -.76$; $p < 0.05$; $r_s = -.71$; $p = 0.06$) that were not consistently significant. However, dyslexia group FSIQ scores were not reported by Nicolson and Fawcett (1997) and Yap and van der Leij (1994), therefore a type-II error may have resulted from the truncated sample size in this analysis. When proportion of effects from samples known to be ADHD exclusive was controlled for a partial correlation between FSIQ dyslexia group and balance effect size showed a small change ($pr = -.59$, $p = 0.12$). Controlling for the percentage of effects drawn from samples known to have excluded participants with ADHD did not account for the shared variance between FSIQ and *d*.

3.3.4.5 Reading Ability

The difference between the dyslexia and control group reading scores expressed as a proportion of the control group score did not predict balance effect size ($z = .57$; $p = 0.28$); the association between reading score deviance and *d* effect size was moderate ($r = -.27$; $p = 0.55$; $r_s = .41$; $p = 0.32$).

3.3.4.6 Balance task paradigm

The proportion of effect sizes yielded by dual task paradigms did not predict balance effect size ($z = .99$; $p = 0.16$) the association between paradigm and effect size was weak ($r = -.28$; $p = 0.48$; $r_s = -.24$; $p = 0.53$).

3.3.4.7 Vision Paradigm

The proportion of effect sizes yielded by eyes closed or blindfolded paradigms did not predict balance effect size (z = .91; p = 0.18). There was a weak association between vision paradigm and balance effect size (r = .14; p = 0.70; r_s = .15; p = 0.70).

3.3.4.8 Level of data yielded

Five studies yielded effects from data accrued from observational ratings, four studies yielded effects obtained from continuous data. The categorical moderator variable level of data accumulated did not predict balance effect size (z = 1.05; p = 0.15).

3.3.5 Reading age comparisons

Three studies (Sheffield Research Group [1992-2001]; Yap and van der Leij, 1994; van Daal and van der Leij, 1999) provided sufficient information for the calculation of effect sizes for dyslexia group comparisons with reading age control participants. The overall combined effect size was d = .36, which is equivalent to a correlation coefficient r_{effect} = .18 and a Fisher Z = .18, a small to moderate effect (Cohen, 1988). The fixed effect of balance is smaller for reading age than chronological age comparisons. A one-sample t-test (t [2] = 2.38; p = 0.1) however, indicated that the effect was not generalisable to the population beyond the meta-analysis.

3.4 Discussion

The purpose of this meta-analysis was to take a theoretical and evidential step forward in the debate surrounding the dyslexia and balance issue by addressing a number of research questions. The first and most important question was whether groups with dyslexia differ from control groups on measures of balance. The results revealed a moderately strong and robust combined effect of balance difficulties in developmental dyslexia (d = .64), that is larger in magnitude than that reported by van Izendoorn and Bus (1994) in a meta-analysis of phonological skills, the core

deficit in dyslexia. At first inspection this suggests that there are substantial balance deficits in dyslexia. However, the set of effect sizes was heterogeneous. This indicates that the standardized mean differences between the experimental and control groups across the set of studies were of inconsistent magnitude, and hence were not likely to have been sampled from the same population. Some individuals with developmental dyslexia differ from normal readers on measures of balance, but not all. The advantage of using meta-analytic techniques enabled progress beyond this point to look for significant contrasts and associations, based upon the initial research questions, to examine why the findings of the set of studies varied.

In the introduction to this review potential subject or stimulus factor moderators of variability among studies were identified. Examination of subject factors revealed that sampling criteria provided the two most important findings. The strongest predictor of balance effect size was whether samples had been screened for ADHD. The smallest effect sizes were yielded by Brown et al. (1985), (d = .05) and Ramus et al. (2003) (d = -.07), that were derived from samples known to be wholly ADHD exclusive. The next smallest effect sizes were yielded by van Daal and van der Leij (1999) (d = .13) and the Salzburg Research Group (d = .18) that included 25% and 65% of effects derived from ADHD exclusive samples respectively. The largest effect sizes were yielded by the Sheffield Research Group (1992-2003) (d = 1.17) and Moe-Nilssen, Helbostad, Talcott and Toennessen (2003) (d = 1.42) that included 24% and 0% of effects from ADHD exclusive samples respectively. This suggests that, as speculated, some samples may have included participants with threshold or sub-threshold ADHD symptoms. It should be noted however, that the Sheffield Research Group studies included a number of participants who, having already participated in previous studies, passed assessment for ADHD on DSM-IV in 1996. However, precisely how many of these participated in each of the previous studies is not apparent. As a precaution, the analysis was repeated assuming that 60% of the entire Sheffield Research Group sample was known ADHD exclusive. Tests of contrast and association remained significant ($z = 2.85$; $p < 0.01$; $r_s = -.72$; $p < 0.05$).

Only Ramus et al. (2003) specifically screened for and excluded cases of DCD. Ramus, Pidgeon and Frith (2003) included one participant with co-existing DCD and two with both DCD and ADHD. This study yielded a *d* effect size of .83, the fourth largest and above the average for the set of studies. Given the high rates of co-morbidity of dyslexia with ADHD, ADHD with DCD and the potential for sub-threshold symptoms of motor problems in the ADHD population (Kadesjö and Gillberg, 2001; Piek, Pitcher and Hay, 1999; Kaplan et al., 1998; 2001) there may well have been participants with co-existing DCD included in some samples within the set of studies. This assumption is speculative but not without foundation given the rates of co-morbidity among developmental disorders. One firm conclusion has been established by this meta-analysis: although recent studies have taken ADHD profiles into consideration, the state of play today with regard to assessment of motor profiles in dyslexia research has barely progressed since Denckla et al. (1985). This paper concluded with the recommendation that future studies of dyslexia should include motor control and attention profiles of their samples (see section1.3.1).

The second strongest predictor of balance effect size was FSIQ scores of the dyslexia samples. However, not all studies reported FSIQ scores for experimental or control groups. Moe-Nilssen et al. (2003) recorded the lowest dyslexia group mean FSIQ score (90.1) and the largest effect size (1.42); Ramus et al. (2003) recorded the highest dyslexia group mean FSIQ score (122.7) and the smallest effect size (-.07). One of the main difficulties identified within the classification of dyslexia participants among studies was standardization of FSIQ. It is possible that at least Moe-Nilssen et al. (2003) included participants with more general than specific learning difficulties. The association between general learning difficulties, academic achievement and motor control difficulties is well established (e.g. Sugden and Wann, 1987). Perceptual-motor difficulties, which impact upon balance skills, have also been reported for DCD samples with low IQ (e.g., Jongmans, Smits-Engelman, & Schoemaker, 2003). Two studies (Van Daal and van der Leij, 1999; Fawcett, Nicolson and MacLagan, 2001) compared 'garden variety' poor readers with discrepant poor readers but with conflicting results. However as the methodologies used were inconsistent it is difficult to draw firm conclusions from this finding. The comparisons between 'garden variety' readers and either control or dyslexia groups,

reported by these studies, were not included in the meta-analysis. Therefore, known poor readers with lowered IQ were eliminated.

Other subject factors were less evident moderators of effect size. Age was weakly and not significantly associated with balance effect size across the set of studies, which suggests that balance difficulties do persist into adulthood. Although Nicolson and Fawcett (1997) and Ramus et al. (2003) were the only adult studies, the Sheffield Research Group studies included some 18 year old participants. If balance difficulties, when present, were due to developmental delay, effect sizes would be expected to decrease with increasing age. This is interesting because previously balance and motor skills deficits had been argued to be attributable either to developmental delay (e.g., Denckla, 1985) or to developmental deviance (e.g., Nicolson and Fawcett, 1995). These findings are more indicative of developmental deviance because effect sizes did not decrease with increase in age of the sample. Alternatively, the small association may arise from inconsistent results across studies. Furthermore, longitudinal studies have found that there are two possible long term outcomes for balance and motor skills in DCD (Losse et al., 1991; Cantell, Smyth and Ahonen, 2001). Children who presented the most severe perceptual motor deficits at 10 years tended to demonstrate persistent deficits at 17 years, whereas those who presented less severe deficits appeared to have resolved their difficulties by adolescence. This evidence adds further weight to the argument that co-morbidity with DCD may well have influenced the results of some studies in the set.

Three studies provided sufficient information for comparisons between children with dyslexia and reading age controls. This analysis yielded a smaller combined effect size, suggesting that balance improves with age for control groups but not dyslexia groups. However, more reading age comparisons are required between young control typical readers and older children with dyslexia at a comparable level of reading ability before these findings can to be generalized to the dyslexia population at large. Additional adult studies may also be needed, although longitudinal studies, of the kind being undertaken currently in Finland (e.g., Lyytinen et al., 2004), would

provide better substantiated evidence for the persistence or resolution of balance deficits in dyslexia and associated developmental disorders.

The method of classification of dyslexia varied among studies but did not significantly predict balance effect size. Studies differed on whether their classification was based upon their own independent assessment of FSIQ and reading ability (Brown et al., 1985; Sheffield Research Group [1992-2001]), dyslexia screening tests (Nicolson and Fawcett, 1997) or whether they relied upon diagnosis by an Educational Psychologist (Ramus et al., 2003; Ramus, Pidgeon and Frith, 2003), diagnosis through the local school system (Yap and van der Leij, 1994; van Daal and van der Leij, 1999) or teacher recommendation supported by independent reading assessment (Salzburg Research Group [1998-2003]; Moe-Nilssen et al., 2003). Within the Sheffield Research Group studies, two used the traditional cut-off method (e.g. FSIQ at least 90 with reading 18 months below chronological age matched controls), whereas more recent studies used the regression method, whereby reading age is 1.5 SD below that predicted by FSIQ (Nicolson, 2001).

The diversity in the classification of dyslexia among studies illustrates the confusion surrounding the formal diagnosis of dyslexia that research needs to address. One means of clarifying this situation would be for future studies to routinely and independently assess experimental and control groups on linked, standardised assessments of cognitive and literacy skills such as the Wechsler Intelligence Scales for Children (WISC-IV) (Wechsler, 2004) and the Wechsler Individual Achievement Test (Wechsler, 2005). These would not only provide uniform and transparent criteria for classifying dyslexia but also provide a standardized basis for the regression method of classification. Although the validity of defining dyslexia based on such an approach compared to that based simply on reading achievement has been questioned (e.g., Vellutino et al., 2004), the meta-analysis has highlighted the potential importance of distinguishing between these methods for research. Between groups reading difference contrast was non-significant, however the non-parametric measure of association between reading score deviance and balance effect size was moderate (r_s =.41; ns). Disparity among reading tests across studies made direct

comparison of reading scores difficult; also, not all studies reported reading scores for both groups. These findings indicate that not only the method of diagnosis and classification of dyslexia but also the method of reading assessment are important areas for consideration in future research.

Examination of the moderator variables hypothesised from stimulus factors proved interesting. The proportion of effect sizes yielded by dual task paradigms contrast was non-significant and its relationship to balance effect size negative and weak. This suggests that studies with the highest percentage of dual tasks tended to generate the smaller effects. This was unexpected because several studies reported that larger effect sizes tended to result from dual task paradigms (e.g., Fawcett and Nicolson, 1992; Nicolson and Fawcett, 1995; Yap and van der Leij, 1994). One of the main arguments of the cerebellar deficit hypothesis for balance deficits in dyslexia is that inability to automate postural responses requires conscious attention to be allocated towards maintaining postural stability in a process of "conscious compensation" (Nicolson & Fawcett, 1990, p.3). Automaticity is generally tested by introducing a concurrent task designed to induce stress in the postural control system causing automaticity to break down. The task causes attention to be drawn away from conscious compensation. Consequently, balance is threatened and postural sway is increased. It has been argued that the effects of secondary tasks on postural sway diminish when samples have been screened for ADHD (e.g., van Daal and van der Leij, 1999; Raberger and Wimmer, 2003; Ramus et al., 2003). The findings of the meta-analysis indicated that dual task paradigms had no greater effect on balance than single task paradigms across studies. This apparently contradictory finding again may arise as a direct result of the inclusion of cases of comorbid ADHD in some samples. It may also be due to a lack of uniformity among the primary and secondary tasks employed across the set of studies.

Table 3:4 shows the wide array of task paradigms and measurements. Balance depends upon the ability to withstand fixed (e.g., gravity) or variable (e.g., a moving platform) forces. These can either be self-imposed, as when standing on one leg, or due to an external perturbation such as receiving a gentle push in the back. Balance

ability can be modulated by sensory manipulation, as when wearing a blindfold or standing on a compliant surface or by performing a secondary attention demanding task while balancing. Single tasks ranged from standing in a tandem Romberg toe-to-heel position with eyes open or closed (e.g., Brown et al., 1985), standing eyes open on a compliant surface (e.g., Moe-Nilsen et al., 2003), to standing on one leg on an elevated balance beam (e.g., Wimmer, Mayringer and Raberger, 1999) or having a force applied to the back when standing on two feet whilst wearing a blindfold (e.g., Nicolson and Fawcett, 1997). Dual tasks ranged from overt backwards counting to pressing a button in a choice reaction task while simultaneously performing a balance task (e.g., Fawcett and Nicolson, 1992; van Daal and van der Leij, 1999). However, the way in which the postural control system maps sensory cues to orientation in the environment to the muscle activity required to maintain balance is not yet fully understood (Balasubramaniam and Wing, 2002; Shumway-Cook & Woollacott, 2001). Different methods of assessing balance control may involve different neural networks and therefore may not be directly comparable (Shumway-Cook and Woollacott, 2001; Winter, 2004).

Table 3:4 Balance tasks, secondary tasks and measurement by study.

	Authors	Primary task	Secondary task	Measurement
1	Brown et al. (1985)	Tandem Romberg EO, EC	none	Strain gauge force transducer
2	*Sheffield Research Group*		a.counting backwards	observational rating: hands feet wobble 10-20°
	Fawcett & Nicolson (1992)	beam balance:2 feet tandem; 1 foot	b. selective choice	
	Nicolson & Fawcett (1995)	beam balance	a.counting backwards	observational rating: 0.5 = minor wobble
		1 foot & 2 foot EO & BF	b. selective choice	1.0 = major wobble; 2.0 = stepping/overbalance
	Fawcett, Nicolson & Dean (1996)	push in back with index finger 2 kg	none	observational rating
				sway rating
	Fawcett & Nicolson (1999)	push in back with index finger 2 kg	none	observational rating: 0 = good
				1 = small sway; 2 = stepping/overbalance
	Fawcett, Nicolson & MacLagan (2001)	push in back with index finger 2 kg	none	observational rating: 0 = good
				1 = small sway; 2 = stepping/overbalance
3	Fawcett & Nicolson (1997)	DAST postural stability component	none	observational rating:
				0 = rock solid to 6 = many steps/fall
4	Ramus et al. (2003)	Feet together EO,EC	counting backwards	Length of path COP, C7 diode normalised to a
		Feet together arms raised EC	feet together arms raised EC	single z score for balance across conditions
5	Ramus, Pidgeon and Frith (2003)	DST Postural stability component	none	observational rating
				0-6 (as Fawcett & Nicolson, 1997)
6	Yap & van der Leij (1994)	beam balance: 1 foot	auditory choice	observational rating: 0.5 = minor wobble
				1.0 = major wobble; 2.0 = stepping/overbalance
7	van Daal & van der Leij (1999)	2 board balance		auditory tone "click" as boards touch
		EO + BF dominant ft fwd. ft bhd		latency to "click"
		walking backwards EO	counting bwds	number of steps
8	*Salzburg Research Group*			measured against background: angle<15^0 = 0
	Wimmer, Mayringer & Landerl (1998)	balance beam I foot	semantic judgement	angle<15- 30^0 =1;angle>50^0 , foot touches floor =2
		opaque goggles	'yes/no'	foot touches floor full weight =3
	Wimmer, Mayringer & Raberger 1999)	as before	as before	as before
	Raberger & Wimmer (2001)	as before 2 feet and 1 foot	as before	as before, transparency over video screen
9	Moe-Nilssen et al. (2003)	2 feet firm EO, EC; 0.02m, 0.1m	none	triaxial accelerometry
		pillow; tandem firm EO		
		provocation test EC		

Furthermore, both arousal and articulation are known to increase postural sway in dual task performance, possibly arising from the effects of increased respiration (Maki and McIlroy, 1996; Yardley, Gardner, Leadbetter & Lavie, 1996). The nature of the dual task itself may be a confounding variable, particularly when a child with dyslexia is asked to perform a task that they anticipate finding difficult or stressful, such as backwards counting in the presence of a researcher, even though the task itself has been individually calibrated to standardise the level of difficulty across participants. Notwithstanding these issues, failure of the meta-analysis to distinguish between effect sizes yielded by single or dual task paradigms suggests that the automaticity deficit hypothesis may not fully account for balance deficits in dyslexia.

The percentage of effects generated by paradigms that included eyes closed or blindfolded in the meta-analysis, was also a weak predictor of balance effect size. The cerebellar deficit hypothesis predicts that removal of vision leads to greater reliance upon impaired vestibular/ cerebellar function, hence greater difficulty in maintaining postural stability. Moe-Nilssen et al. (2003) however, found that their group of children with dyslexia performed significantly worse than their peer control group on all measures of balance with eyes open, but not with eyes closed, suggesting that they were less dependent upon visual cues. This appears to conflict with the prediction of the cerebellar deficit hypothesis, which in turn suggests that the role of vision in maintaining postural stability in dyslexia warrants further investigation. Furthermore, Wann, Mon-Williams and Rushton (1998) compared children with DCD with age-matched controls using the moving room paradigm and found that the children with DCD, who had postural control difficulties, demonstrated bias towards using visual information, which was equivalent to that found in younger, nursery-age children. Based on this evidence, inclusion of participants with threshold or sub-threshold DCD symptoms may potentially be a strong moderator of balance effect size in visual modulation paradigms.

Finally, inconsistency among findings may have resulted from the range of measurement techniques used and the levels of data generated. Data range from the number of observed balance errors, for example, a minor wobble or stepping off a

beam (Nicolson and Fawcett, 1995; Yap and van der Leij, 1994) to the estimation of limb angle displacement against an illustrated background or transparency placed over a video screen as shown in Table 3:5 (Wimmer, Mayringer and Landerl, 1998, Raberger and Wimmer, 2003). Where homogeneity exists among the results of studies the data collected are typically ordinal, comprising subjective observation and performance ratings. Few studies have provided continuous data; among these, comparisons of results may be misleading. For example, Moe-Nilssen et al. (2003) reported latency of postural responses following perturbation of balance. This measure may not be equivalent to Ramus et al. (2003) who reported magnitude of postural sway, as defined by motion of the Centre of Pressure, the distribution of reactive forces generated between the soles of the feet and the support surface. Timing and force in movement may be dissociable, which would indicate that different aspects of postural control might also involve different neural networks (Lundy Ekman, Ivry, Keele & Woollacott, 1991). It is also possible that, in the absence of procedures which yield fine-grained continuous data, subtle balance difficulties may pass unobserved. In dyslexia, where compensation may be mandatory in many aspects of daily life, minor difficulties may be masked.

The findings of this meta-analysis have important implications for future research. First, there are issues to be resolved surrounding the classification of dyslexia between research groups. Future research would benefit from the assessment of experimental and control groups on compatible measures of psychometric intelligence and reading ability in order that their allocation to either group is made upon the basis of the regression method of diagnosis. Discrepant and non-discrepant poor readers should be assigned to separate groups for comparison if we are to tease apart the relationship between reading and balance in good and poor readers. Furthermore, all participants should be independently assessed whenever possible for symptoms of attention and motor control deficits. Second, if we are to understand the processes which underlie balance deficits in dyslexia, future studies should attempt to target specific tasks for close replication. Dual tasks should be carefully calibrated to ensure that levels of both cognitive and attentional demand and physiological arousal are individually calibrated and therefore of comparable difficulty for all participants in both experimental and control groups. Third, future

studies would benefit from making use of the range of advanced digital measurement systems available for the study of balance and movement in order to generate fine-grained, continuous data for quantitative motion analysis.

3.5 Conclusions

The findings of this meta-analysis do not suggest that balance is related to reading but more likely through association with ADHD or lowered IQ. At this point it seems unlikely, therefore, that balance measures can specifically predict risk of reading difficulty. However, methodological issues of sample classification, procedure and measurement have been highlighted. This thesis aims to address these issues in order to assess whether balance is an effective predictor of reading difficulties or of developmental disorders in general.

4 General methods and development of a paradigm

4.1 Taking a step forward

The meta-analysis reported in Chapter 3 identified three key issues which needed to be addressed in order to advance our understanding of the relationship between balance deficits and reading difficulties. Hence, prior to assessing the ability of balance measures to predict reading failure, this chapter describes the development of an experimental method designed to bring a more objective approach to the study of balance in dyslexia.

4.1.1 Ethical considerations

The Aston University Senate Ethics Committee granted approval for the project and a risk assessment was sanctioned by the Health and Safety officer for Psychology. All adults gave their informed consent. They were also advised of their rights in accordance with the British Psychological Society (BPS) Code of Conduct, Ethical Guidelines and Principles (2000) and were given an oral and written debriefing after the testing session. Parents of all children were informed of their rights consistent with these tenets and written informed parental consent was obtained. Verbal assent was obtained from children prior to testing and participants were permitted to withdraw at any stage. Principal researchers had previously obtained Criminal Records Bureau Enhanced Disclosure for working with children and vulnerable adults.

4.1.2 Sampling and classification

The meta-analysis revealed a lack of uniformity in the method of diagnosis and classification of dyslexia among previous studies and in the psychometric and literacy measures applied. At least one sample (Moe-Nilssen et al., 2003) may have included 'garden variety' poor readers within their experimental group. Other samples may have included participants with co-existing attention deficits with potentially high prevalence rates of comorbid motor problems. Furthermore, much of

the most compelling evidence for balance deficits in dyslexia was yielded by the Sheffield Research Group (1992-2001) that repeatedly recruited the majority of their participants from the same pool of participants. In order to address the sampling concerns raised by the meta-analysis, following the validation of the paradigm as described in this chapter, participants in the ensuing studies were opportunity-sampled from the dyslexia and normal reading populations and tested either in the field or in the laboratory. Sample characteristics are presented in each experimental chapter. Adults and children were assessed on the following standardised psychometric and literacy measures and also rated for symptoms of inattention and hyperactivity. Scoring of measures is discussed within the experimental chapters.

4.1.2.1 Psychometric measures: adults

The Wechsler Abbreviated Scale of Intelligence (WASI) was administered to all control participants (Wechsler, 1999). This comprises a subscale of verbal ability assessed by the Vocabulary and Similarities subtests and a subscale of non-verbal ability assessed by the Block Design and Matrix Reasoning subtests. This is a short form of the Wechsler Adult Intelligence Scale (WAIS) and can be administered as either 2 or 4 subtests (Wechsler, 1992). Adults received 4 subtests to allow estimation of both verbal and non-verbal ability as well as an estimate of general intellectual ability. In addition, in study 5 (Chapter6) Digit Span, Digit Symbol Coding and Digit Symbol Copy subtests of the WAIS-III were completed. These assess short-term memory, visual motor coordination and processing speed, and perceptual graphomotor speed respectively. Consent was sought and obtained from participants with dyslexia to take standard scores from the report of their diagnostic assessment. Where these were not available or the individual had been assessed before the age of 16 the WASI was administered. Descriptive classifications consistent with Wechsler scales are used throughout this thesis.

4.1.2.2 Literacy measures: adults

The Wide Range Achievement Test (WRAT 3) Reading and Spelling were administered (Wilkinson, 1993). These are single-word tests of literacy achievement. In addition, in study 5 (Chapter6) two components of the DAST were given (Fawcett

and Nicolson, 1998). One Minute Reading is a measure of orthographic speed and accuracy; Nonsense Passage Reading is a measure of grapheme-phoneme conversion ability within a contextual passage as opposed to single nonsense word reading. The Phonological Assessment Battery (PhAB) Spoonerisms subtest was used as a measure of ability to segment single syllables and reassemble segments to form new words (Frederickson, Frith and Reason, 1997). As with the psychometric measures, these items were administered to participants with dyslexia if they had not already been assessed on these measures within their diagnostic assessment.

Two computerised tests of reading component skills, programmed in SuperlabPro, were also administered to all adult participants in study 5 (Chapter 6). The orthographic choice task is a word-pseudohomophone discrimination test of orthographic ability (Olson et al. 1994). Two letter strings are presented side by side on the computer screen. One string forms a real word (e.g., "rain"), and the other is a pseudohomophone of the word (e.g., "rane"). Participants are asked to choose which string is spelled correctly and then press the appropriate button on a button-box as quickly as possible without making errors. The strings remain on the screen until the participant responds. This is followed by visual feedback ('correct' or 'incorrect'). The letter strings are presented in 18 point Geneva font. The task includes a short practice session of 8 items followed by 80 experimental items. This task depends on orthographic skill as phonological processing of the two items yields the same output. The task is scored by calculation of the mean response time and the percentage of correct responses.

The second computer-based task administered, using the same procedure and software, was a phonological choice task (Olson et al. 1994). In this case three letter strings, all nonwords, are presented. The participant is asked to choose the letter string that sounds like a real word, and to respond as quickly and accurately as possible. For example, when 'nite,' 'dite,' 'hote' are presented, 'nite' is the correct choice as it sounds like 'night'. There is a short practice session followed by 80 experimental items. Again, visual feedback ('correct' or 'incorrect') follows each response. This task depends upon phonological ability as the correct word is a

pseudohomophone of a real word. The task is scored by obtaining the mean response time and the percentage of correct responses.

4.1.2.3 Psychometric measures: children

Child participants assessed in the field were given the 2 subtest form of the WASI (Wechsler, 1999). This comprises the Vocabulary and Matrix Reasoning subtests and provides an estimate of general intellectual ability. Children with dyslexia were administered the Matrices subtest of the British Abilities Scales II (BAS-II; Elliott, Smith & McCulloch, 1998) as a measure of their non-verbal reasoning. Their level of general cognitive ability was adjudged from their Chartered Psychologist's diagnostic assessment.

4.1.2.4 Literacy measures: children

Child participants tested in the field were assessed on the Wechsler Objective Reading Dimensions (WORD) Basic Reading, single word reading and Basic Spelling, written spelling of orally presented single words (Wechsler, 1993). They were also administered the PhAB Nonword Reading test to assess ability to apply grapheme to phoneme conversion rules, and the PhAB Naming Speed Test to assess speed of articulation (Frederickson, Frith and Reason, 1993). They also completed the DST One Minute Reading measure of orthographic fluency (Fawcett and Nicolson, 1996). Children with dyslexia were assessed on the BAS-II single word Reading and Spelling tests (Elliott, Smith & McCulloch, 1998).

4.1.2.5 Attention measures

Adults were asked to complete the Barkley Scale of Attention Current Symptoms Scale self-report form (Barkley and Murphy, 1998). Teachers of children assessed in the field completed the Barkley Disruptive Behaviour Scale teacher form and parents of children with dyslexia completed the Barkley Disruptive Behaviour Scale parent form (Barkley and Murphy, 1998). These provided measures of attention along dimensions of inattention and hyperactivity-impulsivity symptoms. The forms differed in their titles only and all questions on each dimension were identical.

Although the dyslexia groups received parental ratings and the control group received class teacher ratings, the age-related clinical cut-off scores are normalized against separate populations and are comparable (Barkley and Murphy, 1998).

4.1.2.6 Motor assessment

A peg moving task based upon Annett (1985) was used to assess fine motor ability in adults. This comprises a small board with two parallel strips of wood, with 10 holes in each. Participants are asked to move the pegs in the holes from the front to the back row as quickly as possible without dropping any of the pegs. The movement is timed from when the participant touches the first peg until the last one is in position. Performance is averaged over 3 trials.

4.1.3 The Dyslexia Screening Tests Postural Stability Component

The meta-analysis also revealed a scarcity of experimentally controlled procedural replications across studies. An array of single or dual task paradigms (see Table 3:4) were used and these varied in level of difficulty not only across studies but potentially between and within groups. The closest approximation of a standardised metric used in the cohort of studies was a manual perturbation tool, calibrated on kitchen scales, that delivers a 2.5 kg, 3kg or 4kg force to children or adults respectively as shown in Figure 4:1 (Fawcett & Nicolson, 1998, 2004a). This was developed from a manual provocation test whereby the administrator would press their index finger onto kitchen scales for calibration and then administer the same approximation of force to the participant's lower back (e.g., Fawcett & Nicolson, 1999; Fawcett, Nicolson & Dean, 1996; Fawcett, Nicolson & MacLagan, 2001). The tool shown in Figure 4:1 is used in the postural stability component of a number of dyslexia screening batteries for children and adults, for example the Dyslexia Adult Screening Test (DAST) (Fawcett & Nicolson, 1998) and the Dyslexia Screening Test-Junior (DST-J) (Fawcett & Nicolson, 2004a).

Figure 4:1 The DST-J and DAST postural stability tool (Fawcett & Nicolson, 1998, 2004a).
The tool is calibrated by tightening the collar nut and pressing down onto a set of kitchen scales until resistance between the collar and shaft is sufficient to deliver a force of 2.5 kg for younger and 3kg for older children and 4 kg for adults.

Figure 4:2 shows the method of application. The administrator places the end of the tool in the small of the participant's back, just above the lumbar-thoracic junction of the spine. The participant is told to expect a gentle push in the back and instructed to try to keep as still as possible. The collar is pushed towards the participant and the application force is maintained for 1.5 seconds before the tool is removed. The perturbation of balance follows release of the oppositional force generated as the participant pushes against the tool to counter the threat to balance.

Figure 4:2 Application of the DST-J and DAST postural stability component (taken from Fawcett & Nicolson, 2004a).
Application instructions as they appear in the DST-J/ DAST manuals (Fawcett & Nicolson, 2004a, 1998).

The resultant motion of the trunk, heels and feet during recovery is observed and rated from 0 for 'rock solid' performance to 6 for several steps forward (Fawcett and Nicolson, 1998; p.12). The tool is applied four times, twice with the participant's arms resting at the sides, twice with arms raised in parallel in front of the body. The participant wears a blindfold throughout.

Using this test, Nicolson and Fawcett (1997) found that 63% of adults with dyslexia ($N = 15$) compared to 8% of a peer-control group ($N = 150$) displayed an 'at risk' of dyslexia performance. The postural stability component was designed to be an objective measure of balance function (Fawcett and Nicolson, 1999). However, although reported inter-rater reliability of postural sway ratings on the 0 to 6 point scale is high, (between 0.94 and 0.98) (Fawcett and Nicolson, 1999), assessment still depends upon subjective clinical judgement. Also, the force applied is uniform (2.5 - 3kg for children, 4kg for adults) irrespective of individual body mass. In the field of biomechanics the anthropometric measure of mass is an indication of inertia, or the ability to withstand externally generated forces (Winter, 2004). Furthermore, both the ordinal scale used and the potential for floor effects in control subjects threaten the validity of the measure, at least in experimental studies (Moe-Nilssen et al., 2003).

4.1.4 Measurement and level of data

Although three of the set of studies of children and adults employed motion analysis techniques that yielded continuous data (e.g., Brown et al., 1985; Ramus et al. 2003) only Moe-Nilssen et al. (2003) have attempted a precise replication of the postural stability component tool. They applied objective posturography by means of a provocation test, in replication of the DST-J/DAST postural stability component, using an individually calibrated provocation force equal to 5% body mass. Their replication did not discriminate between groups however, possibly because their outcome measure was response latency following perturbation using accelerometry, a departure from the DST-J/DAST metric that rates recovery from perturbation according to the amount of trunk sway, heel raising or stepping. In order to address the procedural concerns raised by the meta-analysis, the paradigm described in this chapter uses a modification of the Moe-Nilssen provocation rig to deliver an

individually calibrated provocation test. To provide an accurate measurement of motion following provocation of postural stability, henceforth postural sway, the method described in this chapter employs digital technology to record postural sway by means of a 3-dimensional optical infrared emitting motion capture system.

The fundamental hardware components of the Qualisys Motion Capture System (Qualisys Systems, Sweden) used are three 120 Hz ProReflex™ Motion Capture Units (MCU120) or cameras. Each camera, shown in Figure 4:3, emits a beam of infrared light and calculates a 2-dimensional position of a reflective target in the measurement volume with high spatial resolution (accurate to 0.03mm).

Figure 4:3 Qualisys Systems ProReflex™ Motion Capture MCU 120 camera.

Reflective targets are lightweight, passive, semi-spherical, retro-reflective markers that can be placed anywhere within a calibrated measurement volume (see Figure 4:4). Figure 4:5 shows a graphic representation of the calibration kit with 19mm markers. During calibration a standard wand is systematically moved around within the measurement volume in which a stationary L-shaped reference object is placed to determine the co-ordinate system for motion capture. The stationary reference object incorporates 4 retro-reflective markers placed at measured locations defined by the system manufacturer. The calibration wand incorporates 2 markers located a fixed distance apart; in this case a 750mm wand was used. All settings for the calibration

process are controlled by Qualisys Track Manager (QTM), Windows-based motion capture software.

Figure 4:4 Graphic representation of a passive retroreflective marker (taken from Qualisys Systems, 2005).
The picture indicates the direction of pulses of infrared light. One flat surface of the marker is for application to skin or clothing with double-sided adhesive tape.

Figure 4:5 Graphic representation of a calibration wand and L-shaped reference object (taken from Qualisys Systems, 2005).
The wand is moved both linearly and rotationally within the measurement volume.

Markers are applied to the body of interest using double sided adhesive tape placed above anatomical landmarks, either onto tightly fitting clothing or directly onto the skin. During motion capture, the cameras emit infrared light pulses that reflect from the markers back to the camera, where their 2D position is recorded at 120 Hz. All camera views are shown on the monitor in real-time and positional change of the markers during recording can be tracked in 2D to ensure that the performance recorded is that which was intended. Multiple processes within the cameras allow the data to be processed immediately and converted to co-ordinates. The resulting digital

data is downloaded to the QTM software (see Figure 4:6). Recording with three cameras allows QTM to combine the 2D data from each camera and calculate 3D positional data by means of an advanced algorithm. 2D and 3D data can then be exported into several formats. In these studies all data were exported into Tab Separated Values (TSV) format for analysis in Microsoft Excel.

Figure 4:6 Graphic representation of 2D to 3D data processing (taken from Qualisys Systems, 2005).
Each camera has one data input and one data output connection. Camera 1 downloads data from all 3 cameras to QTM.

To address the measurement issues revealed by the meta-analysis, the following studies were conducted using a modification of the Moe-Nilssen et al. (2003) provocation paradigm, motion capture and quantitative motion analysis techniques.

4.2 General method

4.2.1 Construction of a provocation rig

A rig was constructed as shown in Figure 4:7 that consisted of a wall mounted shelving bracket, a height adjustable pulley and a weight suspended from a horizontal dragline attached to the participant's waist. This was an adaptation of the Moe-Nilssen et al. (2003) design that had been used with children. The main difference between the two rig designs is that the weight was suspended in front of the participant in the Moe-Nilssen study. In this paradigm the weight was suspended behind. This was in order to include an 'eyes open' condition to assess the effects of

modulating vision on balance and participants would have been able to see and therefore anticipate the release of the weight. The intention was also to ensure that the cameras' view of the measurement volume was not obstructed and that markers were not obscured in consequence. The pulley was adjustable so that the dragline would always be attached to the same location within body segment S4 (trunk area 2 between hips and lower ribs) for each participant in keeping with the administration of the DST-J/DAST tool. The oppositional force generated by the participant in resistance of the suspended weight in our paradigm is in the anterior direction, whereas the DST-J/DAST induces opposition in the posterior direction. However, as both tests generate reactive forces on the same cardinal axis in the sagittal plane it was assumed that postural sway would be comparable.

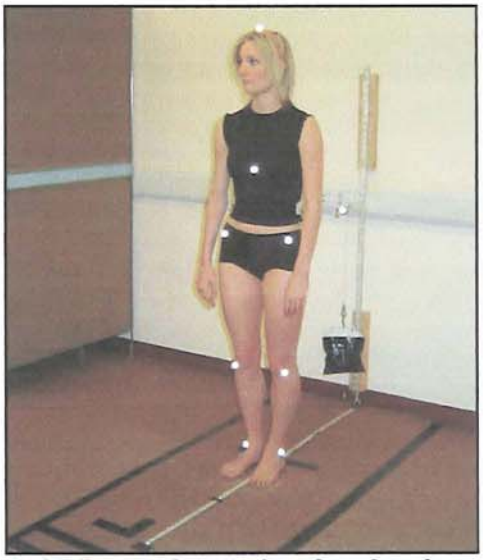

Figure 4:7 Provocation test rig and marker placement.
A model standing within the measurement volume. The weight is suspended from a dragline via a pulley and attached to a belt.

4.2.2 General procedure

Participants wore tight fitting shorts, a Tee shirt and were barefoot. Their height and mass were recorded and a weight equivalent to 5% mass was assembled. 19mm reflective markers were attached to the skin or clothing above the lateral malleolus of the ankle joints, the head of the tibiae, the iliac crests, the xiphoid process and the

crown. A belt was secured around the participant's waist to which the dragline was attached. The height of the pulley was adjusted to ensure that the dragline was horizontal. Participants stood with feet one centimetre apart and the position of the feet was marked to ensure return to the same start position before each trial.

4.2.3 Motion capture

Three tripod mounted MCU120 cameras were placed as shown in Figure 4:8. Figure 4:9 shows the monitor display of the 2D view for each of the three cameras.

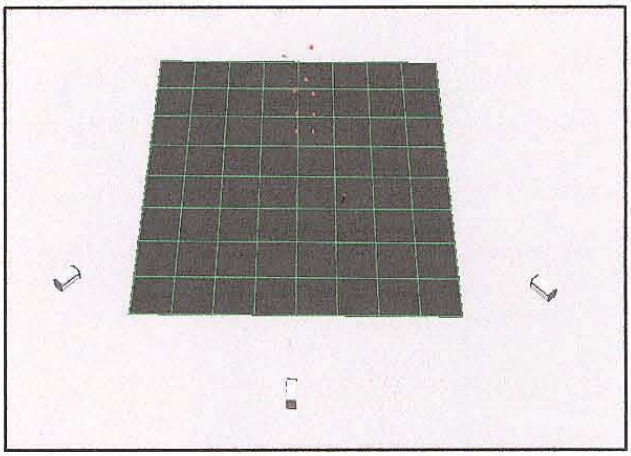

Figure 4:8 Motion capture view of camera placement and measurement volume.
Screen print shows the monitor display. The participant is depicted by the red dots that show the marker placement shown in Figure 4:7.

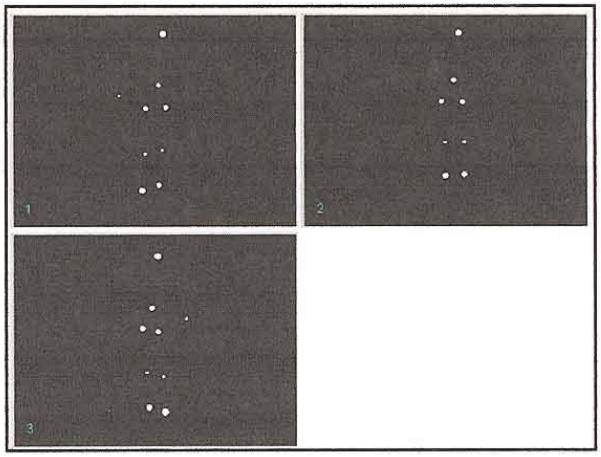

Figure 4:9 Monitor display of camera 2D view of markers within the measurement volume.
Screen print shows the monitor display of the 2D view from cameras 1, 2 and 3.

Motion capture commenced following an auditory cue and the weight was released after a pre-determined interval (6-12 s). Motion of the markers was recorded at 120 Hz for 15 seconds. Figure 4:10 shows the monitor display of the combined 3D view of the measurement volume following download of the data to QTM.

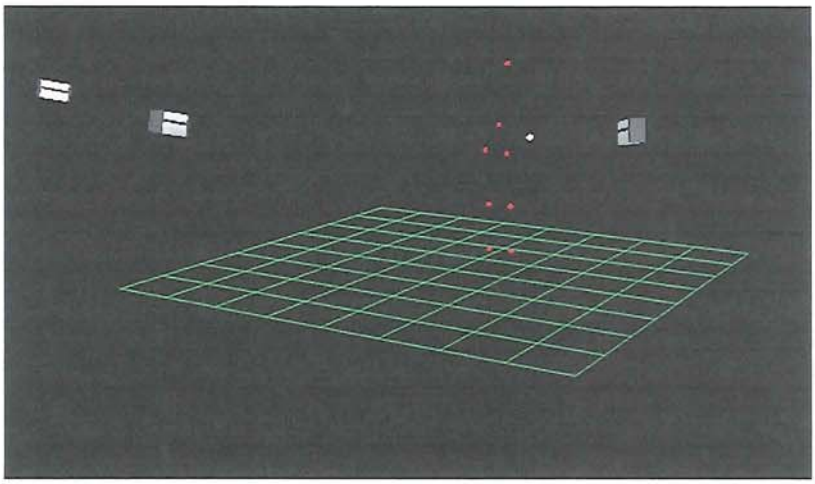

Figure 4:10 Motion capture 3D view of provocation test.
Screen print shows the monitor display of the combined 3D view from 3 cameras. The participant is depicted by the red dots that show the marker placement. The white dot is the marker on the suspended weight.

4.2.4 Motion analysis

A 2s (240 frame) window of data was selected, at a point pre-determined according to the particular study, and exported into TSV format for processing in Microsoft Excel. 3D resultant postural sway was calculated from the position of the markers on each cardinal axis for each frame using the following formula:

Equation 4:1 Calculation of 3D resultant displacement.

$$d = \sqrt{(((x2-x1)(x2-x1))+((y2-y1)(y2-y1))+((z2-z1)(z2-z1)))}$$

Mean point by point resultant displacement over 5 trials was calculated for each marker. The mean sway path of individual markers was then averaged to provide an estimate of total body 3D sway path.

4.3 Pilot studies

Three pilot studies were conducted to assess reliability and validity of the method.

4.3.1 Study 1: Assessment of reliability of the method

The first pilot study assessed reliability of the provocation test and the motion capture data.

4.3.1.1 Participants

Six members of staff and postgraduate students mean age 29 years, range 24 to 47 years, from the Neurosciences Research Institute participated. All were healthy individuals and normal readers.

4.3.1.2 Procedure

Postural stability was tested by provocation in two conditions. First, participants were asked to focus their vision on the illuminated number displayed on camera 2. This was immediately ahead at a distance of 2.5m. Second, participants wore a lightweight opaque visor that removed visual cues to orientation although eyes remained open throughout. This was an industrial safety visor designed to protect the face that was covered with opaque polythene sheeting. The visor was designed to imitate the visual surround oriental lampshade used in Horak, Nashner and Diener (1990) (further described in Chapter 6). There were 5 trials of each condition (10 in total).

4.3.1.3 Results

Multivariate correlations were performed between 240 3D data samples over 5 trials for each participant. Pearson's Correlation Co-efficient range ($r = .85$; $p < 0.05$ to $r = .99$; $p < 0.01$).

4.3.1.4 Discussion

The paradigm is a reliable measure of postural sway in this sample of adults with normal reading.

4.3.2 Study 2: Assessment of task requirements

The second pilot study was intended to assess whether the choice of task influenced postural sway. The first task manipulated visual cues to orientation. The second task perturbed balance by means of the provocation test previously described. The third task assessed the effects of maintaining an external focus of attention on postural sway. Wulf, McNevin and Shea (2001) and McNevin and Wulf (2002) suggest that motor performance is generally enhanced when the individual focuses on the outcome of a task (external focus) rather than the movement itself (internal focus). During initial skill learning, attentional focus is internal and performance is impaired. This theory of 'constrained action' suggests that an internal focus of attention exercises conscious control over automatic postural control. However, the cerebellar deficit hypothesis predicts that imposing an external focus of attention draws attention away from monitoring postural control and impairs balance. Therefore, maintaining an external focus of attention would be predicted to enhance postural stability in control groups but to exacerbate postural instability in participants with dyslexia.

4.3.2.1 Participants

Eleven Psychology undergraduates volunteered and each received one hour credit of research participation. All were healthy individuals and normal readers, mean age 20 years, range 18 to 24 years.

4.3.2.2 Procedure

There were six conditions in this study.

Task 1:

The first two conditions assessed the effect of manipulation of vision on postural sway. In condition 1, participants were asked to maintain visual focus on camera 2; in condition 2 participants wore the visor used in study 1 (Horak, Nashner, & Diener, 1990). The weight was suspended and then released in both conditions and there were 5 trials of each condition.

Task 2:

The next two conditions assessed the effect of maintaining an external focus of attention on postural sway. In condition 3 participants were instructed to keep the weight behind them as motionless as possible (external focus); in condition 4 participants were instructed to keep themselves as motionless as possible (internal focus). The weight was suspended and then released in both conditions and there were 5 trials of each condition. Participants were instructed to maintain visual focus on camera 2 in both conditions.

Task 3:

The final two conditions assessed the effect of provocation on postural. In condition 5 the weight was suspended without being released; in condition 6 the weight was suspended and released. Participants focussed vision on camera 2 and attention on keeping the weight motionless.

4.3.2.3 Results

Figures 4:11, 4:12 and 4:13 show the distribution of postural sway values around the median for each task. A paired *t*-test revealed that removal of visual cues to orientation did not significantly increase postural sway (t [10] = -1.83, $p = 0.1$) in this sample of adults of normal reading ability. A paired *t*-test also revealed that maintaining an external focus of attention by consciously keeping the weight without motion significantly reduced postural sway in this sample of adults (t [10] = -2.68; $p < 0.05$). Finally, a paired *t*-test demonstrated that releasing the weight in the

provocation paradigm significantly increased postural sway in this sample of adults ($t[10] = 13.62, p < 0.001$).

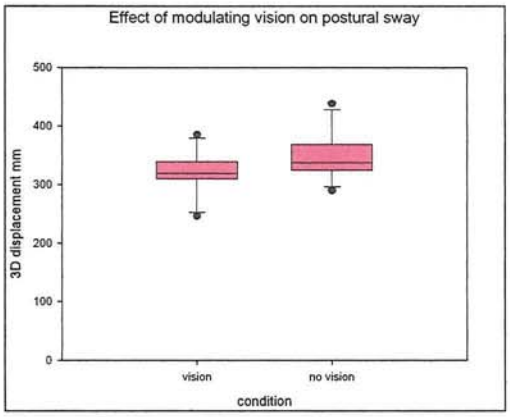

Figure 4:11 Task 1: Assessment of the effect of visual modulation on postural sway.
Left box plot shows postural sway with vision; right box plot shows postural sway without vision. Boxes represent 50% of scores, the horizontal line within is the median. Lower whiskers represent 10th percentile (10% scores below) and upper whiskers represent 90th percentile (90% scores below). Dots represent possible outliers (Clark-Carter, 1997).

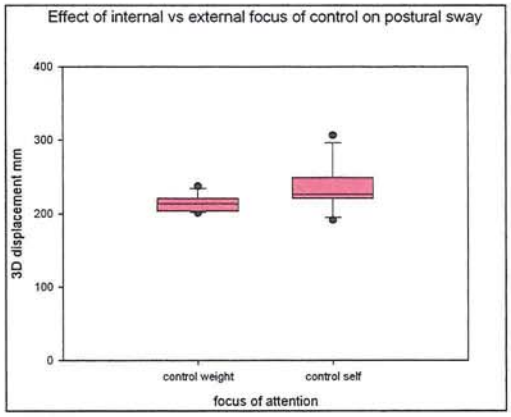

Figure 4:12 Task 2: Assessment of the effect of maintaining an internal versus external focus of attention on postural sway.
Left box plot shows postural sway when instructed to keep the weight motionless; right boxplot shows postural sway when instructed to keep the body motionless.

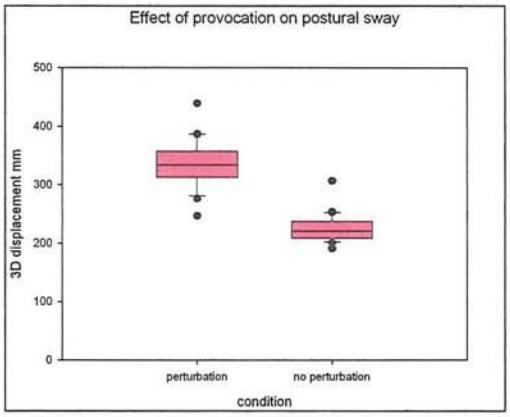

Figure 4:13 Task 3: Assessment of the effect of the provocation test on postural sway.
Left boxplot shows postural sway following perturbation by release of the weight; right boxplot shows postural sway when weight is not released.

4.3.2.4 Discussion

Task 1: Removing visual cues to orientation had a non-significant effect upon postural sway. This group of healthy, young adults however, would be expected to make efficient use of the remaining vestibular and somatosensory cues to orientation.

Task 2: Maintaining an external focus of attention significantly reduced postural sway in upright stance. This suggests that a control group would be predicted to reduce their postural sway by consciously removing their attention from the monitoring of postural stability. A group with dyslexia however, would be predicted by the cerebellar deficit hypothesis to be unable to maintain an external focus of attention as these resources would be required to sustain 'conscious compensation' in controlling postural stability.

Task 3: The release of the weight in the provocation test significantly increased postural sway. The paradigm is therefore a valid measure of postural stability in this sample of adults with normal reading.

4.3.3 Study 3: Comparison of adults with developmental dyslexia and controls

The third pilot study was intended to compare the effects of the provocation test on postural sway and recovery strategy in adults with dyslexia and a peer control group.

4.3.3.1 Participants

Four adults with dyslexia were recruited from the Aston University Dyslexia and Developmental Assessment Centre database. Four Psychology undergraduates of normal reading ability volunteered and received one hour research participation credit. All were healthy individuals, mean age 23 years, range 19 to 34 years.

4.3.3.2 Procedure

Postural stability was tested by the provocation test in two conditions. First, participants were asked to focus their vision on the illuminated number displayed on camera 2. This was immediately ahead at a distance of 2.5m. Second, participants wore a lightweight opaque visor that removed their visual cues to orientation but their eyes remained open throughout (Horak, Nashner & Diener, 1990). There were 5 trials of each condition. The DAST postural stability component was also administered to both groups for comparison.

4.3.3.3 Results

Figure 4:14 shows that there were differences between groups and large Cohen's *d* effect sizes were revealed: .89 for the visual focus and .82 for the visual modulation conditions.

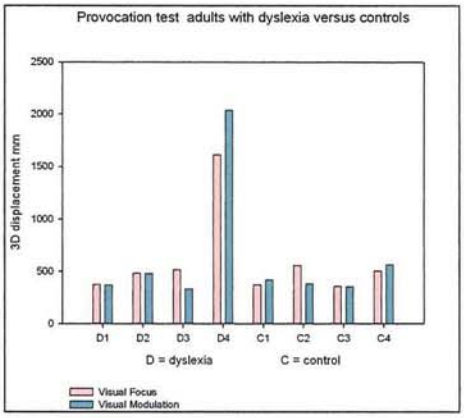

Figure 4:14 Provocation test 3D postural sway Visual Focus and Visual Modulation conditions.
Paired bars represent postural sway by individual participants in each condition.

The DAST Postural Stability component rating scores were summed over four administrations of the test shown in Figure 4:15.

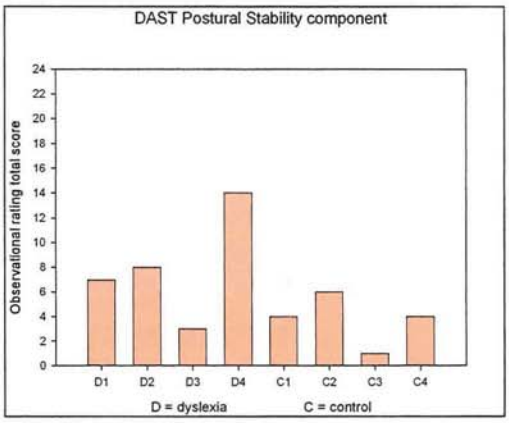

Figure 4:15 DAST Postural Stability component scores.
Bars represent total rating scores over four applications. The maximum score achievable is 24.

4.3.3.4 Discussion

The large effect sizes between-groups suggest that there were adults in the dyslexia group who demonstrated postural instability in both conditions. On closer inspection of the data one participant (D4) was shown to have consistently generated greater postural sway in both conditions. The range of scores indicates that the paradigm discriminates individual differences in postural stability. Inspection of the DAST rating scores revealed that D4 also generated the highest score on this measure. The

distribution of scores looked comparable between paradigms however the small sample size precluded further statistical analysis.

4.4 General discussion

The results suggest that the paradigm is both a valid and reliable measure of postural stability. A weight equivalent to 5% body mass was sufficient to increase postural sway in adult normal reader groups. Removal of visual cues to orientation did not significantly increase postural sway in the normal reading groups in studies 2 and 3, a result that was predicted for healthy young adults. Maintaining an external focus of attention significantly reduced postural sway. This supported the prediction that postural control would improve when attention was focused on the outcome (keeping the weight motionless) rather than the action (keeping the body motionless) of maintaining stability (McNevin and Wulf, 2002). The provocation test therefore, is not only a potentially useful discriminative measure of between-groups postural stability but also a sensitive measure of within group variability, as demonstrated in Study 3 (see Figure 4.14). The paradigm provided a provocation test under experimental control in accordance with the conventions of postural research. Motion capture provided a fine-grained measure of postural sway on each of the 3 cardinal axes and analysis generated a 3D measurement of postural sway.

There was, however, room for improvement in both the procedure and the analysis of the motion capture data if the following experimental studies were to meet the objective criteria set out at the start of this chapter. First, the visor used to remove visual cues was considered to be heavier than anticipated. Second, in order to avoid anticipatory postural adjustments prior to provocation, the distance between the provocation rig and the participant needed to be measured so that their trunk as well as their feet could be returned to the original starting position for trial 1. Third, in order to provide the most accurate, quantitative assessment of postural stability in dyslexia thus far, a more systematic analysis of control of the centre of gravity following provocation needed to be applied. Fourth, in order to make full use of the linear kinematic measurements afforded by the motion capture system, postural sway needed to be assessed separately on A/P and M/L axes. Fifth, the raw data needed to

be low-pass filtered at 5 Hz in order to eliminate the typical signal variability generated by the motion capture system.

The following studies, reported in Chapters 5, 6, 7 and 8, assessed postural stability not only in adults and children with dyslexia but also in children in the mainstream primary school population. The paradigm developed in this chapter, the techniques of kinematic analysis and observational ratings of the manual application of the DST-J/DAST Postural Stability component were used to address the research issues of sample classification, procedure and measurement raised by the meta-analysis and to resolve the research questions defined in Chapter 1. First, is balance related to reading specifically or by association with other developmental disorders? Second, can balance measures be used to predict reading failure? The next chapter describes the initial experimental study that is an evaluation of postural stability in adults with dyslexia.

5 Postural stability in adults with dyslexia

5.1 Introduction

The aim of this chapter was to incorporate the improvements to the method, described below, into an initial experimental study of adults with dyslexia.

5.1.1 Sample classification

Volunteers with dyslexia who showed a discrepancy between FSIQ and reading scores were recruited (see Table 5:1). Likewise, volunteers who were normal readers with IQ within the same classification range of ability were recruited into the control group. The only other criterion applied to both groups was that English was their first language.

5.1.2 Methodological improvements

5.1.2.1 Removal of visual cues to orientation

The lightweight visor initially used to remove visual cues to orientation in the pilot studies was considered to be heavier than anticipated. Participants also complained that it was uncomfortable to wear. The visor was therefore replaced with a pair of plastic industrial goggles made opaque by the addition of layers of air-filled polythene packaging, shown in Figure 5:1.

Figure 5:1 Opaque goggles worn to remove visual cues to orientation.
Goggles are secured with elastic and are adjustable in size.

5.1.2.2 Avoidance of anticipatory postural adjustment

The distance between the provocation rig and the position of the participant's trunk was measured before each provocation to ensure that participants were not making anticipatory adjustments to posture over repeated trials. This improvement was suggested through personal correspondence with Rolf Moe-Nilssen. The position of the feet was also marked and checked before each provocation.

5.1.2.3 Systematic analysis: the inverted pendulum model (Winter, 1995; 2004)

Following the pilot studies, a more systematic method of comparing individual postural sway was sought. This involved a departure from previous studies of dyslexia and balance and an embarkation upon the study of the kinematic techniques typically applied in postural research. Postural sway is typically modelled as an inverted pendulum controlled from the ankle through the centre of pressure (COP) between the soles of the feet and the support surface (Winter, 1995; 2004). The inverted pendulum model predicts that the difference between the COP and the COM is proportional to the horizontal acceleration of the COM, a prediction that has been validated by Winter et al. (1998). Therefore, as acceleration provides an indication of efficiency in the control of motion, measurement of COM motion is proportional to the ability of the CNS to maintain the centre of gravity within the base of support. Within the inverted pendulum model the body is divided into 14 segments that can be used to predict COM motion. The best predictor of COM motion has been shown to be segment 2, the triangular area between the shoulders and the xiphoid process shown in Figure 5:2 (Winter et al., 1998). Measurement of the motion of segment 2 following the provocation test was intended to serve two purposes. First, it provides data for a relatively quick analysis of postural stability in the adult dyslexia population. Second, it provides data for comparison with the total body COM motion data presented in Chapter 6. If segment 2 provided a reliable and valid prediction of COM motion then fewer markers could be used in studies of children. In turn, this would effectively reduce the amount of time required from child participants, which would be particularly beneficial for research in the field.

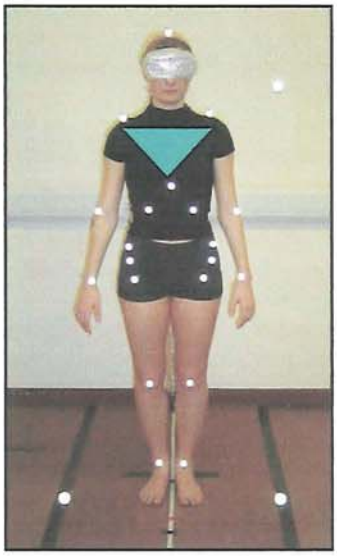

Figure 5:2 Definition of body segment 2 and marker placement.
The green triangle defines the markers of interest and the area of body segment 2.

5.1.2.4 Systematic analysis: recovery strategy

Nashner (1976) first described the development of adaptive motor responses or strategies employed when stability is perturbed (see section 2.1). These strategies are synergistically attuned, timed sequences of muscle activity triggered by the CNS. The most efficient and last to develop involves the body swaying like the inverted pendulum (described in section 5.1.2.3) with all segments in alignment from the ankle (Winter, 1995; 2004). When perturbation is perceived to be faster or greater in force, the hip strategy is employed, whereby the hips are pitched forwards and the trunk is pitched backwards, or *vice versa*, in the sagittal plane. One or both heels may also be raised. A third strategy, the first to develop in upright stance, is employed when the COM and its projected centre of gravity move beyond the limits of the base of support and a step or steps are taken to increase its boundaries and prevent a fall (Nashner, 1976; Horak, Nashner & Diener, 1990).

Assessment of postural stability in the DST-J/DAST component is ranked from 0, rock solid; through 1, slight sway; 2, raising heels; 3, small step; to 6, several steps/fall (Fawcett and Nicolson, 1998; 2004). The main areas of the body observed are therefore the trunk and the feet. The main body segments of interest would be

segment 2 and segments 9 and 10, the lower legs and feet. The motion of segment 2 on the A/P axis would be predicted to increase with swaying, heel raising or taking a step to increase the base of support to maintain balance. Therefore, we surmised that the motion of segment 2 on the A/P axis would provide a valid measure of postural sway for comparison with DAST score ratings.

5.1.2.5 Systematic analysis: marker placement

Figure 5:2 shows the marker placement for the definition of 14 body segments adapted from Winter, Patla, Ishac and Gage (2003). Segment 2 is denoted in green. Other body segments and the method of calculation of COM motion are described in Chapter 6.

5.1.3 Data processing improvements

5.1.3.1 Linear kinematics

For the purpose of this relatively speedy analysis of postural sway data, the motion of segment 2 on the A/P axis was the focus of interest.

5.1.3.2 Data filtering

The data were digitally low-pass filtered (Butterworth 2 pass) at 5 Hz to remove inherent system error from the motion capture data.

5.2 Quantitative motion analysis of postural stability in adults with developmental dyslexia

5.2.1 Introduction

Pilot study 3 generated large *d* effect sizes of 0.89 and 0.82 reported in section 4.3.3.3. The meta-analysis found that the Nicolson and Fawcett (1997) study of

postural stability in adults using the DAST Postural Stability component generated a *d* effect size of 0.9 from a sample of unequal size (dyslexia group: N =15; control group: N =150). Prospective power analysis based on the *d* effect sizes obtained in pilot study 3 revealed that 20 participants would be required to obtain statistical power at alpha level 0.05 with power set at .8 for a one-tailed test. A university-wide recruitment drive was initiated through posters or letters to students who had been assessed for dyslexia through the Disability and Additional Needs Unit. Preliminary results of this study were presented at the British Dyslexia Association (BDA) International Conference (March 2004, see appendix 2). The final results of this study were presented at the annual meeting of the Society for Neuroscience (October 2004) (see appendix 3).

5.2.2 Method

Participants were advised that they would be asked to attend two sessions, and that for one session testing required a change of clothes into shorts and Tee shirt in a private cubicle within the laboratory.

5.2.2.1 Participant characteristics

Fifteen adults, 5 males and 10 females with dyslexia (mean age 21 years 9 months, range 18 years 3 months to 35 years 6 months), were recruited by letter from the Aston University Dyslexia and Developmental Assessment Centre or by poster advertisement in the Disabilities and Additional Needs Unit, the Student Guild and on-campus residence halls. Thirty control adults were recruited from the Psychology Undergraduate Programme, again by poster. Of these, 6 were eliminated as English was not their first language; 4 more were eliminated as they either did not attend or did not complete both required sessions. Twenty control adults, 8 males and 12 females, remained (mean age 19 years 8 months, range 18 years 3 months to 22 years 7 months). The groups did not differ statistically in age. The dyslexia group had all received a formal assessment and diagnosis by a Chartered Psychologist. Informed consent was sought and received from the participants with dyslexia to obtain Wechsler Adult Intelligence Scale (WAIS-III) FSIQ and Wide Range Achievement Test (WRAT3) Reading and Spelling standardized scores from their diagnostic

assessment records. Where different measures had been used by the diagnostician (as for participant D6) or records were unavailable (as for participant D9) the Wechsler Abbreviated Scale of Intelligence (WASI) 4 subtest form and the WRAT3 literacy measures were administered. These measures were also given to the control group, and raw scores were converted to standard scores. Anthropometric measurements (height, weight, body mass index) were recorded for both groups, in keeping with the conventions of postural and balance research. Participants with dyslexia were paid £10, control participants received credit for research participation.

5.2.2.2 Instrumentation

Three ProReflex^{TM} 120 Hz Motion Capture Units (MCUs) (Qualisys Motion Capture Systems, Sweden) and Qualysis Track Manager data acquisition software were used to record the motion of 19mm passive infrared retro-reflective markers. These were placed above the anatomical landmarks shown in Figure 5:1 according to the body segment definition prescribed by Winter's (1995; 2004) inverted pendulum model. The focus of this study was the motion of segment 2. Motion of the markers on each cardinal axis was recorded within a calibrated 3-D coordinate system.

5.2.2.3 Procedure

The DAST postural stability component was administered in accordance with the test manual (see section 4.1.3. and Figure 4:2). Participants were then asked to change clothing and markers were attached to either clothing or skin. Figure 5:3 shows the provocation rig described in section 4.2.1. Participants stood barefoot in the fundamental reference position. They were opposed by a constant horizontal drag equivalent to 5% body mass from a line attached from the waist to a weight via a pulley. The initial position of both feet and trunk were recorded when the weight was comfortably suspended and before the first perturbation. Participants were returned to position before each subsequent trial to prevent anticipatory adjustment of posture. They were instructed to keep the weight as motionless as possible. Two markers were placed on the weight for tracking the point of its release. Motion capture commenced following an auditory cue and, without the participant's prior knowledge, the weight was manually released after a predetermined interval ranging

between 6 and 14 seconds. As for pilot study 3, there were 2 conditions, Visual Focus and Visual Modulation with 6 trials in each condition.

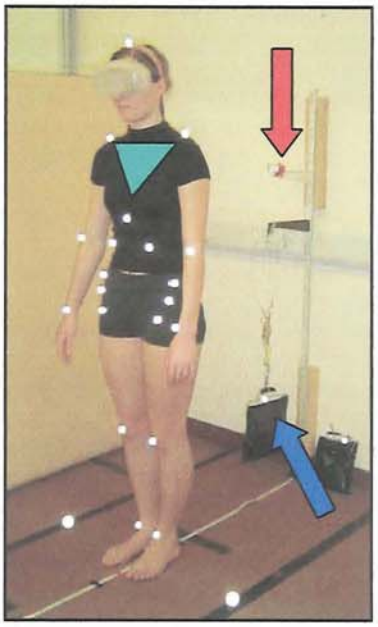

Figure 5:3 The provocation test rig showing modifications.
Model wearing the opaque goggles for the Visual Modulation condition. The red arrow indicates the measurement tape used to return participants to their initial position. The blue arrow indicates the marker on the suspended weight. A spare, identical weight is seen in the background.

5.2.2.4 Analysis

The frame of release of the weight was identified by tracking the trajectory of the weight marker on the vertical axis, as shown in Figure 5:4. From this point a 240 frame (2s) window of data was exported to TSV format. This process was repeated for each of 6 trials. Location of segment 2 centre of mass on the A/P axis was calculated in Microsoft Excel. The root mean square (*RMS*) of the point by point ensemble average across trials was computed using the formula $RMS = \sqrt{(SS / N)}$ (James, 2004), where *SS* is the sum of the squared values of the position of segment 2 centre of mass over time on the A/P axis within the measurement volume, and *N* is the number of data points sampled (i.e. frames). *RMS* sway path was taken as the measure of postural sway. The data were low-pass filtered at 5 Hz and normalized

from the point of release of the weight. Descriptive and inferential statistics for all measures were analysed using SPSS 11.5.

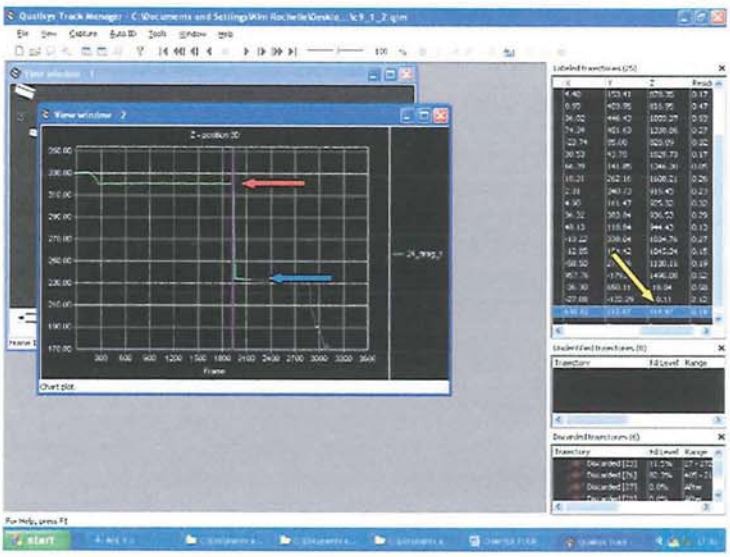

Figure 5:4 Window showing the point of release of the weight.
The green line represents the trajectory of the weight marker on the vertical axis. The red arrow shows the point of release of the weight. The blue arrow shows the endpoint of the downward trajectory of the weight. The yellow arrow shows the 2D position of the marker on the vertical axis.

5.2.3 Results

5.2.3.1 Participant characteristics

Age and gender characteristics for both groups are presented in section 5.2.2.1. Table 5:1 shows the descriptive statistics of the anthropometric, psychometric and literacy measures.

Table 5:1 Descriptive statistics of sample characteristics (mean ± standard deviation).

	Height m	Weight kg	Weight of drag	VIQ	PIQ	FSIQ	WRAT3 Reading	WRAT3 Spelling	FSIQ-Reading
Dyslexia	1.69 ± 9.42	67.76 ± 10.42	3.41 ± 0.51	115.93 ± 13.52	107.27 ± 12.30	112.93 ± 10.95	100.13[a] ± 10.7	96.93[a] ± 15.81	13.80 ± 14.42
Control	1.70 ± 6.64	69.27 ± 14.01	3.46 ± 0.69	117.15 ± 10.04	109.30 ± 10.29	114.65 ± 8.73	112.00 ± 6.66	109.30 ± 5.95	2.65 ± 9.82

[a]Standard score

Differences between groups on anthropometric and psychometric measures were non-significant. Height (t [33] = -.59; p = 0.50), weight (t [33] = -.35; p = 0.80), VIQ (t [33] = -.31; p = 0.80), PIQ (t [33] = -.53; p = 0.60), FSIQ (t [33] = -.52; p = 0.7). Between group differences on the WRAT3 Reading (t [33] = -4.05; p<0.001), WRAT3 Spelling (t [33] = -2.88; p<0.005) and IQ-reading discrepancy (t [33] = 2.72; p <0.05) were significant.

5.2.3.2 DAST Postural Stability component ratings

Figure 5:5 shows the distribution of total scores around the median on the DAST Postural Stability component. The dyslexia group median score was 4 (range 0 – 12); the control group median score was 3 (range 0 – 4). A non-parametric Mann-Whitney U test of group means was used to compare group scores (z = -2.12) which was significant (U=85.5; p <0.05).

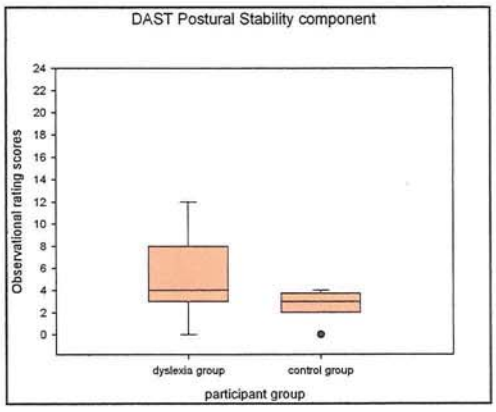

Figure 5:5 DAST Postural Stability component ratings
Boxplots represent the distribution of total rating scores from 4 applications of the test. The maximum score achievable is 24.

5.2.3.3 Postural sway descriptive statistics

Figure 5:6 shows the distribution of *RMS* sway path values around the median in the Visual Focus condition. The presence of outliers demonstrates that one individual within each group (D10 and C3) generated postural sway values beyond the 95th percentile for their group. The descriptive statistics reveal greater variability in *RMS* sway path values in the dyslexia group (mean = 29.40mm ± 33.96mm) than in the control group (mean = 17.90mm ± 8.73mm).

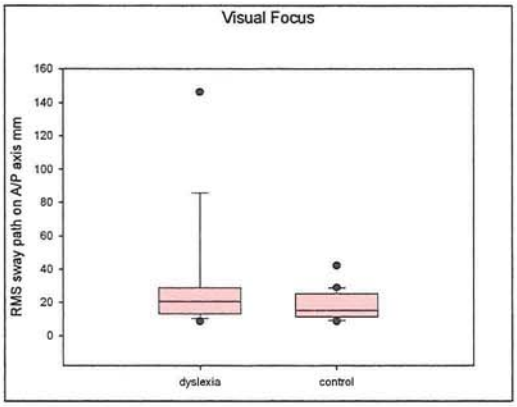

Figure 5:6 Postural sway following provocation in the Visual Focus condition.
Boxplots show the distribution of postural sway values on the A/P axis.

Figure 5:7 shows the distribution of *RMS* postural sway path values around the median value in the Visual Modulation condition.

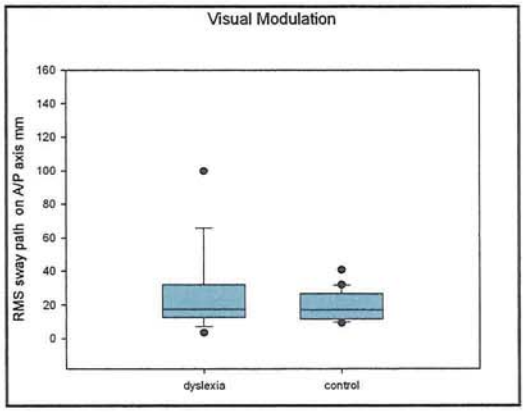

Figure 5:7 Postural sway following provocation in the Visual Modulation condition.
Boxplots show the distribution of postural sway values on the A/P axis.

Two participants (D10 and C12) generated sway values in excess of 90 percent of their group. The descriptive statistics reveal a greater distribution of postural sway values in the dyslexia group (mean = 24.41mm ± 23.30mm) than in the control group (mean = 19.19mm ± 9.15mm).

5.2.3.4 Postural sway inferential statistics

The *RMS* sway path values for both vision conditions were compared using an ANOVA (one within and one between factor). No effect of group (F [1,33] = 1.58, p = 0.22) or vision (F [1,33] = 1.07, p = 0.31) was found and the group by vision interaction was non-significant (F [1,33] = 3.09, p = 0.09). Effect sizes between groups were small (d = .02) for both conditions. This indicates that although some individuals with dyslexia generated considerable postural sway following provocation they were not representative of the majority.

5.2.3.5 Correlational analyses

In view of the *RMS* sway path values generated by individual members of both groups in each condition, non-parametric bivariate correlations (Spearman's Rho) were used to assess the strength of the relationship between postural stability and literacy skills and between the DAST postural stability component scores and the *RMS* values from the provocation test. The results are shown in Table 5:2.

Table 5:2 Non-parametric correlations

Visual Focus	Visual Modulation	WRAT3 Reading	FSIQ-Reading	WRAT3 Spelling	DAST Pos. stability
0.67**					
-0.11	0.20				
0.16	0.08	-0.56**			
-0.06	0.49**	0.46**	-0.27		
0.55**	0.39*	-0.19	0.28	-0.25	

*p <0.05
** p <0.01

5.2.4 Discussion

Measures of central tendency were similar for both groups on the provocation test in both conditions and effect sizes between groups were minimal. There was, however, a much wider distribution of scores across the dyslexia group. One participant (D10)

consistently demonstrated *RMS* segment 2 sway path values in excess of 90 mm on the A/P axis, indicating use of the stepping strategy to increase the base of support in maintaining stability. No other participant generated an *RMS* value in excess of 50mm. It should be noted that D10 generated the most extreme value possible as the paradigm was designed in accordance with health and safety issues raised during the risk assessment. Following provocation, the belt and dragline acted as a restraining harness limiting recovery to a small step or steps thus preventing a fall.

There were significant between group differences on the DAST postural stability component, with which there were also positive, moderate but significant associations with both conditions of the provocation test. This suggested that both measures tapped the same underlying ability but why were the effect sizes so small? It was possible that by focusing on the motion of segment 2 on the A/P axis, use of other strategies to control the COM within the base of support might not be apparent in the data. When perturbation is perceived as a threat to equilibrium that is greater than would warrant use of the ankle strategy, but is smaller than would warrant taking a step, the body ceases to perform as a rigid pendulum and behaves as a linked chain of segments (Winter, 1995; 2004). Segment 2 would be a part of that chain but greater activity in controlling COM motion could be taking place elsewhere within the chain, for example, as demonstrated in heel-raising. This activity is rated on the DAST metric. Therefore motion of segment 2 on one axis may be limited in assessing individual efficiency in maintaining the COM within the base of support. The marker placement used, however, enabled the calculation of body segment and total body COM motion on 3 cardinal axes and this more comprehensive analysis of postural sway is presented in Chapter 6. It was also considered, at this point, whether *RMS* was the best measure of postural sway and study of the methods of kinematic analysis was continued.

Interestingly, neither measure of postural sway shared any more than a small association with reading, as shown in Table 5:2. All correlations were performed for both groups together. This approach is based on the work of the Stein laboratory (Witton et al., 1998; Talcott et al., 2000a; Talcott et al., 2002) that found that good

visual and auditory temporal processing co-occur and predict good reading ability and that generally poor temporal processing predicts reading failure. According to this finding, if postural stability is related to reading by a direct mechanism then good levels of postural stability would predict good levels of reading and instability would predict reading difficulties. This approach is carried through into Chapters 6 and 7.

Nicolson and Fawcett (1997) reported that 63% of their adults with dyslexia were rated 'at risk' of dyslexia on the DAST Postural Stability component. Ramus et al. (2003) reported that 12.5% of their adults with dyslexia generated unusual scores on a measure of deviance from the sample mean. The meta-analysis highlighted the methodological differences between these studies. In the current study, only one adult with dyslexia (6.67%) generated consistent postural instability. This finding is more in keeping with Ramus et al. (2003) who used digital force plate technology to record postural sway. Both the objective assessment of postural stability in adults and the quantitative data afforded by digital technology systems have eliminated the potential for experimenter bias in measurement. This may well account for the difference in findings between these studies. Furthermore, individual calibration of the provocation force in the current study has eliminated individual differences in inertia. It has already been indicated that measurement of the motion of the centre of mass is required to more fully assess postural stability in this sample. What remains to be addressed is how to load the postural control system sufficiently to cause break down of the process of "conscious compensation" (Nicolson & Fawcett, 1990, p.3).

Chapter 2 described theories of motor control and current opinion in postural and balance research. Otherwise healthy adults would be predicted to be able to maintain stability in the absence of either one or two sensory modalities. Individuals with dyslexia however, would be predicted by the cerebellar deficit hypothesis to maintain stability by means of compensatory processes involving conscious control. The growing body of evidence reviewed by Woollacott and Shumway-Cook (2002) and Balasubramaniam and Wing (2002), which was discussed in Chapter 2, has shown that maintenance of stability is a dynamic process requiring both conscious control

and accurate perception and integration of sensory information. Postural stability can be threatened by individual, task or environmental variables. The meta-analysis revealed that only 2 studies (Brown et al., 1985 and Moe-Nilssen et al., 2003) have attempted to assess the effects of sensory modulation on postural stability in dyslexia. Manipulating sensory cues to orientation within the environment would threaten stability. This would be predicted to increase the demand for conscious attention and overload the postural control system leading to a break down of compensatory processes. Maintaining an external focus of attention would be predicted to further impair performance. The effects of sensory modulation on postural stability in dyslexia are therefore further assessed in Chapter 6 where we present COM analysis of the current data and of that generated by two additional conditions.

5.2.5 Conclusions

The results of the provocation test correlate significantly with the DAST Postural Stability component scores suggesting that they tap the same underlying abilities. Calculation of the motion of body segment 2 on the A/P axis provided a quick assessment of postural stability and appears to adequately describe the use of the ankle or stepping recovery strategies. This measurement however, may be limited in its ability to describe use of the hip strategy, which is evidenced through heel-raising in the DAST component. Calculation of the total body COM on 3 cardinal axes from the current data would generate a clearer picture of how individuals are maintaining stability. Also, some adults may be using compensatory processes to maintain stability. These issues will be addressed in Chapter 6 with the introduction of two additional sensory modulation conditions. Also, a comprehensive analysis of further cognitive skills, reading component skills, fine motor skills and behavioural symptoms is conducted to begin to tease apart the nature of the relationship between reading and balance.

6 The sensory hierarchy in postural stability in adults with dyslexia

6.1 Introduction

This study is a further investigation of the data presented in Chapter 5 and of data collected in a further two conditions that are hitherto unreported. The first aim of the current study was to assess the effects of manipulating the sensory hierarchy on postural stability following provocation, in the sample of adults with reading discrepancy and controls. This method of loading the postural control system to induce sway was adopted as an alternative to a dual task paradigm (see section 2.3 and Figures 2:4 & 2:5). Proponents and challengers of the cerebellar deficit hypothesis have typically employed dual task paradigms, whereby a task secondary to the primary balance task was used to draw attention away from the conscious control of equilibrium. Dual tasks typically require a verbal response to a choice reaction task while simultaneously performing a balance task (e.g., Yap & van der Leij, 1994; Nicolson & Fawcett, 1995; Raberger & Wimmer, 2003; Wimmer, Mayringer & Raberger, 1999). Arousal and articulation are known to increase postural sway in dual task performance (Maki & McIlroy, 1996; Yardley, Gardner, Leadbetter & Lavie, 1999). Therefore the nature of the secondary task itself may be a confounding variable in dyslexia research, even though the task may have been individually calibrated to standardise the level of difficulty across the sample. It was predicted that manipulating vision and cutaneous cues to the support surface would reduce redundancy within the sensory frame of reference hierarchy and draw attention from conscious control. Combining the effects of sensory modulation with a provocation paradigm was intended to create a dual task paradigm without using a potentially confounding secondary task.

It was predicted that most adults would be able to compensate adequately on a firm support surface with or without visual cues to orientation because balance deficits in dyslexia are hypothesised to be subtle (Nicolson and Fawcett, 1999). This prediction was supported by the findings of Chapter 5, although motion of body segment 2 may not have adequately described motion of the centre of mass. Redundancy was

therefore reduced within the sensory frame of reference hierarchy by modulating vision and cutaneous perception of the support surface as described in the procedure used by Horak, Nashner and Diener (1990). It was predicted that maintaining an external focus of attention would further threaten hypothesised compensatory processes in dyslexia, which would be exacerbated in the presence of co-existing attention deficits. Measures of inattention and hyperactivity/impulsivity symptoms were therefore also administered to the sample.

The second aim was to provide the most comprehensive analysis of postural stability in adults with dyslexia to date. The measure of postural sway was motion of the COM within the measurement volume on each of the 3 cardinal axes. It was predicted that motion on the anterior-posterior (A/P) and medial-lateral (M/L) axes would reveal trunk sway, indicating use of either an ankle or a stepping strategy. Motion on the vertical axis would indicate heel-raising during use of a hip strategy. Finally, the psychometric, literacy and behavioural correlates of balance skills and deficits would be examined and the results compared with the general findings of the meta-analysis (Chapter 3). The results of this study were presented at the International Society for Postural and Balance Research Conference (May, 2005) (see appendix 4).

Motion of the total body COM represents the motion of the body as a whole and it is continuously changing position. Even during quiet standing, minor adjustments are made over time as the body withstands gravity. Calculation of the centre of mass is therefore essential in studies of posture and balance (Winter, 1995; 2004). Infrared digital optical motion capture systems, such as Qualisys, have the capability to record marker displacement with a precision of 0.03mm. It has been shown that estimates of the total body COM are possible to a precision of 3mm in the A/P direction and 1.5mm in the M/L direction (Winter, Patla, Ishac and Gage, 2003). Measurement of postural sway based upon motion of the total body COM on the A/P and M/L axes would therefore provide a level of systematic analysis previously unexplored in the study of balance in dyslexia. Although provocation on the A/P axis would be expected to generate an equal, opposite and collinear reaction, according to

Newtonian principles, deviation on the M/L axis might be indicative of atypical postural sway and warrant further investigation. In previous dyslexia research, only Brown et al. (1985) had examined postural sway on the A/P and M/L axes separately using a strain gauge force transducer placed beneath their participants' feet.

6.2 Method

6.2.1 Participant characteristics

The same 15 adults with dyslexia participated as described in section 5.2.2.1. Of the control group two participants did not complete the additional conditions therefore their data were removed. This left 18 adults (7 males and 11 females, mean age 19 years 7 months, range 18 years 3 months to 22 years 7 months). Removal of the data from these two participants did not affect chronological or FSIQ status of the control group. In addition to the psychometric and literacy measures administered in Chapter 5 (see section 5.2.2.1) participants also completed the measures described in sections 4.1.2.1, 4.1.2.2, 4.1.2.5 and 4.1.2.6.

6.2.2 Instrumentation

As described in section 5.2.2.2.

6.2.3 Procedure

There were six trials, as described in section 4.2.2.3, in each condition. In condition 1: *Visual Focus (VF),* subjects stood on a firm surface and were instructed to focus their vision on a target, the illuminated numeral displayed on camera 2, at a distance of 2.5m. In condition 2: *Visual Modulation (VM)* subjects stood on a firm surface while wearing lightweight industrial goggles rendered opaque to eliminate visual cues to orientation. In condition 3: *Cutaneous Modulation (CM)* subjects stood on a compliant surface of 8cm depth with eyes fixated as for VF. In condition 4: *Bimodal Modulation (BM)* subjects stood on the compliant surface while wearing the opaque

goggles. Conditions 1 and 2 were described in Chapter 5. Figure 6:1 shows the four sensory modulation conditions.

Figure 6:1 The four sensory modulation conditions.
The marker placement shown here is the Winter et al. (2003) specification. This was adapted by replacing the ear markers with a single crown marker as used in the pilot studies.

6.2.4 Analysis

The path of the marker on the weight was charted on the vertical axis to determine the frame of its release (see Figure 5:4). A 2 second post-provocation window of data was exported and low pass filtered at 5 Hz, then normalized from the point of release of the weight. The fundamental requirement for the calculation of the location of COM is marker placement according to the 14 body segment definition model, described by Winter et al. (2003). This adaptation was necessary in view of the difficulties encountered in placing the ear markers, opting instead to use a single marker on the crown as shown in Figure 6:2.

Figure 6:2 Marker placement and body segment definition (adapted from Winter, Patla, Ishac and Gage (2003).
The coloured lines define individual body segments. The numbers denote the number of the segment according to the Winter et al. (2003) definition.

As total body mass increases so does the mass of individual body segments. The mass of each segment can therefore be expressed as a percentage of total body mass as shown in column 3 of Table 6:1. The body segment definition, marker placement and mass fractions shown in Figure 6:2 and Table 6:1 were used to calculate the motion of the COM on each cardinal axis using Equation 6:1: where COM is the position of the total body centre of mass during one frame of motion capture, m represents the mass fraction, x represents the co-ordinate of the segment centre of mass in one frame of motion capture and M is the total body mass. Calculations were performed using Microsoft Excel.

Table 6:1 Segment definitions and COM coordinates (Winter, Patla, Ishac and Gage, 2003).

Equation 6:1 Calculation of total body centre of mass location (adapted from Winter et al., 2003).

$$COM = \frac{(m1x1)+(m2x2)+(m3x3)+(m4x4)+(m5x5)+(m6x6)+(m7x7) \text{ to } (m14x14)}{M}$$

The point by point ensemble average across trials and the curve average variability, for each participant were calculated (James, 2004). These measures enabled us to present both discrete and continuous measurements of postural sway for all conditions. Descriptive and inferential statistics for all measures were computed using SPSS 11.5.

6.3 Results

6.3.1 Sample characteristics

Following the removal of data from two control participants (described in section 6.2.1), Table 6:2 shows the revised descriptive statistics of the anthropometric measures for both groups. Differences between groups on anthropometric measures were not significant (height [*t* {31} = -.21, *ns*] and weight [*t* {31} = -.30; *ns*]).

Table 6:2 Descriptive statistics of sample characteristics: anthropometric measures

	Height m	Weight kg	Weight of drag
Dyslexia	1.69	67.76	3.41
N = 15	±	±	±
	9.42	10.42	0.51
Control	1.70	69.04	3.45
N = 18	±	±	±
	6.04	13.66	0.67

Table 6:3 shows the revised descriptive statistics of the psychometric and literacy measures for both groups. Differences between groups on psychometric measures were not significant (VIQ [t {31} = -.22; *ns*]; PIQ [t {31} = -.12; *ns*]; FSIQ [t {31} = -.23, *ns*]). Between group differences on WRAT3 Reading (t [31] = -3.74; $p<0.005$), WRAT3 Spelling (t [31] = -3.31; $p<0.005$), and FSIQ-WRAT3 Reading (t [31] = 2.57; $p<0.05$) a measure of IQ and reading discrepancy, were significant. This confirmed the difference in reading ability between the groups.

Table 6:3 Descriptive statistics of sample characteristics: psychometric and literacy measures

	VIQ	PIQ	FSIQ	WRAT3 Reading	WRAT3 Spelling	FSIQ-Reading
Dyslexia	115.93	107.27	112.93	100.13	96.93	12.80
N = 15	±	±	±	±	±	±
	13.52	12.30	10.95	10.7	15.81	12.92
Control	116.83	107.72	113.72	110.94	110.22	2.78
N = 18	±	±	±	±	±	±
	10.29	9.59	8.55	5.49	5.48	9.44

6.3.2 WAIS-III Digit Span, Digit Symbol and Digit Copy

The descriptive statistics summarising group performance on the WAIS-III subtests are presented in Table 6:4.

Table 6:4 Descriptive statistics of WAIS-III subtests.

	WAIS-III		
	Digit Span	Digit Symbol	Digit Symbol Copy
Dyslexia *N* = 15	97.67 ± 11.16[a]	90.33 ± 9.54[a]	25[b] (5 – 50)
Control *N* = 18	99.72 ± 11.02	100.83 ± 7.91	50 (25 – 50)

[a]standard score
[b]cumulative percentage

The difference between groups on the Digit Span sub test was not significant (t [31] = -.53, p = 0.30) and the distribution of scores for both groups was similar (± 11 standard scores) indicating that the groups demonstrated similar variability in scores. This was unusual as individuals with dyslexia usually demonstrate significant weakness on this measure of working memory. Between groups difference on the Digit Symbol subtest however, revealed that the dyslexia group demonstrated significantly weaker visual motor coordination and processing speed than the control group (t [31] = -3.46; $p < 0.05$). A non-parametric Mann-Whitney U means test was used to compare group cumulative percentage rankings for the Digit Symbol Copy subtest (z = -2.96). This was found to be significant (U = 63.00, p <.005), indicating that this group also demonstrated significantly weaker perceptual and graphomotor speed.

6.3.3 Reading component skill measures

Descriptive statistics summarising group performance on the reading component skill measures are presented in Table 6:5. Between groups differences on the orthographic choice task were not significant for either latency (t [30] = 1.45, *ns*) or accuracy (t [30] = -1.44, *ns*). Data for one control participant on this task were missing. Between groups differences on the phonological choice task were significant on both response

latency (t [31] = 2.72, $p < 0.05$) and accuracy (t [31] 2.30, $p < 0.05$). There were significant differences between groups on DAST measures of orthographic fluency, speed and accuracy, (t [31] = -3.58; $p < 0.005$), and of phonological ability (t [31] = -5.09; $p < 0.001$). The dyslexia group scores for the PhAB Spoonerisms subtest were also significantly lower than those of the control group (t [31] = -3.23; $p < 0.005$). Although the dyslexia group demonstrated fluency, accuracy and phonological difficulty their ability on the orthographic choice task suggests that either they did not have orthographic recognition difficulties or their compensatory reading skills were well established.

Table 6:5 Descriptive statistics summarising group reading component skills.

	Olson et al., (1994)				DAST		PhAB
	Orthographic choice		Phonological choice				
	mean RT[a]	% correct[b]	mean RT[a]	% correct[b]	One Minute Reading[c]	Nonsense Passage[d]	Spoonerisms[e]
Dyslexia group	1281ms ± 504ms	92.42 ± 8.34	4170ms ± 1177ms	81.11 ± 11.96	89.33 ± 23.07	77.67 ± 13.32	97.07 ± 9.29
Control group	1073ms ± 290ms	95.5 ± 2.77	3207ms ± 852ms	88.98 ± 7.55	111.17 ± 10.69	94.55 ± 4.27	110.17 ± 13.19

[a]Average response time following presentation of word.
[b]Percentage of correct responses.
[c]Number of words attempted minus errors and passes, plus bonus if less than 60 seconds.
[d]Number of real and nonsense words correct plus or minus time bonus or penalty.
[e]Standard score at test ceiling 14.06-14.11 years.

6.3.4 Measures of inattention and hyperactivity/impulsivity symptoms

Figures 6:3 and 6:4 summarise the group self-rating scores for their behaviour over the previous 6 months, on the Barkley Current Symptoms scales of attention. The number of self-rating behaviour scores of either 2 (i.e., behaviour is often like the given statement) or 3 (i.e., is very often like the given statement) were summed on each dimension.

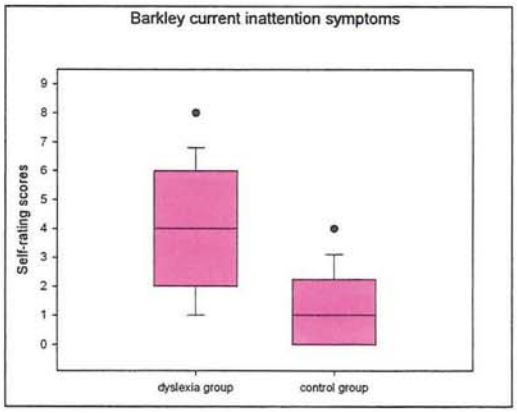

Figure 6:3 Barkley Current Symptoms self-rating scores for inattention.
Boxplots show the distribution of total self-rating scores for each group. The maximum score achievable is 9.

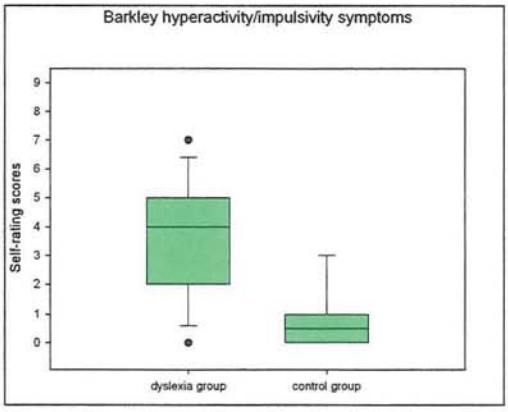

Figure 6:4 Barkley Current Symptom self-rating scores for hyperactivity/impulsivity.
Boxplots show the distribution of total self-rating scores for each group. The maximum score achievable is 9.

Mann-Whitney U tests revealed significant differences between groups on ratings for Inattention Symptoms (z =-3.62; U = 36.5; $p < 0.001$) and Hyperactivity Symptoms (z = -4.01; U = 26.5; $p < 0.001$). The dyslexia group included one participant who exceeded the DSM-IV *TR* clinical cut off score of six for inattention symptoms and another who exceeded the cut-off score of six for hyperactivity/impulsivity symptoms. Nine participants with dyslexia exceeded the Barkley and Murphy (1998) age standardized cut off score for inattention symptoms, two exceeded the level for hyperactivity and a further four were just one score below the cut-off threshold.

6.3.5 Fine motor skills assessment

Table 6:6 shows the descriptive statistics for the peg moving task (Annett, 1985).

Table 6:6 Descriptive statistics for peg moving task

The mean time taken to complete the task was calculated across 3 trials for each hand. The between groups differences were not significant for either right hand (t [31] = -1.13, *ns*), left hand (t [31] = -1.14; *ns*) or difference between hands expressed as function of time (t [31] = -.19; *ns*). This indicated that the groups were comparable in terms of fine motor skills and lateral control on this measure.

6.3.6 Postural stability following the provocation test

The outcome measure of postural sway was the motion of total body COM on the A/P, M/L and vertical axes. First, the discrete measure of maximum excursion on the A/P axis was assessed. Summary statistics of the balance measures are shown in Table 6:7. An ANOVA (one between and one within-groups factor) revealed no main effects of group or sensory modulation condition and a non-significant group-condition interaction. Effect sizes between groups were small with the greatest magnitude revealed in the Cutaneous Modulation condition (see Table 6:7). Continuous measures of postural sway were also computed. The measure of continuous sway following provocation was the standard deviation (SD) of the point by point ensemble average for each participant on the A/P, M/L and vertical axes. A main effect of group (F [1,31] = 4.10; p = 0.05), but not condition was revealed on the A/P axis with a non-significant group-condition interaction. There were no main effects and no interaction between factors on the M/L or the vertical axes. The measure of continuous sway yielded between-groups effect sizes that were smaller for the Visual Modulation than for the Visual Focus condition on the A/P and M/L

axis. This indicated that the dyslexia group demonstrated greater stability without visual cues to orientation. The largest effect sizes on all axes were obtained in conditions where cues to the support surface were modulated. This indicated that the dyslexia group generated the weakest postural stability when somatosensory cues to orientation were manipulated.

To assess individual variability of postural sway across trials the curve average standard deviation ($_{avg}$SD) (i.e., the mean point by point standard deviation across trials; James, 2004) was calculated for each participant, on each axis, across conditions. A significant effect of group was found on the A/P axis (F [1, 31] = 7.11, $p < 0.05$), and on the M/L axis (F [1,31] = 6.37; $p < 0.05$), but no within-groups effect of condition and no interaction between factors. No main effects were found on the vertical axis. Between-groups sway variability effect sizes were, in general, larger than those generated for the continuous sway measures. This highlighted the greater variability of postural stability across conditions shown by the dyslexia group.

Table 6:7 Descriptive and inferential statistics summarising group performance on the provocation test.

	Dyslexia group A/P		Control group A/P		between groups effect size *d*	Dyslexia group M/L		Control group M/L		between groups effect size *d*	Dyslexia group Vertical		Control group Vertical		between groups effect size *d*
	Mean	SD	Mean	SD		Mean	SD	Mean	SD		Mean	SD	Mean	SD	
Peak sway mm VF[a]	28.94	30.98	22.13	10.42	0.31	-	-	-	-	-	-	-	-	-	-
Peak sway mm VM[a]	20.32	13.67	21.29	7.28	-0.09	-	-	-	-	-	-	-	-	-	-
Peak sway mm CM[a]	27.91	19.99	20.68	9.60	0.47	-	-	-	-	-	-	-	-	-	-
Peak sway mm BM[a]	23.97	20.67	21.32	5.49	0.18	-	-	-	-	-	-	-	-	-	-
Recovery latency ms	822	377	807	353	0.04	-	-	-	-	-	-	-	-	-	-
Recovery latency ms	750	306	796	355	-0.14	-	-	-	-	-	-	-	-	-	-
Recovery latency ms	751	330	603	196	0.57	-	-	-	-	-	-	-	-	-	-
Recovery latency ms	589	264	590	152	-0.12	-	-	-	-	-	-	-	-	-	-
SD[b] sway mm VF	9.71	10.36	6.22	3.50	0.47	2.26	2.02	1.73	0.96	0.35	1.81	2.82	0.94	0.48	0.45
SD sway mm VM	7.95	5.55	6.51	1.77	0.36	1.97	1.00	2.05	3.01	-0.03	1.96	3.34	0.80	0.41	0.51
SD sway mm CM	10.40	8.31	6.34	3.43	0.66	2.54	1.48	2.29	1.34	0.18	2.86	3.53	1.03	0.81	0.75
SD sway mm BM	12.28	8.13	7.23	1.64	0.90	3.45	2.28	2.22	1.44	0.66	3.28	4.67	1.09	0.57	0.69
$_{avg}$SD[c] sway mm VF	18.29	19.16	10.65	4.30	0.57	5.34	4.85	4.56	2.94	0.21	3.33	4.51	1.89	1.23	0.45
$_{avg}$SD sway mm VM	11.35	5.45	9.62	4.63	0.35	5.85	2.04	4.45	2.47	0.63	2.22	2.36	1.61	1.04	0.34
$_{avg}$SD sway mm CM	17.24	15.08	8.86	2.78	0.81	6.86	4.26	4.29	2.54	0.75	4.53	4.68	2.28	1.37	0.68
$_{avg}$SD sway mm BM	17.53	12.37	7.84	3.32	1.15	8.62	5.97	5.37	3.11	0.70	3.57	3.85	1.98	1.19	0.58

Note: Dash indicates item was not calculated.

[a]VF = Visual Focus, VM = Visual Modulation; CM = Cutaneous Modulation, BM = Bimodal Modulation

[b]SD sway = standard deviation of the ensemble average displacement during 2 seconds post-provocation (continuous sway).

[c]$_{avg}$SD = average of the point by point standard deviation, during 2 seconds post-provocation, across trials (sway variability).

Table 6:8 Non-parametric correlations among the psychometric, literacy, behavioural and postural sway measures.

	WASI	WRAT3		FSIQ-	WAIS		Olsen et al.(1994)		DAST		PhAB	Barkley	
	FSIQ	Reading	Spelling	WRAT3 Reading	Digit Symbol	Digit Copy	Ortho RT	Phono RT	One Minute	Nonsense Passage	Spoonersms	Inattention	Hyperactivity
COM SD sway path													
VF[a] A/P	-0.04	-0.12	0.03	0.14	-0.03	-0.32*	0.28	0.22	-0.12	-0.07	-0.22	0.01	0.40*
VF M/L	-0.06	-0.02	-0.08	-0.04	-0.08	-0.01	0.35*	-0.13	0.05	-0.04	-0.14	-0.17	0.02
VF V	-0.17	-0.26	-0.13	0.18	0.06	-0.13	0.07	0.10	-0.14	-0.06	-0.15	-0.04	0.26
VM[a] A/P	0.14	0.19	0.25	0.01	0.02	0.01	0.20	-0.32	0.19	0.22	0.08	-0.04	0.14
VM M/L	-0.05	-0.27	-0.13	0.28	-0.02	0.07	0.04	0.08	-0.26	-0.30	-0.14	0.14	0.35*
VM V	-0.02	-0.17	0.04	0.18	-0.09	0.01	-0.09	0.03	-0.08	-0.09	-0.24	0.15	0.39*
CM[a] A/P	0.02	-0.14	-0.16	0.23	-0.13	-0.01	0.31*	-0.16	0.01	-0.03	-0.24	0.19	0.31*
CM M/L	-0.01	-0.10	-0.03	0.01	-0.14	0.03	0.23	-0.10	-0.11	0.08	-0.07	-0.07	0.20
CM V	0.11	-0.41**	-0.31	0.49**	-0.16	0.13	0.07	0.10	-0.23	-0.32*	-0.43**	0.26	0.52**
BM[a] A/P	0.22	0.14	-0.15	0.26	-0.02	0.13	0.15	-0.25	0.14	-0.13	-0.22	0.31*	0.25
BM M/L	-0.05	-0.37*	-0.27	0.22	-0.16	-0.25	0.12	0.14	-0.13	-0.26	-0.41**	0.10	0.32*
BM V	0.40*	-0.31*	-0.26	0.65**	-0.05	0.01	0.06	-0.06	-0.04	-0.21	-0.16	0.37*	0.35*
COM sway variability across trials													
VF A/P	-0.09	-0.25	-0.14	0.18	-0.16	-0.06	-0.18	-0.02	-0.13	-0.22	-0.23	0.12	0.35
VF M/L	-0.07	-0.19	-0.14	0.08	-0.26	-0.16	-0.06	0.19	-0.39*	-0.30*	-0.01	-0.12	0.23
VF V	-0.10	-0.39*	-0.13	0.30	-0.12	-0.07	-0.04	0.02	-0.13	-0.09	-0.18	0.15	0.24
VM A/P	0.02	-0.00	0.14	0.03	-0.24	-0.02	0.10	-0.02	0.06	-0.00	0.03	0.05	0.24
VM M/L	-0.06	-0.16	-0.26	0.14	-0.08	-0.06	0.01	-0.05	0.04	-0.29*	-0.56**	0.15	0.37*
VM V	-0.09	-0.14	-0.16	0.08	-0.22	-0.04	0.24	-0.07	-0.01	0.08	-0.16	0.05	0.25
CM A/P	-0.07	-0.21	-0.15	0.19	-012	-0.09	0.38*	0.19	-0.04	-0.13	-0.30*	0.22	0.47**
CM M/L	-0.18	-0.39*	-0.38*	0.26	-0.54**	-0.26	0.14	0.03	-0.15	-0.28	-0.26	0.18	0.61**
CM V	0.05	-0.16	-0.02	0.26	-0.06	0.13	0.26	-0.14	0.06	-0.00	-0.29*	0.20	0.41**
BM A/P	-0.20	-0.26	-0.27	0.22	-0.18	-0.32**	-0.04	0.18	-0.15	-0.36*	-0.35*	0.17	0.36*
BM M/L	-0.30*	-0.11	-0.15	-0.00	-0.26	-0.08	-0.07	0.13	-0.24	-0.37*	-0.21	0.08	0.51**
BM V	0.15	-0.29	-0.11	0.36*	-0.02	-0.14	0.17	0.02	0.01	-0.15	-0.26	0.20	0.26

[a]VF = Visual Focus, VM = Visual Modulation; CM = Cutaneous Modulation, BM = Bimodal Modulation

*$p<0.05$

**$p<0.01$ $N = 33$, critical value = 0.41

6.3.7 Correlation analyses

In view of the varied levels of data and distributions of scores generated by the measures non-parametric correlations were performed among the psychometric, literacy, attention and balance scores. No correction was performed for multiple comparisons in these correlational analyses, which may have inflated the Type 1 error rate. Only correlations with an associated alpha level 0.01 (1-tailed) were therefore considered significant. Table 6:8 shows that the strongest associations between reading and postural stability were found between continuous sway on the vertical axis in the Cutaneous and Bimodal Modulation conditions and the measure of reading discrepancy (FSIQ-WRAT3 Reading). Cutaneous and Bimodal Modulation continuous sway values also correlated moderately and significantly with Barkley Attention Scale hyperactivity/impulsivity self-rating scores. Reading discrepancy was also associated with Barkley inattention self-rating scores (r_s = .57, $p < 0.001$) but not significantly with hyperactivity/impulsivity scores (r_s = .34, p = 0.03). WRAT 3 Reading scores ratings and Barkley Scales of inattention (r_s = -.49; $p < 0.005$) and hyperactivity/impulsivity (r_s = -.52; $p < 0.005$) were also moderately but none the less significantly associated. These results indicated a potential link between reading and postural stability by means of a third variable, hyperactivity/impulsivity, which shared an association of similar strength with both reading and postural stability when cutaneous perception of the support surface was modulated. The associations between age, continuous sway in the Cutaneous (r_s = -.37; p = 0.02) and Bimodal Modulation conditions (r_s = -.32; p = 0.04) and Reading Discrepancy (r_s = -.32; p = 0.04) were of equal strength and significance.

6.3.8 Identification of individual differences from the sample mean

SD sway path values on the AP axis across conditions were standardized for each participant. Figure 6:5 illustrates the stacked *z* scores. Participants D3, D7, D10, D11 and D13 generated the greatest deviance from the sample mean overall, with scores in excess of 1 SD above the mean in at least 1 condition. Table 6:9 shows *z* scores for these participants to illustrate individual patterns of balance, literacy and behaviour scores. D3, D7, D10 and D13 also generated hyperactivity/impulsivity ratings 1 SD beyond the sample mean. Participant D10's consistently unusual scores across

measures support the conclusion that the relationship between reading difficulties and postural instability is by association with hyperactivity/impulsivity. The scores for participants D3, D7 and D13, although not as consistent as those of D10, support the suggestion that the relationship between balance and either hyperactivity/impulsivity or combined inattention and hyperactivity/impulsivity symptoms is stronger than that between balance and reading.

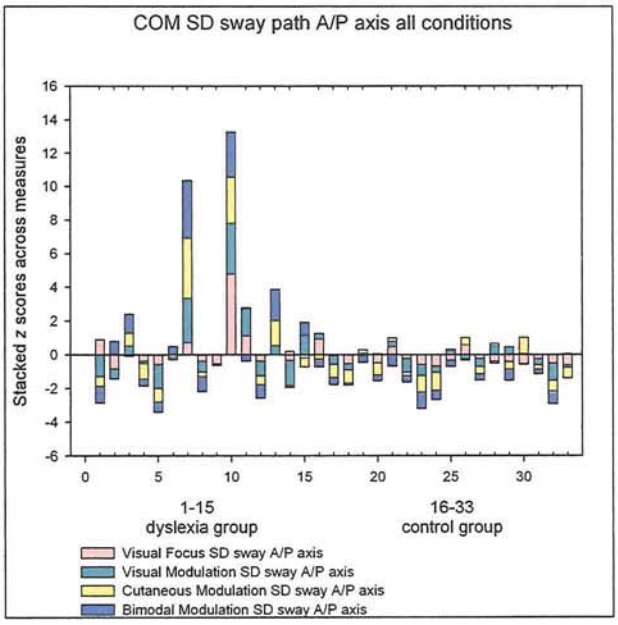

Figure 6:5 Stacked *z* scores for continuous sway on the A/P axis across conditions
Each coloured block represents the standardised value (*z* score) of postural sway averaged across trials for one participant in one condition. Baseline (0) represents the sample mean. Where individual *z* scores across conditions consistently fall either above or below the mean they are shown as unidirectional, cumulative stacks. Where scores across conditions are mixed (i.e., ± the sample mean) stacks intersect the baseline. Stacks above the baseline represent scores above the mean. Stacks below the baseline represent scores below the mean.

Table 6:9 Individual differences in standard deviation from the sample mean across measures.

	Postural sway A/P axis				WRAT3		Barkley Scale	
	VF	VM	CM	BM	Reading	Spelling	Inattention	Hyperactivity
D3	-0.07	0.56	0.73	1.11	0.41	-0.17	1.5	1.86
D7	0.74	2.61	3.58	3.46	0.71	0.52	1.1	1.38
D10	4.78	3.03	2.73	2.74	-1.64	-1.01	0.63	2.34
D11	1.15	1.60	0.03	-0.37	0.81	-0.17	-0.67	-0.54
D13	0.13	0.55	1.48	1.82	-0.21	0.14	-0.67	1.38

6.3.9 Individual differences in recovery strategy within the dyslexia group

In view of the variability in performance across trials in the dyslexia group, the acceleration of segment 6, the hip and pelvic region of the body, on the A/P axis was next examined. This measure was selected to assess the efficiency of the postural control system across the dyslexia group. The A/P axis was selected in view of the directional axis of the provocation force. Acceleration (the second derivative of postural sway displacement) was calculated and the standard deviation of acceleration over 240 frames was taken as the outcome measure. Standard deviations of acceleration in mm/s^2 on the A/P axis were converted to standardized *z* scores. Large positive *z* scores were interpreted as indications of reduced efficiency and weak postural control. Figure 6:6 represents the efficiency of recovery strategy on the A/P axis.

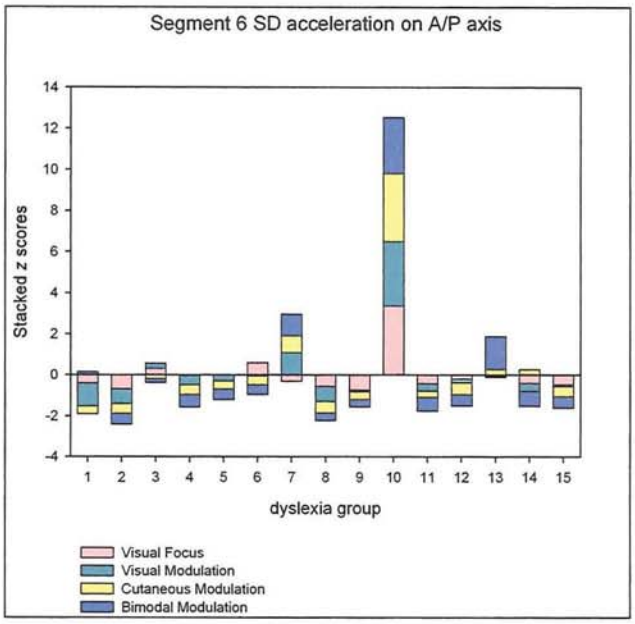

Figure 6:6 Acceleration of hip and pelvis on A/P axis: efficiency of recovery strategy. Each coloured block represents the standardised value (*z* score) of postural sway acceleration averaged across trials for one participant in one condition. Baseline (0) represents the group mean. Where individual *z* scores across conditions consistently fall above or below the mean they are shown as unidirectional, cumulative stacks. Where scores across conditions are mixed (± group mean) stacks intersect the baseline. Stacks above the baseline represent scores above the mean. Stacks below the baseline represent scores below the mean.

Participant D7 presented scores above 1 SD in the Visual Modulation and Bimodal Modulation conditions and approaching 1 SD (0.81) in the Cutaneous Modulation condition. Participant D10 presented scores in excess of or approaching 3 SDs above the group mean in all conditions. Less than .03% of the population would be expected to achieve scores this deviant from the group mean. Participant D13 presented scores below the mean in the first two conditions and above the mean in the Cutaneous Modulation (0.28) and Bimodal Modulation (1.60) conditions. No other participants demonstrated unusual acceleration of the hip and pelvic region in maintaining postural stability.

Finally, relationships between efficiency of recovery from provocation and measures of literacy and attention for the dyslexia group were examined. Non-parametric correlations were conducted between *z* scores for SD acceleration of body segment 6 and all psychometric and literacy measures. Acceleration of segment 6 on the A/P axis in the Visual Focus and Visual Modulation conditions and Barkley

Hyperactivity/impulsivity Scale scores correlated strongly (r_s = .74, p <0.005; r_s = .73, p < 0.005). Acceleration of this segment in the Cutaneous and Bimodal Modulation conditions was moderately associated with hyperactivity/impulsivity self-rating scores (r_s = .48, *ns*; r_s = .45, *ns*). Associations between acceleration of segment 6 and reading scores ranged from near zero in the Visual and Bimodal Modulation conditions (r_s = .04; *ns;* r_s = ..00, *ns*) to small in the Visual Focus and Cutaneous Modulation conditions (r_s = . .32, *ns*, r_s = .32, *ns*). This provided further support for our prediction that inefficient postural control was more strongly associated with symptoms of hyperactivity/impulsivity than with reading.

6.4 Discussion

In this study, the first aim was to assess the effects of manipulating the sensory frame of reference hierarchy on postural stability in dyslexia. The second aim was to introduce the most objective, quantitative and comprehensive analysis of postural stability in adults with dyslexia to date, using procedures and techniques adopted from postural and balance research. The principles of linear kinematics were applied to the data to measure and analyse the motion of centre of mass on the anterior-posterior, medial-lateral and vertical axes. The procedural and measurement criteria, defined in sections 4.1.2 and 4.1.3, were therefore satisfied. Finally, the correlates of postural instability were examined.

In addition to the psychometric and literacy measures administered, individuals rated their behaviour over the previous six months against the Barkley Inattention and Hyperactivity/impulsivity Current Symptoms Scales significantly higher than the control group. Several participants with dyslexia exceeded the clinical cut off score for age standardised population norms for inattention (e.g., D12) and hyperactivity/impulsivity (e.g., D10). The dyslexia group therefore, included some participants with co-existing inattention or hyperactivity/impulsivity or combined symptoms. It was predicted that the effects of modulating sensory cues to orientation

would be exacerbated in the presence of co-existing attention deficits. This prediction was supported.

Between groups effect sizes for continuous sway were revealed that fell within or above the confidence interval of the meta-analysis across conditions on at least one cardinal axis. The largest between group effect sizes were yielded when cutaneous cues to the support surface were manipulated, irrespective of visual cues to orientation. The strongest associations between continuous sway, reading discrepancy and hyperactivity/impulsivity self-rating scores were revealed on the vertical axis in the Cutaneous and Bimodal Modulation conditions.

Elevation of the COM on the vertical axis indicated use of the hip strategy to control balance in some individuals. This suggested that the individuals who were raising their heels and consequently elevating their centre of mass to control postural stability. It was predicted that these individuals were likely to have rated themselves highly on the Barkley Scale of hyperactivity/impulsivity symptoms. To test this hypothesis, *z* scores yielded from the acceleration of the hip and pelvic region of the body on the A/P axis were calculated. This was the outcome measure of efficiency of postural control following provocation of stability. Scores greater than 1 SD above the group mean were interpreted as greater acceleration of the body segment 6 hence less efficient control than the group average. Of the sample of 15 participants with dyslexia, 12 (80%) maintained the COM within the base of support efficiently. Three participants (20%) demonstrated inefficiency in maintaining postural stability of which one (6.7%) was consistently weak across all conditions. Again, these findings are more in keeping with those of Ramus et al. (2003) than Nicolson and Fawcett (1997). Furthermore, the same participant scored above the DSM-IV-*TR* clinical cut-off score for hyperactivity/impulsivity symptoms. The association between inefficient postural control and hyperactivity/impulsivity was either strong or moderate across conditions. This was interpreted as confirmation that inefficient postural control was therefore associated with hyperactivity/impulsivity symptoms in the dyslexia group. Examination of individual differences in standard deviation from the sample mean on measures of postural sway and hyperactivity/impulsivity

supported this conclusion. This strongly suggests that impaired postural stability in dyslexia arises from co-occurring symptoms of ADHD. Abnormality of the posterior inferior vermis of the cerebellum (see section 1.3.4), is one hypothesis to explain this relationship although causal attribution is beyond the scope of the thesis.

Individual postural stability across trials was examined by comparing the sway variability ($_{avg}SD$) of each participant. The dyslexia group performance across trials was more variable than the controls, on all axes, in all conditions. Once again the largest effect sizes were generated when standing on a compliant surface which was designed to obscure accurate perception of the support surface. These findings indicated that reducing sensory redundancy, by manipulating the CNS' reference frame hierarchy, increased variability in adaptive responses to provocation in some individuals with dyslexia across trials. Sway variability was moderately but none the less significantly associated with hyperactivity/impulsivity self-rating scores on at least one axis in the Visual Focus and Visual Modulation conditions and at least two axes in the Cutaneous and Bimodal Modulation conditions. The strongest associations were found between sway variability on the M/L axis when cutaneous cues to orientation were manipulated, irrespective of vision, and hyperactivity/impulsivity self-rating scores. This suggested that there was not only an association between continuous postural sway and hyperactivity/impulsivity scores across conditions, but also an association between variability in performance across trials and hyperactivity/impulsivity scores. Sway variability on the M/L axis also indicates unusual patterns of recovery at an individual level.

In common with dyslexia, ADHD is associated with heterogeneity of symptoms (Castellanos et al., 2005) and both inter and intra-individual variability of performance on measures of cognitive, attentional and motor function (Biederman, 2005). Castellanos et al. (2005) suggested that response variability associated with ADHD, known as 'ARV', is omnipresent in experimental samples and one characteristic that may be central to developing our understanding of the aetiology of ADHD. Variability may be therefore, a potential endophenotype for ADHD. They further suggest that because the diagnostic boundaries between developmental

disorders, in particular those in DSM-IV (American Psychiatric Society, 2000), are subjective and consequently fairly undependable, the pathological physiology and related processes that underpin intra-individual variability may transverse diagnostic categories. Performance variability in ADHD may therefore share the same causal mechanism as performance variability in dyslexia. Willcutt et al. (2005) have suggested that slowness and variability of responses on measures of temporal processing may be an endophenotype for co-occurring ADHD with dyslexia.

In the meta-analysis (Chapter 3) it was argued that balance deficits were more likely to be related to reading through a third variable, attention. The findings of the current study indicate that postural instability is associated with reading difficulties through either hyperactivity/impulsivity or combined inattention andhyperactivity/impulsivity symptoms. However, if reading and postural control are linked by a direct causal mechanism, some adults may have acquired compensatory balance strategies, in line with their development of compensatory reading strategies, that our paradigm was unable to penetrate. It is possible that these strategies may only be threatened in the presence of co-existing attention deficits in adults. In order to better understand the nature of the relationship between reading and balance and consequently evaluate whether measures of postural stability are valid predictors of reading difficulty, these skills need to be assessed before a level of reading compensation has been achieved.

6.5 Conclusions

The study satisfied the sample classification, procedure and measurement criteria raised by the meta-analysis in Chapter 3. Sensory modulation increased postural sway and variability of sway across trials for 20% of participants with dyslexia who consistently demonstrated greater effort in maintaining stability than their peers. These participants also generated self-rating hyperactivity/impulsivity scores that were borderline or in excess of clinical cut-off norms. The results dovetail with the findings of the meta-analysis, that postural instability is not specifically related to reading but is associated through a third variable, hyperactivity/impulsivity or combined inattention and hyperactivity/impulsivity symptoms. The most salient hypothesis at this point is that this may indicate abnormality of the posterior inferior

vermis of the cerebellum, although investigation of this is beyond the scope of the thesis. This chapter has revealed that postural stability is only weakly associated with reading ability. However, some adults may have been adequately compensating for the effects of manipulating the sensory frame of reference hierarchy, which may be exacerbated by co-existing hyperactivity/impulsivity symptoms in dyslexia. The study reported in Chapter 7 therefore, examines the relationship between balance and reading among children with dyslexia.

7 Postural stability in children with dyslexia

7.1 Introduction

Chapter 5 concluded with the prediction that some adults with dyslexia had developed compensatory balance strategies in line with their compensatory reading strategies that would only be threatened in the presence of co-existing attention deficits at an individual level. Would younger readers with dyslexia who have not yet fully learned compensatory reading strategies therefore, be more likely to demonstrate postural instability than compensated adults? The two largest effect sizes revealed in the meta-analysis (d =1.42, 1.17) were generated by studies of children (Moe-Nilssen et al., 2003; Sheffield Research Group, 1992-2001). Furthermore, if motor control deficits are related to reading difficulties by a shared causal mechanism (Nicolson and Fawcett, 1999) then they would be less likely to be remediated through the course of development than by the course of improvement in normal reading. It could therefore be predicted that readers who have not yet developed compensatory strategies in their reading would demonstrate postural instability irrespective of their chronological age. Similarly, children with normal reading development would be expected to demonstrate normal development of postural stability, which typically reaches maturity between the ages of 7 and 10 years (Shumway-Cook and Woollacott, 2001). Alternatively, the absence of balance difficulties in a sample of older children with persistent reading difficulties would falsify the hypothesis of a causal link between reading and balance.

Fawcett and Nicolson (1999) reported a d effect size of 2.17 for postural stability between children with dyslexia and control children of similar chronological age. This effect size was calculated based on the mean and standard deviation of the control group (i.e., dyslexia group mean-control group mean/ control group SD). Based upon this statistic five participants per group would be needed to obtain a statistical power of 80% at alpha level 0.05. The combined d effect size for the Sheffield Research Group calculated in the meta-analysis in Chapter 2, using Cohen's pooled variance method, was a smaller 1.17. Based on this statistic 11 participants would be required per group to obtain a statistical power of 80% at alpha

level 0.05. In order to compare the findings of the meta-analysis with this study and to further assess the validity of using balance measures to predict reading skills, an otherwise unselected sample of children was recruited and administered measures of non-verbal reasoning, literacy and recent behavioural symptoms, in addition to the DST Postural Stability Component and the provocation test paradigm.

The study described in this chapter compared three groups of children on measures of balance, literacy, inattention and hyperactivity/impulsivity symptoms to assess the relationship between balance and reading in children. The comparison groups were primary school aged children with dyslexia, older children and adolescents with dyslexia and mainstream primary school children with normal reading development. The younger children with dyslexia were first compared with the control children who were within the same age range. In this way the question of whether the children with dyslexia demonstrated weaker postural stability than a normally reading peer group was assessed. The two dyslexia groups were next compared in order to assess whether balance difficulties persisted in the elder group or were remediated through development and therefore potentially related to chronological age. The elder dyslexia group was then compared with the younger control group that were predicted to have achieved a similar level of reading ability. Impaired postural stability compared with control children at a similar level of reading achievement would indicate developmental disorder as opposed to developmental delay (Bryant and Goswami, 1986).

The Sheffield Research Group (1990 to 2001) had compared groups of children aged 8 years, 12 years and 16 years. In the current study the opportunity sample was divided more in accordance with typical stages of postural and balance development (Shumway-Cook and Woollacott, 2001). A group of primary school children with typically developing reading skills, of an age when the postural control system would be expected to be approaching maturity (above 7 years) was first recruited. Then a second group of similarly aged primary school children with dyslexia and a third group of older children with dyslexia aged 12 to 16 years, when the postural control system would be expected to be mature, were also recruited. (I would like to

acknowledge and thank the head teacher, staff, parents and pupils of St Francis Church of England Primary School, Birmingham, UK, and the parents and children recruited from the Aston University Dyslexia and Developmental Assessment Centre for their assistance with this study).

7.2 Method

In previous studies a strong association was revealed between the sway paths of body segment 2 and the COM in the Visual Focus condition ($r_s = 0.84$, $p < 0.01$) therefore the number of retro-reflective markers used to track motion was reduced to five and these were handed to the child on adhesive Velcro to place above their clothing as directed.

7.2.1 Participant characteristics

Following a wide dissemination of letters to primary schools within the Birmingham, UK Local Education Authority (LEA), a one-form entry mainstream primary school agreed to assist with the recruitment of participants for the control group and to provide accommodation for testing. A comprehensive analysis of the literacy and balance abilities of this sample is presented in Chapter 8. Parental consent was received for 15 participants who completed all measures. There were 8 girls and 7 boys (mean age 9 years 9 months, range 8 years 3 months to 11 years 2 months). They were administered the Matrix Reasoning subtest of the WASI and the WORD Basic Reading and Spelling tests described in sections 4.1.2.3 and 4.1.2.4 (Wechsler, 1993; 1999). Teachers were asked to complete the inattention and hyperactivity/impulsivity sections of the Barkley Disruptive Behaviour Rating Scale Teacher Form for each child (Barkley and Murphy, 1998). This Scale is usually administered by teachers who have regular periods of contact with children as an assessment of their pupil's behaviour over the previous 6 months. In this study, teachers had observed their pupils' behaviour in the classroom for approximately 30 hours per week over 3 months. All children were assessed on the school premises.

For the groups of children with dyslexia, thirty children were recruited from the Aston University Dyslexia and Developmental Assessment Centre. These participants had participated in an ongoing collaborative investigation of the genetic basis of dyslexia with the Oxford University Laboratory of Physiology and the Wellcome Trust Centre for Human Genetics. The balance measures required for this study were administered in addition to the psychometric and literacy measures specified in sections 4.1.2.3. and 4.1.2.4. The younger group comprised 11 children, 2 girls and 9 boys (mean age 10 years 4 months, range 8 years 3 months to 11 years 6 months). The elder group comprised 19 children and adolescents, 7 girls and 12 boys (mean age 14 years 1 month, range 12 years 2 months to 16 years 5 months). These groups completed the Matrices, Single Word Reading and Single Word Spelling subtests of the BAS-II (Elliott et al., 1998). For these groups, parents completed the Barkley Disruptive Behaviour Scale Parent Form (Barkley and Murphy, 1998). These children were assessed on the Aston University campus in a clinical laboratory.

Weight and height measurements were recorded for all participants. The control group was assessed in November 2004 and the dyslexia groups were assessed during the Spring half-term and Easter school holidays 2005. Due to the difference in psychometric measures administered to the groups (section 4.1.2.3), FSIQ scores for the dyslexia groups were not obtained. However, a well documented history of reading and IQ was available for each participant from their assessment centre records. Access to these was obtained following written parental consent.

7.2.2 Instrumentation

Motion capture was described in section 4.2.3. Five 19 mm retro-reflective markers were used and attached to the child's clothing with adhesive Velcro. Markers were placed above the right clavicle, left clavicle, xiphoid process and the head of the talus of the right and left foot. Data were sampled at 120 Hz.

7.2.3 Procedure

The DST-J Postural Stability component was administered and scored in accordance with the test manual described in section 4.1.3 (Fawcett and Nicolson, 2004a). Children up to 11 years 6 months were administered a manually calibrated force of 2.5 kg; children older than 11 years 6 months were administered a force of 3 kg. The provocation test was then administered to assess postural stability. For this study a portable version of the perturbation rig, was constructed as shown in Figure 7:1. The base of the rig was weighted to guarantee stability.

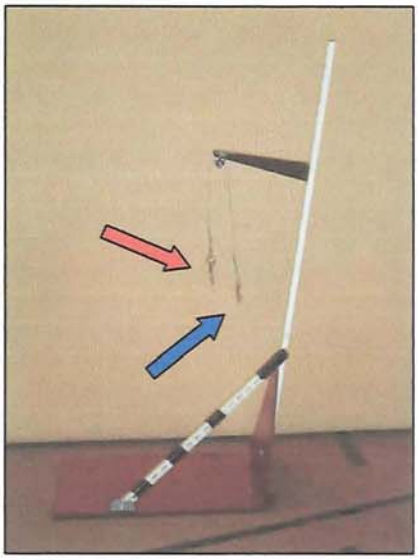

Figure 7:1 Portable provocation rig used in the assessment of postural stability in children. The child stood with feet 0.01m apart with heels touching the front of the base. The red arrow depicts the end of the drag line that was attached to a belt worn around the child's waist. The blue arrow depicts the end of the drag line that was attached to the weight suspended behind the child.

As for the adult studies, an individually calibrated weight, equivalent to 5% of body mass from the rig, was suspended using a dragline and pulley, and attached it to a belt worn by the child. Children were asked to remove their shoes and stood with their feet 0.01m apart in front of the rig so that the weight was suspended behind them. This was to ensure that when the weight was released the perturbation force was delivered to the same body segment and on the same axis as when the DST-J Postural Stability instrument was used. In this study the aim was to closely replicate the DST metric. The weight was therefore released and postural sway was measured

twice with the arms relaxed at the sides of the body and twice with the arms raised in parallel in front of the body. Stability was tested in both the Visual Focus and Visual Modulation conditions. In each condition the child was told that the weight would be released, but not when, and that they should try to stand as still as possible. The weight was released, reattached and re-released to deliver four trials in each condition to replicate the DST Postural Stability component.

7.2.4 Analysis

The frame of release was identified by tracking the suspended weight marker on the vertical axis (see Figure 5:4). A 240 frame window of data was exported and filtered as described in section 5.2.2.4. The location of Segment 2 COM was calculated on each cardinal axis to assess trunk sway. The location of the foot markers was averaged on each cardinal axis as motion of the feet in the DST is rated from 2 (heels raised) to 6 (several steps forwards). No distinction is made between either foot. The point by point ensemble average and the curve average variability were calculated across trials for each participant. The continuous measures of postural sway comprised the standard deviation (SD) of the sway path of Segment 2 on the A/P and M/L axes and of Segments 9/10 on the vertical axis, and the curve average variability across trials. Descriptive and inferential statistics for all measures were computed in SPSS 11.5.

7.3 ***Results***

7.3.1 Sample characteristics

Table 7:1 shows a summary of the descriptive statistics of the anthropometric, psychometric and literacy measures for the three groups.

Table 7:1 Descriptive statistics of sample characteristics (mean ± standard deviation).

	Age months	Height m	Weight kg	Weight of drag kg	Non-verbal reasoning[1] T score	Reading T score[2]	Spelling T score[2]
1. control group	117.27	1.42	37.57	1.88	59.67	58.87	59.60
$N = 15$	±	±	±	±	±	±	±
	13.01	0.08	6.03	0.30	8.32	8.79	8.71
2. younger	123.63	1.54	42.84	2.13	52.91	45.64	40.70
dyslexia group	±	±	±	±	±	±	±
$N = 11$	14.09	0.11	20.44	1.04	10.97	6.05	6.06
3. elder	168.74	1.65	58.11	2.90	48.42	38.56	36.00
dyslexia group	±	±	±	±	±	±	±
$N = 19$	13.0	0.09	11.84	0.56	9.34	9.99	7.87

[1]WASI Matrix Reasoning subtest was administered to the control group and the BAS Matrices subtest to the dyslexia groups.
[2]Standard scores were converted to T scores (mean =50, SD=10) for comparison.

7.3.2 Anthropometric measures

The descriptive statistics show the expected height and weight increases associated with differences in chronological age between the younger and elder groups. A one-way independent measures ANOVA confirmed a significant age difference among the groups (F [2, 42] = 74.89, $p < 0.001$). Levene's test revealed that equal variances could be assumed. Post-hoc comparisons using Scheffe's test confirmed that the difference in age between the young control group and the elder dyslexia group and between the dyslexia groups was significant ($p < 0.001$) but that between the two younger groups was not significant. This showed that the control group was a suitable chronological age comparison group for the younger dyslexia group. The same statistical tests were applied to recorded weight (F [2, 42] = 11.24, $p < 0.001$), and height (F [2, 42] = 31.77, $p < 0.001$). Scheffe's test confirmed significant differences between the control group and the elder dyslexia group, and between the 2 dyslexia groups on both measures associated with alpha level 0.001. These findings confirmed that weight and height differences among the groups were in keeping with typical maturation, although there was greater variability of body weight in the younger dyslexia group than within the other two groups.

Examination of the descriptive statistics for the weight of the drag used in the provocation test showed that the average weight used for the two younger groups

was below the 2.5 kg force used for all children in the DST metric. The average weight used for the elder dyslexia group was just below the 3 kg force associated with the DST. The drag weight differed significantly across groups (F [2, 42] = 11.07, $p < 0.001$). Scheffe's test demonstrated a significant difference between the control group and the elder dyslexia group ($p < 0.001$) and between the two dyslexia groups ($p = 0.14$). A non-significant difference was found between the two younger groups ($p = 0.69$). This confirmed that our criterion for individually calibrating the weight according to postural research conventions, as prescribed by Moe-Nilssen et al. (2003), had been met and that the two younger groups would be suitable for comparison in terms of both chronological age and anthropometric variables.

7.3.3 Non-verbal Reasoning measures

Descriptive statistics showed that the elder dyslexia group had a lower average T score on the measure of non-verbal reasoning than either of the younger groups. A one-way ANOVA revealed significant differences among groups (F [2, 42] = 5.96, $p < 0.005$). A post hoc comparison using Scheffe's test revealed that only the control group and the elder dyslexia group differed significantly ($p < 0.005$). This indicated that the dyslexia groups included children with weaker non-verbal ability than the control group.

7.3.4 Literacy measures

Examination of the descriptive statistics revealed that the mean scores of the control group were clearly higher than either of the two dyslexia groups on measures of reading and spelling. For reading, a one-way ANOVA revealed a significant difference among the groups (F [2, 42] = 22.57, $p < 0.001$). Scheffe's test comparison revealed significant differences between the control group and both the younger and elder dyslexia groups ($p < 0.005$; $p < 0.001$). However, a non-significant difference was found between the two dyslexia groups ($p = 0.12$) because reading scores are standardised by chronological age. These descriptive and inferential statistics suggested that the elder dyslexia group were more impaired in reading for their age and might therefore be predicted to demonstrate a greater degree of postural instability in the event of a causal link between balance and reading. The

literacy discrepancy between the control and dyslexia groups was supported by the results of the spelling measures. A one-way ANOVA showed a significant difference among groups (F [2, 42] = 40.71, $p < 0.001$) with significant differences between the control group and both dyslexia groups ($p < 0.001$; $p < 0.001$) and a non-significant difference between the dyslexia groups ($p = 0.28$). This indicated that there was a degree of overlap in literacy ability between the dyslexia groups arising from the greater variability among the reading and spelling scores demonstrated by the elder group.

7.3.5 Attention measures

Figures 7:2 and 7:3 show the distribution of rating scores around the median score on the Barkley Disruptive Behaviour Scale.

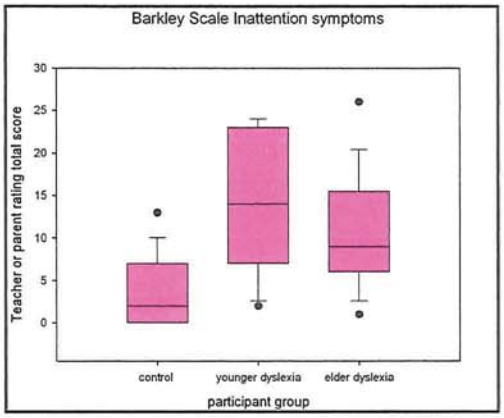

Figure 7:2 Barkley Disruptive Behaviour Ratings: Inattention symptoms.
Boxplots show the distribution of teacher or parent ratings around the median score. The clinical cut-off scores for ratings of inattention symptoms at ages 8-10 years for teacher ratings are: 24 (boys) and 21 (girls); for parent ratings: 15 (boys) and 12 (girls). At ages 11-13 years cut-off scores for teacher ratings are: 24 (boys) and 19 (girls); parent ratings are: 18.5 (boys) and 12.8 (girls). The maximum score achievable is 27.

Rating scores 0-3 are summed over 9 measures of inattention and 9 of hyperactivity/impulsivity symptoms, the maximum achievable score is 27. None of the children in the control group were rated above the clinical cut-off score on either Scale. Five children in the younger dyslexia group obtained parental ratings in excess of the clinical cut-off score for inattention symptoms. Of these, two were also rated above the cut-off score for hyperactivity/impulsivity symptoms. One parent declined

to complete the forms for two siblings in the elder dyslexia group. Five members of this group were rated in excess of the cut-off score for inattention symptoms however no scores exceeded the cut-off score for hyperactivity/impulsivity.

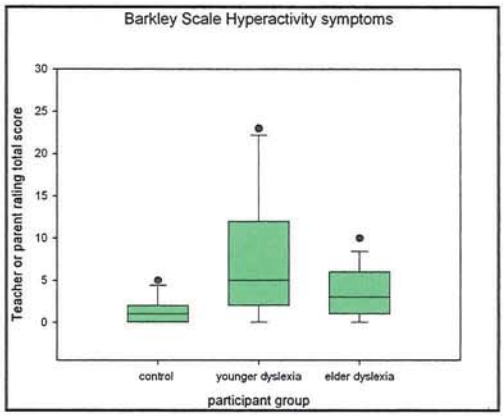

Figure 7:3 Barkley Disruptive Behaviour Ratings: Hyperactivity symptoms.
Boxplots show the distribution of teacher or parent ratings around the median score. The clinical cut-off scores for ratings of hyperactivity/impulsivity symptoms at ages 8-10 years for teacher ratings are: 22 (boys) and 16.7 (girls); for parent ratings: 15 (boys) and 9 (girls). At ages 11-13 years cut-off scores for teacher ratings are: 18 (boys) and 14.8 (girls); parent ratings are: 16 (boys) and 9 (girls). The maximum score achievable is 27.

A Kruskal-Wallis non-parametric independent samples test (H converted to χ^2 in SPSS) revealed significant differences in both inattention (χ^2 [2, $N = 43$] = 15.20, $p <$ 0.001) and hyperactivity/impulsivity scores (χ^2 [2, N = 43] = 11.22, $p < 0.005$) among groups. To examine the differences between specific groups, non-parametric tests were used. A Mann-Whitney U of 19.5 (z = -3.30, $p < 0.005$) for inattention symptoms and 29 (z = -2.84, $p < 0.005$) for hyperactivity symptoms between the control group and the younger dyslexia group showed that the latter group included children with co-existing symptoms of inattention and hyperactivity/impulsivity. The same statistical test was applied to compare the control group with the elder dyslexia group, which yielded a Mann-Whitney U of 40.0 (z = -3.32, $p < 0.005$) for inattention symptoms and 58.0 (z = -2.68, $p < 0.005$) for hyperactivity/impulsivity symptoms. This showed that the elder group also included children with co-existing attention deficits. Comparison of the two dyslexia groups yielded a Mann-Whitney U of 77.0 (z = -0.78, p = 0.22) for inattention symptoms and 64.5 (z = -1.37, p = 0.08) for hyperactivity symptoms, which were not significant. The findings indicated that

both dyslexia groups included children with co-existing inattention and hyperactivity/impulsivity symptoms.

7.3.6 DST Postural Stability Component

Figure 7:4 shows the distribution of total scores around the median score, out of a maximum 24, on the DST Postural Stability component. The median score of the control group was 2 (range 1 to 6). The median score of the younger dyslexia group was 5 (range 2 to 9). The median score of the elder dyslexia group was 4 (range 1 to 8). A Kruskal-Wallis test revealed that scores among groups (χ^2 [2, N = 45] = 12.32, p <0.005) were significantly different. To examine specific differences between groups, non-parametric independent samples tests were used. A Mann-Whitney U of 20.5 (z = -3.34, p < 0.001) revealed a significant difference between the control group and the younger dyslexia group. A Mann-Whitney U of 77.5 (z = -2.34, p < 0.05) also revealed a significant difference between the control group with the elder dyslexia group. The difference between the dyslexia groups bordered on significance (U = 66.5, z = -1.67, p = 0.05). These results indicated that both dyslexia groups demonstrated a greater degree of postural instability than the control group. However, the interquartile range indicated that scores between the 25th and 75th percentile were more variable in the elder dyslexia group.

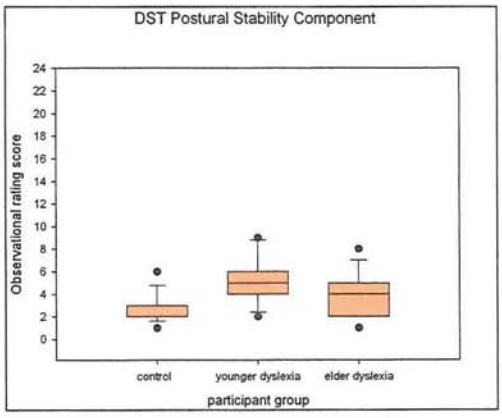

Figure 7:4 DST Postural Stability Component scores.
Boxplots show the DST Postural Stability component observational rating scores.

7.3.7 Provocation test measures: continuous postural sway

Figure 7:5 shows continuous postural sway following provocation in the Visual Focus condition on the A/P axis. The control group (mean 9.46mm ± SD 9.86mm) was influenced by extreme values generated by a minority of individuals. The younger (mean 8.29mm ± SD 4.16mm) and elder (mean 9.09mm ± SD 6.95mm) dyslexia groups scores were within a smaller distribution of their group mean than the control group. A one-way ANOVA revealed no significant differences among group means (F [2, 42] = 0.08, p = 0.93). Effect sizes between groups were calculated for continuous postural sway, as shown in Table 7:2. Effect sizes were small in the Visual Focus condition on the A/P axis.

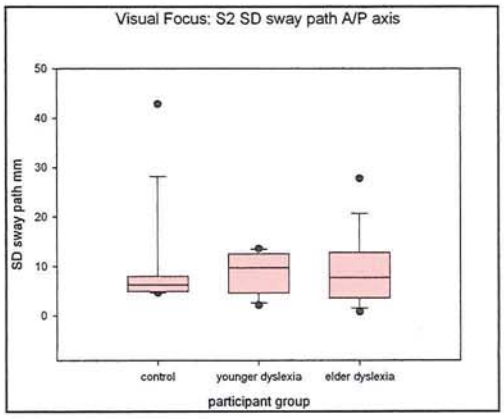

Figure 7:5 Postural sway on the A/P axis in the Visual Focus condition.
Boxplots show the SD of the sway path of segment 2 on the A/P axis in the Visual Focus condition.

Continuous postural sway on the M/L axis is shown in Figure 7:6. Sway values were small for the control group (mean 2.78mm ± SD 1.92mm) and both younger (mean 6.40mm ± SD 5.95mm) and elder dyslexia groups (mean 4.01mm ± SD 3.69mm). A one-way ANOVA revealed a significant difference among the groups (F [2, 42] = 3.62, $p < 0.05$). Tamhane's T2 test was used for post hoc comparison. Specific between groups differences were not significant. Effect sizes between the younger dyslexia group and both the control and elder dyslexia groups however, were large. This suggested that the sample size was too small to achieve alpha level 0.05.

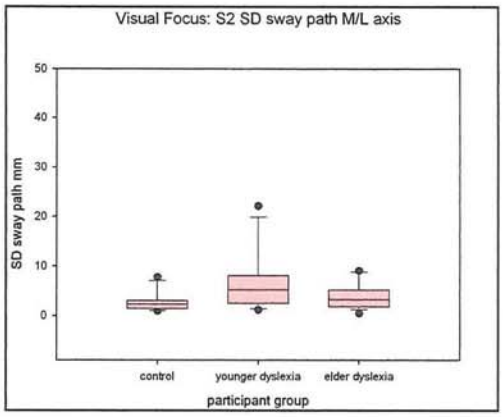

Figure 7:6 Postural sway on the M/L axis in the Visual Focus condition.
Boxplots show the SD of the sway path of segment 2 on the M/L axis in the Visual Focus condition.

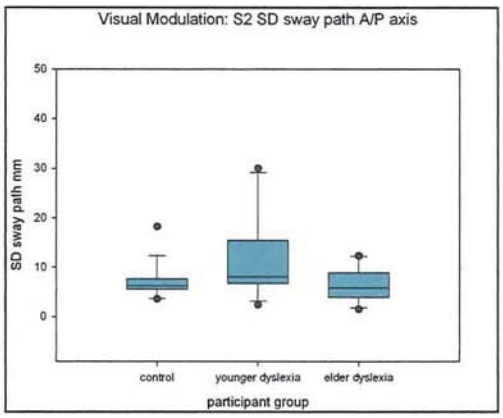

Figure 7:7 Postural sway on the A/P axis in the Visual Modulation condition.
The boxplots show the SD of the sway path of segment 2 on the A/P axis in the Visual Modulation condition.

Continuous postural sway on the A/P axis in the Visual Modulation condition is shown in Figure 7:7. The younger dyslexia group (mean 12.38mm ± SD 8.66m) demonstrated a wider distribution of sway values than either the control group (mean 6.88mm ± SD 3.46mm) or the elder dyslexia group (mean 6.40mm ± SD 4.66mm). This measure differed significantly among groups (F [2, 42] = 5.15, p <0.01). A Tamhane's T2 test was used for post hoc comparison however between groups differences were not significant. Again, effect sizes between the younger dyslexia group and both the control and elder dyslexia groups were large (see Table 7:2). This suggested that the sample size was too small to achieve an alpha level of 0.05.

Table 7:2 Continuous postural sway and postural sway variability effect sizes between groups.

	Participant comparison groups	between groups effect size d [1]	Participant comparison groups	between groups effect size d [1]	Participant comparison groups	between groups effect size d [1]
Segment 2 SD sway[2] A/P axis VF[3]	control vs younger dyslexia	0.15	control vs elder dyslexia	0.04	younger vs elder dyslexia	0.13
Segment 2 SD sway M/L axis VF	control vs younger dyslexia	0.87	control vs elder dyslexia	0.38	younger vs elder dyslexia	0.68
Segment 9/10 SD sway vertical axis VF	control vs younger dyslexia	0.40	control vs elder dyslexia	0.01	younger vs elder dyslexia	0.37
Segment 2 SD sway A/P axis VM[3]	control vs younger dyslexia	0.88	control vs elder dyslexia	0.14	younger vs elder dyslexia	1.24
Segment 2 SD sway M/L axis VM	control vs younger dyslexia	0.89	control vs elder dyslexia	0.39	younger vs elder dyslexia	0.68
Segment 9/10 SD sway vertical axis VM	control vs younger dyslexia	0.84	control vs elder dyslexia	0.63	younger vs elder dyslexia	0.64
Segment 2 $_{avg}$SD sway[4] A/P axis VF[3]	control vs younger dyslexia	0.08	control vs elder dyslexia	0.27	younger vs elder dyslexia	0.61
Segment 2 $_{avg}$SD sway M/L axis VF	control vs younger dyslexia	1.06	control vs elder dyslexia	0.35	younger vs elder dyslexia	0.98
Segment 9/10 $_{avg}$SD sway vertical axis VF	control vs younger dyslexia	0.03	control vs elder dyslexia	0.13	younger vs elder dyslexia	0.15
Segment 2 $_{avg}$SD sway A/P axis VM[3]	control vs younger dyslexia	0.27	control vs elder dyslexia	0.19	younger vs elder dyslexia	0.30
Segment 2 $_{avg}$SD sway M/L axis VM	control vs younger dyslexia	0.96	control vs elder dyslexia	0.45	younger vs elder dyslexia	0.62
Segment 9/10 $_{avg}$SD sway vertical axis VM	control vs younger dyslexia	0.44	control vs elder dyslexia	0.58	younger vs elder dyslexia	0.78

[1] d effect size
[2] standard deviation of the ensemble average displacement during 2 seconds post provocation [continuous postural sway].
[3] VF = Visual Focus; VM = Visual Modulation.
[4] average of the point by point standard deviation, during 2 seconds post provocation, across trials [postural sway variability].

Continuous postural sway on the M/L axis is shown in Figure 7:8. Sway values were small for the control group (mean 2.15mm ± SD 0.68mm) and both the younger (mean 5.57mm ± SD 5.83mm) and elder (mean 2.82mm ± 2.17mm) dyslexia groups. A one-way ANOVA revealed a significant difference among groups (F [2, 42] = 3.95, p <0.05). Tamhane's T2 test was used for post hoc comparison, however between group differences were not significant. Effect sizes between the younger dyslexia group and both the control and elder dyslexia groups were large (see Table 7:2). Again, the elder dyslexia group demonstrated postural stability closer to the control group than the younger dyslexia group.

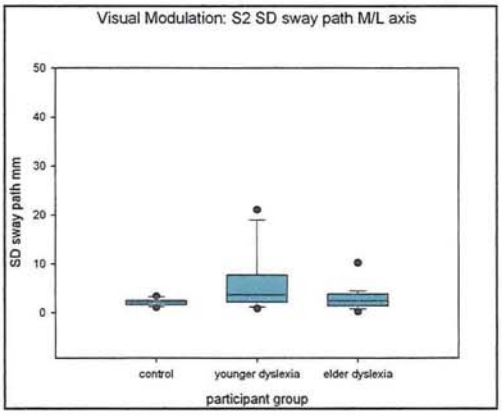

Figure 7:8 Postural sway on the M/L axis in the Visual Modulation condition.
Boxplots show the SD of the sway path of segment 2 on the M/L axis in the Visual Modulation condition.

Finally, continuous sway of the foot markers (S9&10) on the vertical axis in the Visual Focus condition is shown in Figure 7:9. Sway values were small for the control group (mean 0.73mm ± SD 0.64mm) and both the younger (mean 1.01mm ± SD 0.78mm) and elder (mean 0.72mm ± SD 0.88mm) dyslexia groups. A one-way ANOVA revealed between group differences that were not significant (F [2, 42] = 0.57, p = 0.57). Effect sizes between groups were small (see Table 7:2) which suggested that the groups demonstrated similar patterns of heel-raising to maintain stability.

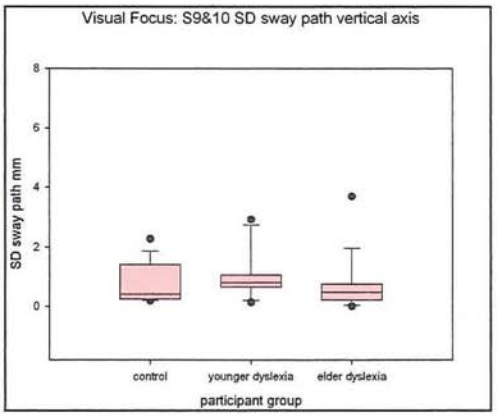

Figure 7:9 Postural sway on the vertical axis in the Visual Focus condition.
Boxplots show the SD of the sway path of segments 9&10 on the vertical axis in the Visual Focus condition.

Figure 7:10 shows continuous sway on the vertical axis in the Visual Modulation condition. The sway values were again small for the control group (0.30mm ± SD 0.48mm) and both younger (mean 1.86mm ± SD 2.8mm) and elder (0.71mm ± SD 0.75mm) dyslexia groups. No significant differences between groups were revealed by ANOVA. Effect sizes between all three groups were large however. This indicated that the elder dyslexia group demonstrated less heel-raising to maintaining stability than either of the two younger groups (see Table 7:2).

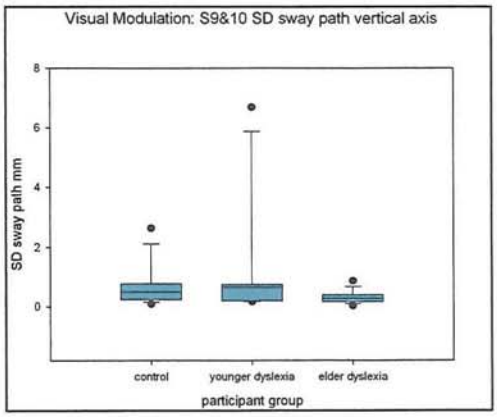

Figure 7:10 Postural sway on the vertical axis in the Visual Modulation condition.
Boxplots show the SD of the sway path of segments 9&10 on the vertical axis in the Visual Modulation condition.

In summary, the findings indicated that the elder dyslexia group have postural control systems that share more similarities with the younger control group than the younger dyslexia group.

7.3.8 Provocation test measures: postural sway variability

To assess individual variability of sway across trials the curve average standard deviation ($_{avg}$SD) was calculated for each participant, on each axis and in each condition. This also measured the effect of raising the arms in parallel on postural stability for 2 perturbations. There were significant differences among groups on the M/L axis only in the Visual Focus (F [2, 42] = 6.58, p <0.005) and the Visual Modulation (F [2, 42] = 4.65, p <0.05) conditions. Tamhane's T2 post hoc test revealed no significant differences between groups. Table 7:2 also shows between group effect sizes that were calculated to assess the size of differences in sway variability among groups. Effect sizes between the younger dyslexia group and both the control group and the elder dyslexia group were large in both conditions. This suggested that raising the arms in the provocation test generated a more variable adaptive response in the frontal plane in the younger dyslexia group. Maintenance of postural stability was revealed to be more consistent across groups in the sagittal plane in both conditions, suggesting that performance was either consistently good or consistently poor.

7.3.9 Within-subjects assessment of the effects of modulating vision

To assess the effects of modulating vision on postural stability paired samples t- tests were performed between SD sway values in the Visual Focus and Visual Modulations on 3 axes. Comparison of sway values revealed non-significant effects of modulating vision on the A/P axis (t [44] = 1.25, *ns*) on the M/L axis (t [44] = 0.97, *ns*) and on the vertical axis (t [44] = 0.67, *ns*). This indicated that postural stability was little affected by visual manipulation. Moe-Nilssen et al. (2003) suggested that some children with dyslexia were not making use of visual cues to orientation, therefore the Romberg quotient was calculated to assess the contribution of vision to postural stability in this sample (e.g., Hayes and Riach, 1989). This measure represents the Visual Modulation (no vision) continuous sway score

expressed as a percentage of the Visual Focus (vision) continuous sway score (Shumway-Cook and Woollacott, 2001). Table 7:3 shows the descriptive statistics for the groups. Between group differences were not significant on the A/P axis (F [2,42] = 2.68, *ns*), the M/L axis (F [2,42] = 0.11, *ns*) and the vertical axis (F [2,42] = 0.02, *ns*). There was however, greater variability in the contribution of vision to postural stability within the dyslexia groups than the control group on each axis.

Table 7:3 The Romberg quotient representation of the contribution of vision to balance

	A/P axis	M/L axis	Vertical axis
control group	89.51%[a] ± 26.00%	108.01% ± 75.89%	140.52% ± 161.11%
younger dyslexia group	185.72%[b] ± 159.00%	104.71% ± 74.47%	147.77% ± 258.63%
elder dyslexia group	123.63% ± 105.90%	120.22% ± 122.82%	161.22% ± 361.24

[a]Scores under 100 indicate greater magnitude of sway when visual cues to orientation are available.
[b]Scores over 100 indicate greater magnitude of sway when visual cues to orientation are removed.

7.3.10 Correlation analyses

In view of the various levels of data and distributions of scores generated by the measures non-parametric correlations were performed among the psychometric, literacy, attention and balance data. No correction was performed for multiple comparisons in these analyses. Correlations with an associated alpha level 0.01 (1-tailed) were adjudged significant, as shown in Table 7:4. Provocation measures of postural stability correlated moderately and significantly with the DST measure of postural stability. This confirms the findings of Chapter 4, that the provocation test and the DST metric tapped the same underlying abilities. The DST metric was also significantly associated with literacy and inattention scores. Associations between literacy scores and the provocation measures of continuous postural sway however were weak. The association between reading and postural stability is therefore stronger for the DST metric than for the provocation measure.

The strongest association revealed between postural sway values and reading scores was on the M/L axis in the Visual Focus condition. However, this sway measure was also more strongly associated with Barkley Inattention and Hyperactivity ratings.

Furthermore, it yielded the largest effect sizes between the younger dyslexia group and each of the other groups in the Visual Focus condition. Section 7.3.5 demonstrated that the younger dyslexia group included children with co-existing symptoms of inattention and hyperactivity. Postural sway on the vertical axis in the Visual Modulation condition moderately correlated with age. The effect sizes yielded by this measure between the elder dyslexia group and each of the younger groups were equal in magnitude. These findings supported the prediction that the younger and not the elder children were raising their heels to maintain stability.

Table 7:4 Non-parametric correlations between psychometric, literacy, behavioural and postural stability measures.

		BAS-II/WASI/WORD[a]			Barkley		DST
	Age	Non-verbal Reasoning	Reading	Spelling	Inattention	Hyperactivity	Postural Stability
SD sway path							
VF[b] A/P	0.03	0.02	-0.04	-0.08	0.23	0.17	0.35*
VF M/L	-0.19	-0.19	-0.24	-0.29	0.40**	0.39**	0.41**
VF Vertical	-0.27*	-0.09	0.05	-0.04	0.29*	0.20	0.25*
VM A/P	-0.10	0.10	-0.06	0.03	0.01	0.21	0.35*
VM M/L	-0.09	-0.13	-0.16	-0.09	0.26*	0.23	0.39**
VM Vertical	-0.39**	0.14	0.14	0.12	0.23	0.03	0.25*
Age	1.00	-0.32*	-0.63**	-0.66**	0.20	0.13	0.16
Non-verbal Reasoning	-	1.00	0.56**	0.58**	0.51**	-0.19	0.25*
Reading	-	-	1.00	0.91**	-0.51**	-0.34*	0.50**
Spelling	-	-	-	1.00	0.59**	-0.41**	0.44**
Barkley Inattention	-	-	-	-	1.00	0.38**	0.38**
Barkley Hyperactivity	-	-	-	-	-	1.00	0.14
DST Postural Stability	-	-	-	-	-	-	1.00

[a]See sections 4.1.2.3. and 4.1.2.4.
[b]VF = Visual Focus, VM = Visual Modulation.
*$p<0.05$
**$p<0.01$ $N = 35$, critical value = 0.35.

7.3.11 Individual performance profiles

Continuous postural sway values were converted to standardized *z* scores to identify individual differences in postural stability across measures. Figure 7.11 shows the stacked *z* scores for continuous sway of body segment 2 on the A/P and M/L axes and of body segments 9&10 on the vertical axis in the Visual Focus condition. Individual differences in postural stability were apparent in each group.

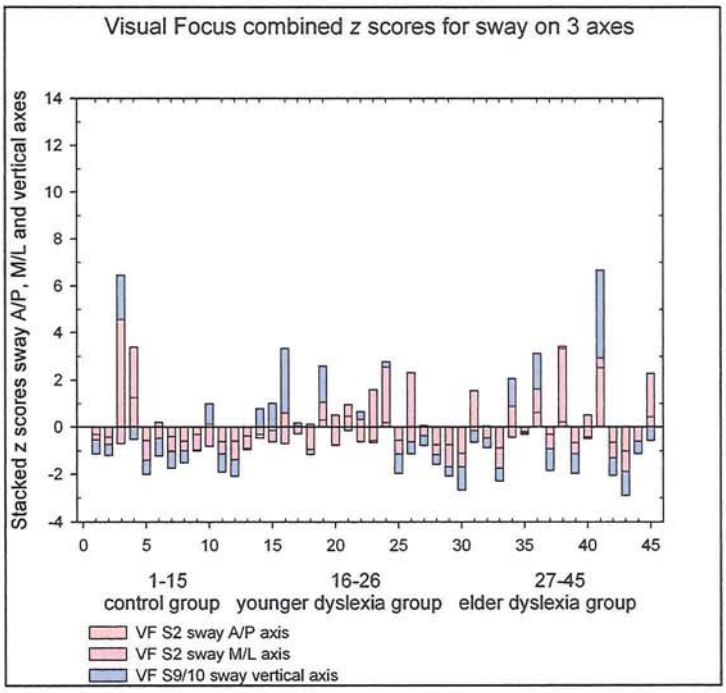

Figure 7:11 Chart demonstrating stacked *z* scores for Visual Focus postural sway on 3 axes. Each coloured block represents the standardised value (*z* score) of postural sway averaged across trials for one participant on one axis. Baseline (0) represents the sample mean. Where individual *z* scores across axes consistently fall either above or below the mean they are shown as unidirectional, cumulative stacks. Where scores across axes are mixed (i.e., ± the sample mean) stacks intersect the baseline. Stacks above the baseline represent scores above the mean. Stacks below the baseline represent scores below the mean.

Figure 7.12 shows the stacked *z* scores for continuous sway on 3 axes in the Visual Modulation condition. Again, individual differences in postural stability were evident in each group.

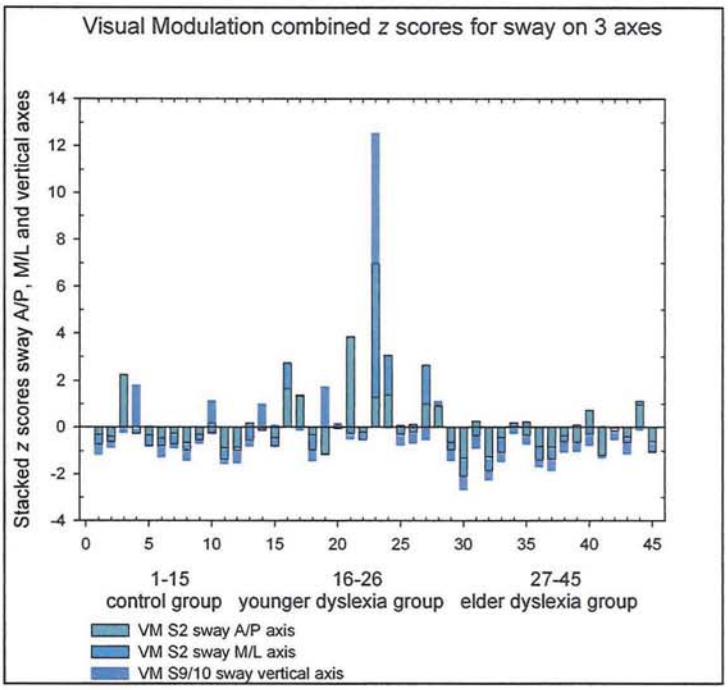

Figure 7:12 Chart demonstrating stacked *z* scores for Visual Modulation postural sway 3 axes. Each coloured block represents the standardised value (*z* score) of postural sway averaged across trials for one participant on one axis. Baseline (0) represents the sample mean. Where individual *z* scores across axes consistently fall either above or below the mean they are shown as unidirectional, cumulative stacks. Where scores across axes are mixed (i.e., ± the sample mean) stacks intersect the baseline. Stacks above the baseline represent scores above the mean. Stacks below the baseline represent scores below the mean.

The scores for reading, inattention and hyperactivity/impulsivity symptoms were also standardized for comparison across the sample. Of the 15 control participants, one generated a postural sway *z* score in excess of 1.96 SD of the sample mean on at least one axis in the Visual Focus condition. This child demonstrated reading, inattention and hyperactivity scores within 1 SD of the sample mean. Of the younger dyslexia group, YD1, YD6, YD8, and YD9 generated sway *z* scores greater than 1.96 SD above the sample mean in at least one condition. All four presented *z* reading scores within 1 SD of the sample mean. Two of these children, YD1 and YD9 were rated 3 SD above the mean on both the inattention and the hyperactivity scales and exceeded the clinical cut-off scores (see section 7.2.2.5). Behaviour scores for YD6 and YD8 were within 1 SD of the mean, however YD6 was found to have a history of motor

difficulties from birth that continued to mildly impair both posture and gait. Of the 19 elder children with dyslexia ED1, and ED15 generated sway *z* scores above 1.96 SD of the sample mean on at least one axis in one condition. These participants generated *z* reading scores within 1 SD of the sample mean and were rated within 1 SD of the mean on both the inattention and the hyperactivity scales. The most unusual postural sway *z* scores therefore, were associated either with clinical ratings of combined inattention and hyperactivity symptoms or a previous history of motor difficulties.

7.4 Discussion

The aim of this study was to examine whether balance difficulties in dyslexia are related to age and remediable by development or alternatively, related to the child's level of reading development and therefore persistent with reading difficulties. If the latter prediction were true, reading scores would covary with postural sway scores. Within this sample of children, the control group's level of reading achievement was significantly higher than both of the dyslexia groups. Effect size analyses of our provocation test data revealed larger differences between the younger dyslexia group and both the elder dyslexia and the control group than were found between the two latter groups. This suggests that where balance difficulties are revealed in dyslexia they are related more to chronological age than to level of reading development. Furthermore, the elder dyslexia group were significantly more impaired in reading than the younger control group yet demonstrated a more mature level of postural stability. This provides evidence of developmental delay rather than developmental difference for postural control in the dyslexia sample (Bryant and Goswami, 1986). If balance difficulties in dyslexia are not related to level of reading achievement, they are therefore more likely to be remediated over the time course of development than by the acquisition of reading skills or compensatory reading strategies. Correlation analysis supported these findings. The negative correlations between age and both reading and spelling were stronger than any other association among variables. As the measures used were standardized by age this reflects the level of relative impairment in reading demonstrated by the elder dyslexia group. The next strongest

associations were between reading and spelling and non-verbal intelligence. Significant differences in non-verbal intelligence were revealed among the groups.

Effect size analyses of the provocation test data between the control group and the elder dyslexia group were either small or just above the lower medium boundary, with the exception of continuous sway on the vertical axis in the Visual Modulation condition, $d = 0.63$. This almost replicated the average finding of the meta-analysis. Furthermore, the effect size between the younger and elder dyslexia groups in the Visual Modulation condition on the A/P axis was very large ($d = 1.24$) and exceeded the effect size yielded by the Sheffield Research Group (1992 to 2001), the second largest ($d = 1.17$) in the meta-analysis (Chapter 3). These findings suggest that the elder dyslexia group and the control group demonstrated less difficulty in maintaining stability than the younger dyslexia group. Effect sizes between all three groups on the vertical axis in this condition fell within the higher band of the medium range (Cohen, 1988), which dovetailed with the average effect size found in the meta-analysis. Removal of vision generated greater trunk sway in the younger dyslexia group and heel-raising in the control group. The elder group demonstrated a mature level of postural control. These findings therefore suggest that postural stability is more likely related to age than to reading achievement. Non-verbal intelligence and inattention symptoms are third variables associated with both reading and balance difficulties in this sample.

All correlations between reading ability and measures of continuous sway on 3 axes, in both conditions, were either minimal or small. The strongest associations were found between reading and postural sway on the M/L axis in the Visual Modulation condition. This measure was also significantly associated with inattention and hyperactivity/impulsivity ratings. Forty five per cent of the younger dyslexia group achieved parental ratings in excess of the age-related clinical cut-off score for inattention symptoms. Of these, 40% also achieved hyperactivity/impulsivity ratings above the cut-off score. The elder dyslexia group was found to include 26% of participants with co-existing inattention symptoms in excess of the clinical cut-off score. These proportions are similar to the findings of previous studies described in

section 1.3.1. (e.g., Kadesjö and Gillberg, 2001; Wilcutt et al., 2005). The dyslexia groups therefore included participants with co-existing inattention or combined inattention- hyperactivity/impulsivity symptom ratings. Combined inattention and hyperactivity/impulsivity (ADHD-C) has been shown to be strongly associated with deficits in gross motor control (Piek, Pitcher and Hay, 1999). Sway on the M/L axis is unusual in view of the A/P direction of the provocation test. This indicates that some of the children with higher ADHD-C ratings were demonstrating an unusual pattern of recovery. This again suggests some deviance in performance which plausibly could be attributable to abnormality of lobules VII-X of the posterior infererior vermis of the cerebellum, because this is the most robust finding in MRI studies of both boys and girls with ADHD. Furthermore, M/L sway following perturbation of balance has been observed in cases of posterior lobe (vestibulocerebellum) lesions whereas A/P only sway is associated with anterior lobe lesions (Morton & Bastian, 2004, see section 2.1). Greater sway variability on the M/L axis across trials in the dyslexia groups was also revealed. This again replicates the findings of variability in performance across trials revealed in the adult sample in Chapter 6. As Castellanos et al. (2005) suggested, variability may be a potential endophenotype for ADHD, this further supports the hypothesis that impaired postural stability in dyslexia arises from co-occurring symptoms of ADHD. Both factors of deviance in adaptive response and variability in performance across trials warrant further research.

The role of vision in postural stability in dyslexia was also examined in view of the findings of the meta-analysis and the adult studies. Calculation of the Romberg quotient (e.g., Hayes & Riach, 1989) revealed that the elder dyslexia group quotient was 123.63%, which is typical of an adult proportion (Chiari, Bertani & Capello, 2005; Prieto et al., 1996) with a distribution of scores ± 105.90% that indicated large within group variability. This variability was also apparent in the younger dyslexia group. The control group showed greater dependence on visual cues to orientation and less variability in performance. Stein (2001) suggested that children with dyslexia would have difficulty in adapting to transient visual cues to orientation and experience binocular instability in fixating on a target as required for the Visual Focus condition. Moe-Nilssen et al. (2003) found that all of the children were more

impaired without vision however the children with dyslexia did not demonstrate the same degree of improvement with vision as the control group. The findings of this study highlight the variability in the contribution of vision to postural stability in the dyslexia groups and suggest that this question warrants further study.

The findings of this chapter strongly suggest that quantitative, objective tests of postural stability are not useful predictors of reading difficulties. The first measure of postural stability applied was the DST-J Postural Stability component. The association between DST-J Postural Stability component observational ratings and the provocation test outcome sway values indicated that both measures were tapping the same underlying abilities. The DST-J metric however, indicated that both dyslexia groups were impaired on this measure. DST-J Postural Stability scores were significantly associated with Reading, Spelling and inattention ratings. However, the provocation paradigm yielded results that contradict the findings of the DST-J Postural Stability component in this sample. So how can this contradiction be accounted for? One reason may be that the mean weight used in the drag generated a force that was less than the 2.5 or 3 kg force administered in the DST-J. However, the provocation test has been extensively used with 5% body mass as the benchmark for inducing postural sway in children and healthy adults and is reduced to 3% when used with elderly samples (personal correspondence with Rolf Moe-Nilssen). Five percent of body mass should therefore be sufficient to induce sway in this sample and this was demonstrated successfully in the studies described in previous chapters.

I would suggest that it is the sensitivity of the motion capture system to measure nuances of motion on 3 axes and the ability to discriminate between individuals within both control and experimental groups that accounts for the contradictory findings. By identifying subtle differences among the control group with typical reading development, a distinction that is not afforded by the observational rating scheme of the DST-J component, the relationship between reading and balance was ale to be teased apart. Furthermore, the provocation paradigm and motion capture method have eliminated potential experimenter bias. Reading and postural stability as measured by the provocation paradigm did not covary. However, postural sway on

the vertical axis did covary with chronological age and sway on the medial-lateral axis covaried with either hyperactivity/impulsivity or combined ADHD-C symptoms. These findings also suggest that dyslexia screening tests that include tests of postural stability may be more useful predictors of difficulties associated with hyperactivity/impulsivity or ADHD-C symptoms than reading difficulties.

7.5 Conclusions

The findings suggest that balance difficulties in children with dyslexia are associated with development and not with level of reading achievement. Balance difficulties are likely related to reading by association with combined inattention and hyperactivity/impulsivity symptoms (ADHD-C). These findings concur with the results of the meta-analysis. This evidence does not suggest that balance is related to reading by a direct causal mechanism. Tests of postural stability are therefore, more likely to be useful predictors of co-occurring developmental disorders than specifically of risk of reading failure. Chapter 8 examines whether reading skills and postural stability covary in the general population by further examination of the control group data presented in this chapter plus additional psychometric and literacy data hitherto unreported.

8 Postural stability and reading in a mainstream primary school sample

8.1 Introduction

In Chapter 7 younger children with dyslexia demonstrated greater postural instability than either children in the same peer age group with typically developing reading skills or elder children with dyslexia. It was concluded that balance difficulties in dyslexia were related to chronological age or co-occurring symptoms of ADHD-C and not level of reading achievement. However, considerable variability of postural stability within each of the comparison groups was also revealed. The cerebellar deficit hypothesis predicts that balance and reading are related by a shared causal mechanism, mild cerebellar impairment (Nicolson and Fawcett, 1999). Studies of reading and balance in dyslexia however, have focussed on comparing groups of children with normal reading and children with dyslexia. If postural stability is important for the development of reading skills in the general population then it should covary with reading achievement. Sampling children with different patterns of cognitive and behavioural strengths and weaknesses within the general population might reveal a more accurate picture of the relationship between reading and balance (and ultimately with motor control). Furthermore, two groups of children at different levels of development, separated by two years in chronological age, would be predicted to demonstrate different levels of both reading and postural stability.

Sensitivity to visual and auditory stimuli has been shown to be a robust predictor of orthographic and phonological skills respectively in primary school populations Talcott et al., 2000a; 2002). The relationship between reading and balance skills and deficits in the normal population is as yet unexplored, although the importance of motor development in the assessment of child development is universally acknowledged. Presently in the UK, assessment of balance is subsumed under the physical development category of the statutory National Curriculum assessment for all 3 to 5 year-olds receiving government-funded education (Education, Foundation Stage Profile Assessment Arrangements, 2003). Progression from the Foundation Stage to Key Stage One is dependent upon the successful acquisition of all of the skills within the Profile, including Physical Development. If postural stability and

reading are linked by a shared causal mechanism, as proposed by the cerebellar deficit hypothesis, then early identification of balance difficulties in the mainstream school population would constitute a major step forward in the prevention of many of the negative psychological characteristics associated with reading failure within schools (see section 1.3.1). The aim of this chapter is to examine whether postural stability is a useful predictor of reading ability in children. The following study examines the relationship between literacy, cognitive and balance skills in an opportunity sample of mainstream primary school children. This group of children formed the control sample in Chapter 7. The current chapter includes re-analysis of control group data that were presented in Chapter 7 and analysis of data from additional psychometric and literacy measures hitherto unreported.

8.2 Method

The postural stability, WASI, WORD Basic Reading and Spelling and Barkley Disruptive Behaviour Scale data collected from the children described in this study were compared to the younger and elder dyslexia groups in Chapter 7. Ethical considerations were described in section 4.1.1.

8.2.1 Participant characteristics

A one-form entry primary school agreed to assist with the recruitment of participants from Years 4 and 6 (mean age 9 years 9 months, range 8 years 3 months to 11 years 2 months). Parental consent was received for 28 children. Two children with English as a second language and one child with a physical disability that was adjudged to threaten stability in the provocation test were eliminated from the selection process. Of the remaining 25, 17 were randomly selected and tested, which was the maximum number possible within the time constraints imposed by the school timetable. Data from one student in Year 6 were eliminated from the analysis because testing was not completed within the time limit; also, this student had undergone surgery for a brain tumour. Data from one student in Year 4 were also eliminated as the child was unwilling to co-operate with the postural stability procedure. 15 participants completed all measures (5 girls and 4 boys in Year 6, mean age 10 years 7 months,

range 12 months; 3 girls and 3 boys in Year 4, mean age 8 years 6 months, range 8 months). Experimenters had no prior knowledge of the reading or behavioural status of any child in the sample.

8.2.1.1 Anthropometric measures

Weight and height measurements were recorded for each child.

8.2.1.2 Cognitive, literacy and reading component skill measures

The two-subtest form of the Wechsler Abbreviated Scale of Intelligence described in section 4.1.2.3 was administered. The T-scores were combined and converted to yield an FSIQ-2 score to provide a general summary of intellectual ability. The WASI was used because of time constraints and because equivalency with the Wechsler Intelligence Scale for Children-Third Edition (WISC-III) has been established through a large standardization sample (Wechsler, 1999). Reliability coefficients of the WASI subtests and the FSIQ-2 for children age 8 and age 10 are respectively 0.86, 0.92 (Vocabulary), 0.94, 0.89 (Matrix Reasoning) and 0.92, 0.94 (FSIQ-2) (Wechsler, 1999).

Literacy skills were assessed with the Wechsler Objective Reading Dimensions Test (WORD) Basic Reading and Basic Spelling, the DST One Minute Reading test, the PhAB Non-word Reading and Naming Speed Pictures subtests described in section 4.1.2.4. Total scores on the DST One Minute Reading subtest were used as it is the "At Risk Index" and not the score that is standardised with age compatible norms. For all other measures the age appropriate standard score was used. In practice, this means that the DST is the only literacy and processing speed variable that was not controlled for age effects.

8.2.1.3 Attention measures

Teachers completed the Barkley Disruptive Behaviour Rating Scale Teacher form (see section 7.2.1).

8.2.2 Instrumentation

Motion capture was described in section 4.2.2.2. Marker placement and data sampling rate were described in section 7.2.2.

8.2.3 Procedure

Postural stability was first assessed using the DST Postural Stability instrument described in section 4.1.2. The provocation test was then administered using the portable version of the perturbation rig and the procedure described in section 7.2.3. The minimum drag-weight used was 1.5kg and the maximum 2.6kg.

8.2.4 Analysis

Analysis of the motion capture data was described in section 7.2.4.

8.3 Results

8.3.1 Participant characteristics

Table 8:1 shows the participant codes for 15 children with their gender and school year distribution.

Table 8:1 Participant year and gender distribution

Year 4	Girls	C1	C3	C6		
	Boys	C2	C4	C5		
Year 6	Girls	C9	C11	C12	C13	C15
	Boys	C7	C8	C10	C14	

8.3.1.1 Anthropometric characteristics

Table 8:2 shows the descriptive statistics of the sample anthropometric characteristics.

Table 8:2 Descriptive statistics of anthropometric characteristics (mean ± SD).

	Age in months	Height m	Weight kg
Year 4	102.50 ± 3.88	1.35 ± 0.04	34.62 ± 5.77
Year 6	127.11 ± 3.76	1.47 ± 0.07	39.53 ± 5.65

8.3.1.2 Psychometric, literacy and processing speed characteristics

Table 8:3 shows the descriptive statistics of the sample psychometric, literacy and processing speed characteristics by school year.

Table 8:3 Descriptive statistics of psychometric, literacy and processing speed measures (mean ± SD).

	WASI			WORD		DST	PhAB	
	Vocabulary[1]	Matrix[1] Reasoning	FSIQ-[2]	Basic[3] Reading	Basic[3] Spelling	One[4] Minute Reading	Nonword[3] Reading	Naming[3] Speed Pictures
Year 4	56.00 ±	58.5 ±	113.67 ±	116.50 ±	116.17 ±	56.83 ±	113.5 ±	106.17 ±
N =6	12.20	11.26	18.44	16.13	11.21	20.19	9.67	10.42
Year 6	57.44 ±	60.44 ±	115.67 ±	111.22 ±	113.56 ±	75.33 ±	113.0 ±	106.77 ±
N =9	10.77	6.35	14.58	11.51	14.66	25.55	16.12	15.22

[1]T scores.
[2]Full Scale IQ score converted from summed T scores.
[3]Standard scores.
[4]Total scores.

Year 4 presented a wider distribution of scores on the WASI measures than Year 6. The mean FSIQ-2 score for each year fell within the high average range. The standard deviation for Year 4 exceeded the population SD (15). These results indicated that the majority of children in each year were of above average general intellectual ability. Year 4 included children with general cognitive abilities that unusually deviated from the group mean. Mean scores on the WORD Basic Reading and Basic Spelling tests were also within the high average range. Again, the

distribution of scores on the WORD Basic Reading Test was wider for Year 4 than for Year 6. This variability in scores might be predicted in a group of 8 year old children when reading skills are still developing. The greater variability in WORD Basic Spelling scores for Year 6 than Year 4 however, indicated heterogeneity in spelling ability in this group typical of the general population (within 1 SD, 15).

The group means on the DST One Minute Reading Test confirm the difference in the level of reading fluency and accuracy development between the two age groups. This is not apparent in the scores of any other literacy variable because they are age-standardized. The distribution of total scores on this measure was wider for Year 6 than for Year 4. This indicated that Year 6 included children with more unusual scores. The mean score on PhAB Non-word Reading subtest for both groups fell within the high average range. The distribution of scores for Year 6 was wider than for Year 4. The standard deviation was larger than that of the general population, which demonstrated the presence of unusual scores. The psychometric, literacy and processing speed profiles of the sample were therefore considered to be heterogeneous.

8.3.1.3 Attention measures

For Year 4, the median score for ratings of inattention symptoms was 6.5 (range 0 to 13). For Year 6 the median score was 0 (range 0 to 7). For Year 4 the median score for hyperactivity symptoms was 1 (range 0 to 5). For Year 6 the median score was 0 (range 0 to 3). None of the children within this sample were rated above the clinical cut-off score on either measure (see section 7.2.2.5).

8.3.2 DST Postural Stability component

Figure 8:1 shows the distribution of observational ratings around the median score for the DST Postural Stability component. For Year 4 the median score on the DST Postural Stability Component was 2 (range 2 to 3). The median score was 2 for Year 6 (range 1 to 6). One child in Year 6 demonstrated an 'at risk' of dyslexia performance on this measure.

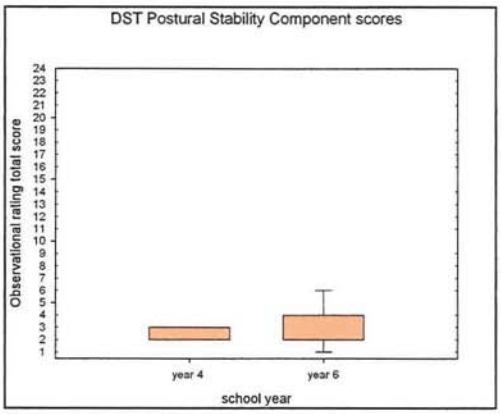

Figure 8:1 DST Postural Stability component total scores.
Boxplots show the DST Postural Stability component observational rating scores.

8.3.3 Provocation test continuous measures

Table 8:4 shows the descriptive statistics for continuous postural sway following provocation in the Visual Focus and Visual Modulation conditions on three cardinal axes. The distribution of scores on each axis was much greater for Year 6 in the Visual Focus condition than for Year 4. The sway values on the M/L axis were very small. Year 6 also generated the larger distribution of scores on the A/P axis in the Visual Modulation condition however sway values were smaller than in the Visual Focus condition.

Table 8:4 Descriptive statistics for continuous sway following provocation test by school year.

	Visual Focus			Visual Modulation		
	A/P[1]	M/L[1]	Vertical[2]	A/P[1]	M/L[1]	Vertical[2]
Year 4	6.25mm ± 1.30mm	2.36mm ± 0.86mm	0.77mm ± 0.60mm	5.91mm ± 0.94mm	2.21mm ± 0.50mm	0.65mm ± 0.58mm
Year 6	11.60mm ± 12.51mm	3.06mm ± 2.41mm	0.71mm ± 0.71mm	7.35mm ± 4.39mm	2.12mm ± 0.76mm	0.79mm ± 0.84mm

[1]SD sway path Segment 2
[2]SD sway path Segments 9&10

Effect sizes between the two Year groups were calculated. In the Visual Focus condition the largest effect size was revealed on the A/P axis (d = .55). This result exceeded the effect size revealed on this axis in the same condition between the control group (Years 4 and 6 combined) and either of the dyslexia groups reported in Chapter 7. This result also fell within the confidence interval of the combined effect size yielded by our meta-analysis (95%*CI* .44-.78) (Chapter 3). The descriptive statistics confirmed that Year 6 included children who demonstrated weaker postural stability than younger children in Year 4. In Chapter 7 we speculated that the effect size between the control and younger dyslexia groups was limited due to large sway values generated by a minority of children in the control group on this measure. This was supported by these findings. Effect sizes between Year groups on the M/L (d = .36) and vertical axes (d = .09) were small. In the Visual Modulation condition the largest effect size was again revealed on the A/P axis (d = .42). This result fell below the lower confidence limit of the meta-analysis. It was smaller than that revealed between the control group and the younger dyslexia group however it exceeded the effect size between the control group and the elder dyslexia group in Chapter 7. Effect sizes between Year groups on the M/L (d = .14) and vertical axes (d = .19) were small. These findings further suggest that developmental differences between Year 4 and Year 6 may be obscured by children in the elder group who demonstrated weak postural control.

To assess the effect of modulating vision on postural stability in this sample of 8 to 11 year-old children, a paired samples t test was computed on data for each axis. There were no significant differences between vision and no vision conditions revealed on either the A/P axis (t [14] = 1.48, *ns*), the M/L axis (t [14] = 1.29, *ns*) or the vertical axis (t [14] = 0.01, *ns*).

8.3.4 Correlation analyses

As the aim of this study was to develop our understanding of the relationship between cognitive, literacy and balance skills in the general population, non-parametric bivariate correlations were performed among all of the anthropometric, psychometric, literacy, behavioural and postural stability measures. Multiple

comparisons increase the potential for a Type-1 error therefore variables associated with alpha level 0.01 (1-tailed) were adjudged significant. Table 8:5 shows the non-parametric correlations between the postural stability, anthropometric, psychometric, literacy and behavioural measures in our sample.

Literacy variables inter-correlated strongly. FSIQ and literacy measure associations were small. DST-J Postural Stability rating scores were significantly associated with not only single word reading but also speed and accuracy of single word reading. DST-J ratings were also moderately associated with teacher ratings of inattention and hyperactivity symptoms. This measure also correlated strongly with postural sway on the A/P and M/L axes in the Visual Focus condition. The strongest associations found for the provocation test measures were between postural sway on the vertical axis in the Visual Modulation condition, all literacy measures and teacher ratings of hyperactivity symptoms. These associations equalled or exceeded those of the DST-J Postural Stability ratings. This indicated a relationship between heel-raising to maintain stability, reading skills and hyperactivity/impulsivity symptoms. Furthermore, hyperactivity rating scores were moderately, negatively and significantly associated with WORD Basic Reading and Basic Spelling, DST One Minute Reading and PhAB Nonword Reading scores.

Table 8:5 Non-parametric correlations among the anthropometric, psychometric, literacy, behavioural and postural stability measures.

	Anthropometric characteristics			WASI	WORD		DST	PhAB		Barkley Scale		
	Age	Height	Weight	FSIQ-2	Basic Reading	Basic Spelling	One Minute Reading	Non-word Reading	Naming Speed Pictures	Inattention symptoms	Hyper-activity symptoms	DST Postural Stability
VF A/P	-0.15	-0.12	0.07	0.04	-0.51*	-0.30	-0.38	-0.58*	-0.32	0.26	0.37	0.73**
VF M/L	-0.03	-0.16	0.34	-0.04	0.46*	-0.36	-0.61**	-0.55*	-0.46*	0.29	0.44*	0.64**
VF V	-0.31	-0.27	0.36	-0.39	-0.38	-0.28	-0.29	-0.43	-0.19	0.11	0.34	0.40
VM A/P	0.08	0.11	0.37	-0.17	-0.37	-0.19	-0.31	-0.53*	-0.53*	0.05	0.09	0.43
VM M/L	-0.31	-0.26	-0.33	0.16	0.01	-0.15	-0.04	-0.24	-0.31	0.10	0.25	0.08
VM V	-0.09	-0.17	0.36	-0.29	-0.69**	-0.83**	-0.76**	-0.76**	-0.27	0.07	0.55*	0.41
FSIQ[a]	-	-	-	1.00	0.29	0.34	0.34	0.32	0.23	-0.01	0.01	0.14
Reading[a]	-	-	-	-	1.00	0.79**	0.61*	0.79**	0.08	-0.22	-0.56*	-0.61**
Spelling	-	-	-	-	-	1.00	0.70*	0.84**	0.11	-0.17	-0.67**	-0.26
DST[a]	-	-	-	-	-	-	1.00	0.67**	0.50*	-0.46	-0.55	-0.44*
NWR[a]	-	-	-	-	-	-	-	1.00	0.33	-0.22	-0.45	-0.39
NSP[a]	-	-	-	-	-	-	-	-	1.00	-0.06	0.08	-0.18
Barkley I[a]	-	-	-	-	-	-	-	-	-	1.00	0.59*	0.55*
Barkley H	-	-	-	-	-	-	-	-	-	-	1.00	0.51*

* $p < 0.05$,
** $p < 0.01$ level (1-tailed) $N = 15$, critical value $p < 0.01 = 0.61$.

8.3.5 Individual differences in postural stability

Figures 8:2 and 8:3 show the stacked *z* scores in the two conditions.

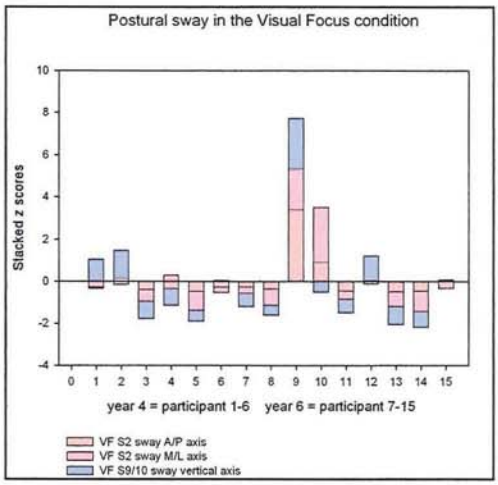

Figure 8:2 Chart demonstrating stacked *z* scores for postural sway on 3 axes in the Visual Focus condition.

Each coloured block represents the standardised value (*z* score) of postural sway averaged across trials for one participant on one axis. Baseline (0) represents the sample mean. Where individual *z* scores across axes consistently fall either above or below the mean they are shown as unidirectional, cumulative stacks. Where scores across axes are mixed (i.e., ± the sample mean) stacks intersect the baseline. Stacks above the baseline represent scores above the mean. Stacks below the baseline represent scores below the mean.

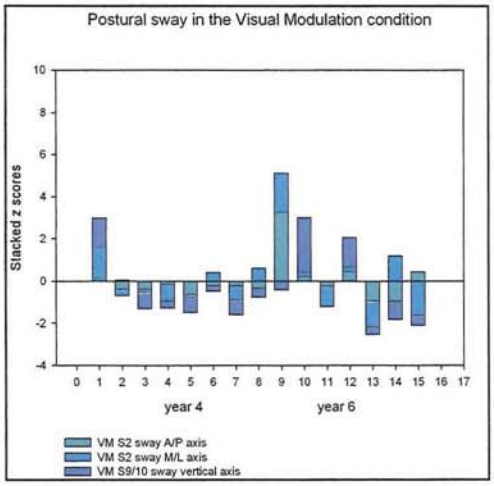

Figure 8:3 Chart demonstrating stacked *z* scores for postural sway on 3 axes in the Visual Modulation condition.

Large continuous sway values were generated by participant C9 on each axis in excess of either 2 or 3 SDs of the sample mean in both conditions, which indicated that this child experienced consistent difficulty in maintaining stability. This participant generated cognitive and literacy scores that fell close to the sample mean with the exception of the score for speed of articulation as measured by the PhAB Naming Speed Pictures test. This score was the lowest of the sample and 1.95 SDs below the sample mean. Only 3% of the population would be expected to achieve a score this unusual. This participant presented an example of impaired postural stability with cognitive and literacy skills within the high and high average range, with the exception of articulatory speed and reading fluency, which also requires rapid articulation. This indicated a mild but more generalized motor deficit, putatively of cerebellar origin, that included oromotor ability (e.g., Fawcett and Nicolson, 2002), that would warrant further assessment beyond the scope and time limit of this study. Alternatively, it indicated a temporal processing deficit that impaired reading fluency but not accuracy. As no prior knowledge of the status of the sample was this participant may have had a history of motor control difficulties, although no indications beyond a general slowness in completing tasks were observed during the session. This child however, appeared to demonstrate some evidence of potential mild cerebellar impairment in the absence of reading difficulties.

Participant C10's FSIQ-2 score fell within the high average range. However, the Basic Reading and Spelling scores fell within the low average range in excess of 2 SDs below the sample mean. This child's DST One Minute Reading total score at age 10 years 11 months equalled that of C4, aged 8 years 3 months. C10's Non-word Reading score fell within the low average range 1.94 SDs below the sample average and the Naming Speed Pictures score fell just inside the lower boundary of the average range. Observations during the session noted that this child responded to the WASI subtests quickly and confidently, however, performance on the literacy measures was laboured and effortful. This child scored the highest total rating on the DST and demonstrated heel-raising. This child also achieved *z* scores in excess of 2.5 SDs of the sample mean on the M/L axis in the Visual Focus condition and in the Visual Modulation condition on the vertical axis. These findings indicated unusual

sway and heel-raising to maintain stability following provocation. Teacher ratings for this child were in excess of 1 SD above the mean on both Barkley measures of inattention and hyperactivity/impulsivity symptoms.

Three other children, C1 and C2 in year 4 and C12 in Year 6 yielded *z* scores in excess of 1 SD above the sample mean on the vertical axis in both conditions. All other cognitive, literacy and behavioural scores fell close to the sample mean. However, C2 obtained teacher ratings of hyperactivity/impulsivity symptoms in excess of 2 SD above the sample mean.

8.4 Discussion

The aim of this chapter was to examine the relationship between literacy, cognitive and balance skills in an opportunity sample of the mainstream primary school population. No significant association between age and postural stability was revealed. Five children in this sample of 15 (33.33%) demonstrated postural instability in at least one condition in comparison to their peers. Two children (13.3%) in Year 6 demonstrated consistent balance difficulties and consequently limited the balance effect sizes between the two Years. This effectively reduced the strength of association between age and balance that had been predicted. None of the children in the sample approached the age and gender appropriate clinical cut-off score for either inattention or hyperactivity/impulsivity symptoms on the Barkley Disruptive Behaviour teacher form ratings. Strong associations were revealed however, between heel-raising to maintain stability, reading component skills, reading fluency and spelling ability and also hyperactivity/impulsivity. These associations equalled those revealed for the DST-J Postural Stability component in strength and level of significance. The strength of these relationships indicates that reading is related to balance by association with hyperactivity/impulsivity in this sample of the general population. Postural sway on the M/L axis in the Visual Focus condition, that indicated a deviant adaptive response to provocation, was also significantly ($p<0.05$) associated with hyperactivity/impulsivity teacher ratings. This shows that when the data reported in Chapter 7 for the two dyslexia groups are removed, the association between hyperactivity/impulsivity symptoms and impaired

postural stability is unaffected or strengthened. This potential link between behaviour in the classroom, literacy and balance skills warrants further investigation in a larger sample of the mainstream primary school population.

There was also evidence of balance difficulties occurring with above average reading component skills and unusually slow processing speed that again indicates a need for further research. Of the two children who showed consistent difficulty in maintaining stability following the provocation test, one demonstrated a profile of high cognitive and literacy ability but unusually weak processing and articulation speed. The other child presented a profile of cognitive ability within the high average range with reading component skills within the low average range, which was commensurate with a profile that might indicate a specific reading difficulty (Miles, 1993). This child's inattention and hyperactivity rating scores were also unusually high in comparison to the sample mean. One other child who demonstrated less consistent postural instability also achieved high inattention and hyperactivity rating scores with average literacy and cognitive scores in relation to the sample. Talcott et al. (2000a; 2002) found that tests of sensitivity to transient stimuli were robust predictors of reading component skills. Good performance predicted good reading skills, weak performance predicted poor reading skills. Analysis of the data from this sample of the mainstream primary school population further suggests that tests of postural stability may be useful predictors of development and developmental disorders but not specifically of reading disability.

The anthropometric data demonstrated typical developmental associations between age, height and weight variables with no extraordinary trends. Although there were wide distributions of psychometric and literacy scores, the sample mean scores for FSIQ-2, reading, spelling and processing speed were all within the average or high average range, which replicates the school's Ofsted Report (2002) profile and recent NFER literacy test results. The majority of children in the sample had normally developing orthographic and phonological reading component skills commensurate with their cognitive ability. Recruitment of a one-form entry school and random selection of participants appear to have been successful in providing a heterogeneous

sample in terms of cognitive, literacy and balance skills and also behaviour in the classroom. Furthermore, taking the provocation paradigm and motion capture system into the field was both viable and useful. The difficulties encountered and the consequent reduction in experimental control appear to be no more or less than in any other transfer from laboratory to field research. A larger project would therefore be feasible.

8.5 Conclusions

Removal of the data from the dyslexia groups reported in Chapter 7 and further analysis of the control group data have revealed a link between behaviour in the classroom, literacy and balance skills that warrants further investigation in a larger sample of the mainstream primary school population. Evidence of balance difficulties in the *presence* of good reading skills was also revealed. Tests of postural stability are likely useful predictors of developmental disorder, ADHD in particular, but not specifically of reading difficulty.

9 Deficits in motor control: causes or correlates of reading disability?

9.1 The contribution of the thesis to resolving the question

9.1.1 The aim of the thesis

The aim of the thesis was to bring a more objective and quantitative approach to the study of motor control deficits in developmental dyslexia where previous research findings had been contradictory. Chapter 1 summarised the main causal theories of dyslexia and described their accounts of the nature of the relationship between reading and motor control difficulties. Chapter 1 also placed dyslexia and motor control deficits within contemporary frameworks for studying developmental disorders with particular emphasis placed upon comorbidity. Chapter 2 then reviewed current theory in motor control research. The most frequently tested motor skill in the literature is balance and it is from these paradigms that the most notable evidence for motor control deficits in dyslexia has been found. Moreover, tests of postural stability are included in several dyslexia screening batteries. Two research questions were consequently generated. First, is balance related specifically to reading or by association with other developmental disorders? Second, can balance measures predict risk of reading failure?

Chapter 3 reported a meta-analysis of previous research by systematically comparing the results of studies using effect sizes between-groups. Effect sizes were combined across studies and a moderately large and robust effect of balance difficulties was revealed. This exceeded the magnitude of the combined effect size yielded by a meta-analysis of phonological ability (van Izendoorn and Bus, 1994) that is widely acknowledged to be the core deficit in dyslexia (Stanovich, 1988). In the meta-analysis, however, effect sizes were significantly heterogeneous across studies and furthermore, the strongest predictor of balance effect size between–groups was whether samples had been screened for co-existing attention deficits. It was concluded that sampling heterogeneity among studies, along with other methodological inconsistencies, were responsible for the lack of homogeneity of

effects. These would need to be addressed in order to advance the study of balance in dyslexia.

In Chapter 4 a paradigm was developed to address the methodological issues revealed by the meta-analysis with particular regard to sample classification, procedural variability and measurement. Dyslexia samples were recruited on the basis of IQ-reading discrepancy and no other exclusionary or inclusionary criteria. Current theories of postural control were applied to assess balance in adults and children with dyslexia. A provocation test was used, designed in replication of the DST-J/DAST Postural Stability component, using an individually calibrated weight to deliver a force proportional to body mass to perturb stability. This was in accordance with the anthropometric conventions of postural and balance research. Balance was assessed in the primary school-aged, senior school-aged and adult dyslexia population and in the general population. Postural sway following perturbation of balance was measured using a digital optical motion capture system and the techniques of linear kinematic analysis. Finally, it was predicted that if reading and balance were directly related through a common set of neural mechanisms, then level of performance on one measure would be strongly correlated with that on the other and that this pattern of results would hold across the general population as well as the dyslexia population. Although correlation does not demonstrate causation, persistently weak associations between reading and balance would bring a causal relationship into question. Furthermore, persistent associations between either reading or balance and other cognitive and behavioural variables would imply that any relationship between reading and balance was only by association with a third variable, for example low IQ or symptoms of comorbid ADHD. The strength of association between balance, cognitive and literacy variables was therefore assessed across the samples in each study. It was also predicted that if the relationship were causal then balance deficits would not be remediated by the developmental time course. Therefore, older readers with dyslexia would demonstrate reading and balance difficulties in equal measure.

9.1.2 The rationale for the thesis

The success of intervention techniques is strongly related to the early identification of literacy failure and prompt commencement of support (Bradley, 1988). Currently, assessment of static and dynamic balance is routinely conducted for all 3-5 year-old children undergoing government funded education in the UK. This is part of a profile assessment of general physical, cognitive and social skills that must be accomplished before a child can progress to Key Stage One, the first level of education within the National Curriculum and assessment within the National Framework for Qualifications. Balance assessment is therefore one means of evaluating child development in general and it also serves to identify early onset of symptoms of developmental disorder. Furthermore, a number of dyslexia screening tests include manually calibrated tests of postural stability that are relatively quick and easy to administer (see section 1.1). When used in conjunction with measures of literacy and related skills they can highlight a need for further investigation in cases that are deemed 'at risk' of dyslexia based on a score profile.

Within the broader phenotype of dyslexia, children who demonstrate deficits in balance and motor skills may well experience greater difficulties in both school and home life than those children who manifest a more 'pure' form of dyslexia. Consequently they may be at even greater risk of developing the negative psychological aspects associated with reading difficulties (described in section 1.1). Balance assessment, therefore, may play an important role not only in defining the broader phenotype of dyslexia but also in identifying a child's particular needs beyond those of literacy support. The importance of understanding whether balance difficulties are causally related to reading difficulties or by association with symptoms of other developmental disorders has one other practical implication. If balance and motor control deficits could be shown to be a correlate of specific reading difficulties by association with other developmental disorders and not a cause, then this would have direct consequences for intervention techniques in dyslexia. Intervention regimes that train balance and coordination skills to progress the development of cerebellar function and claim to improve reading skills would be of questionable value to children with good balance skills.

9.1.3 The findings of the thesis

Studies 1 and 2 (Chapter 4) evaluated the reliability and validity of the provocation test paradigm that had been adapted from the measure available in the DAST. Study 3 (Chapter 4) demonstrated that the paradigm discriminated individual differences in postural stability in adults with dyslexia and also highlighted a need for minor adjustments to both procedure and analysis. Study 4 (Chapter 5) revealed that the provocation test correlated significantly with the DAST postural stability component ratings, therefore both measures tapped the same underlying construct although the strength of association was moderate. The results also indicated variability within the dyslexia group and a need for more in depth analyses of the motion capture data by examining the motion of the COM on 3 cardinal axes.

Study 5 (Chapter 6) introduced sensory modulation conditions to assess the ability of adults with dyslexia to weight and select the available information within the sensory reference hierarchy (Horak, Nashner & Diener, 1990). Combining the effects of sensory modulation with a provocation paradigm was intended to create a dual task paradigm without using a potentially confounding secondary task. It was hypothesised that the effects of sensory modulation would be exacerbated in the presence of symptoms of comorbid ADHD. Greater inter- and intra-participant variability was revealed within the dyslexia group than in the control group on the provocation test of balance. Inter-participant variability indicated that although a minority of the sample demonstrated postural instability across trials they were not however, representative of the majority. Intra-participant variability indicated that some members of the dyslexia group generated inconsistent amounts of postural sway across trials. In general, the results replicated the findings of the meta-analysis. Postural instability was associated with high ADHD symptom self-rating scores. This study further identified the ADHD dimension that correlated with balance difficulty, in this case hyperactivity/impulsivity. This study also included a broad assessment of reading component skills. It was therefore concluded that reading difficulties were related to balance difficulties by association with a third variable, hyperactivity/impulsivity, in this sample. Analysis of the strategy used to maintain stability among the dyslexia group strengthened the association between balance and hyperactivity/impulsivity.

Deficits in motor control: causes or correlates of reading disability?

Castellanos et al. (2005) suggested that variability on measures of behaviour and cognitive function associated with ADHD, known as 'ARV', is ubiquitous in experimental samples. They proposed that intra-subject measures of variability might be a potential endophenotype for ADHD. They further suggested that, because the diagnostic boundaries between developmental disorders, in particular those in DSM-IV (American Psychiatric Society, 2000), are subjective and consequently fairly undependable, the pathological physiology and related processes that underpin intra-individual variability may transverse diagnostic categories. Variability may also be an endophenotype for co-occurring ADHD with dyslexia. Willcutt et al. (2005) also indicated that the most promising endophenotype for dyslexia and ADHD is variability of temporal processing. This strongly suggested that impaired stability and variability of postural sway across trials likely arose from co-occurring symptoms of ADHD. The causal mechanism of impaired postural stability in the sample was therefore hypothesised to be structural and/or functional abnormality within the posterior inferior vermis region of the cerebellum, putatively arising from state dysregulation of levels of the neurotransmitters dopamine and noradrenaline associated with ADHD. Elevated levels of DAT dopamine transporter have been observed in ADHD adults (Dougherty et al., 1999; Krause et al., 2000). Elevated DAT density leads to increased re-uptake of DA and consequently reduced magnitude and duration of the signal (see section 1.3.4). Further study using structural MRI methods would be required to initiate the testing of the hypothesis of PIV abnormality. To test the hypothesis of dysregulation of the neurotransmitters in maintaining postural stability first, the effects of methylphenidate on postural sway following perturbation of balance could be examined using the paradigm described in Chapter 6. Second the effects of methylphenidate could then be examined using single photon emission computed tomography (Krause et al., 2000).

It was also hypothesised, however, that if reading were associated with balance by a direct causal mechanism then some adults might have acquired compensatory balance strategies in line with their reading strategies that are only threatened in the presence of co-existing hyperactivity symptoms. Consequently, balance would need to be assessed before compensatory reading strategies had been acquired. Study 6, therefore, tested the hypothesis of a direct causal relationship by comparing an

opportunity sample of children from the mainstream primary school population with a similarly aged group of children with dyslexia. Each of these two young groups was also compared with a group of elder children with dyslexia.

The aim was to assess whether balance difficulties could be demonstrated in a group of elder children with reading ability compared to a group of younger children with typical reading development. If the elder children were more impaired on the balance measures than the younger children, this would indicate that their difficulties were attributable to their reading difficulties rather than to straightforward developmental delay (Bryant & Goswami, 1986). The elder group demonstrated postural stability closer to that of the control group, than to the younger dyslexia group, even though their level of reading ability was significantly lower. It was concluded that balance difficulties improve with development even though reading difficulties persist. Postural instability in developmental dyslexia therefore, is more likely to arise from developmental delay than from reading difficulties. This conclusion challenges the hypothesis that postural stability is deviant (Nicolson & Fawcett, 1999) rather than delayed in dyslexia and is therefore, according to this causal hypothesis, unlikely to be associated with reading skills by a causal mechanism. Where postural stability appeared to arise from function deviance and not developmental lag in adults in study 5 (Chapter 6), deviance was associated with symptoms of hyperactivity/impulsivity.

Minimal association with reading was revealed across balance measures although there were moderate correlations between the DST-J Postural Stability component and literacy measures. These inconsistent findings were attributed to the objectivity and sensitivity of the motion capture system to discriminate individual differences in both dyslexia and control groups that is not afforded by the subjective assessment used with the DST-J metric. It was concluded that due to the objective methodology employed in the thesis studies, both sampling and experimenter bias had effectively been excluded.

Finally, the data collected from the children with dyslexia in study 6 (Chapter 7) were removed in Chapter 8 and the data collected from the children in the control group were further analysed. This analysis included other measures that were not reported in study 6. In this way the relationship between reading and balance was examined in a primary school population. Only one child demonstrated poor balance, above average cognitive skills and weak literacy skills. Another child demonstrated poor balance, cognitive and literacy skills within the high and high average range and slow speed of articulation. The strength of the relationships between reading and balance and hyperactivity were similar or increased in this small sample to those found in the previous adult and child studies (Chapters 6 & 7). This suggested that further investigation in a larger sample would be warranted. Identification of the variables associated with postural instability in a large community sample would likely yield a clearer representation of the role of postural stability in predicting developmental disorders.

9.1.4 The implications for theory

There is a growing body of evidence for atypical development of both structure and function of the cerebellum in dyslexia (described in section 1.2.2). The findings of the studies reported here assessed the outcome motion of the COM in response to provocation of balance and not directly the underlying neural activity. To assess putative cerebellar function more directly, movement generated during administration of a battery of behavioural cerebellar measures, such as Dow and Moruzzi (1958) upon which the Fawcett, Nicolson and Dean (1996) test battery was based, would need to be recorded using motion capture. The evidence presented in the thesis however, contradicts predictions based upon the hypothesis that balance and reading are related by a direct causal mechanism of cerebellar impairment.

Whilst the cerebellar deficit hypothesis appears to provide a parsimonious account of the multiple behavioural manifestations associated with the broader phenotype of dyslexia, its generality is its main weakness. Although the cerebellum is similar in neural structure (Purkinje cells, granule cells, Golgi cells and stellate/basket cells) throughout its volume, it is clearly divided into functional regions and localised

abnormality of structure has been shown to underpin impairment in specific functions (Ghez & Thach, 2001; Morton & Bastian, 2004; Voogd & Glickstein, 1998). No research to date has used neuroimaging methods to investigate the regions of the cerebellum that are associated with balance and motor skills impairment in dyslexia, apart from motor learning (Nicolson et al., 1999).

The evidence for impairment of the posterior inferior vermis (PIV) of the cerebellum is the most robust finding in structural MRI studies of boys with ADHD although whether these anomalies are related to specific dimensions of ADHD is not known (Berquin et al., 1998; Bussing et al., 2002; Castellanos et al., 2001; Hill et al., 2003; Mostofsky et al., 1998). Furthermore, Castellanos et al. (2003) revealed significantly smaller volumes in the PIV region in girls with ADHD than controls. No other regions, even those revealed in boys, were found to be significantly smaller after covariance. This region is known to be involved in the regulation and synchronisation of vestibular and visual cues to orientation, balance, locomotion, eye movements, postural and vascular tone and respiration (Anderson, Lowen & Renshaw, 2006; Ghez & Thach, 2000; Morton & Bastian, 2004; Voogd, 2003; Voogd & Glickstein, 1998). Regulation of these functions is essential to the maintenance of stable, upright posture (Anderson, Lowen & Renshaw, 2006; Balasubramaniam & Wing, 2002; Horak, Nashner & Diener, 1990; Maki & McIlroy, 1996; Melvill-Jones, 2001; Shumway-Cook & Woollacott, 2001; Yardley, Gardner, Leadbetter & Lavie, 1999). The association between balance difficulties and ratings of hyperactivity/impulsivity or ADHD-C symptoms reported in the thesis suggest that impaired postural stability likely arises from impaired cerebellar function. However, this impairment is more likely due to co-occurring symptoms of the hyperactivity/impulsivity dimension of ADHD than to dyslexia. The greater intra-participant variability in postural stability across trials in the dyslexia samples gives further support to this premise, as performance variability is one of the few ubiquitous characteristics observed in ADHD and potentially, dyslexia with ADHD (Castellanos et al., 2005; Willcutt et al., 2005).

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Impaired postural stability can be explained in terms of the causal hypotheses of ADHD: response inhibition, delay aversion and regulation of arousal/activation. The response inhibition account might predict that recovery responses following perturbation of balance would be slowed and postural sway would be consequently exaggerated. The delay aversion account might predict that postural sway would be exaggerated as a compensatory process following enforced delay between the individual's preferred and anticipated earlier time of release of the weight and the longer actual time. The regulation of arousal/activation account might predict that exaggerated postural sway arises from a disproportionate withdrawal of effort from the energy pool to compensate for state dysregulation between the perceived requirements of the task and the resources available in the arousal and activation pools (see section 1.3.3.3). The neural basis of ADHD also offers a plausible explanation, although to date there are no studies in which participants with structural anomalies of the cerebellum have also been assessed on measures of postural stability.

Pathophysiology of the posterior inferior vermis of the cerebellum would impair the co-regulation and synchronisation of visual and vestibular information about the orientation and posture of the individual in the environment (Ghez & Thach, 2000; Melvill-Jones, 2000; Morton & Bastian, 2004). In children with ADHD this would be predicted to impair stability during development because mapping between the visual and vestibular systems emerges at around 2.5m and is the basis of postural control (Shumway-Cook and Woollacott, 1985). Within the sensory hierarchy, vision is initially predominant but with maturity, somatosensory information becomes more important (Foudriat, DiFabio and Anderson, 1993; Shephard & Telian, 1996). Study 6 revealed greater inter-participant variability in the contribution of vision to balance in the younger and elder children with dyslexia, even though the elder group's average Romberg quotient indicated an adult-like level of independence of vision (Chiari et al., 2005; Pieto et al., 1996). Moe-Nilssen et al. (2003) and a recently published on line study by Stoodley, Fawcett, Nicolson and Stein (2005) found that children with dyslexia demonstrated less improvement than control children when balancing with visual cues to orientation. The contribution of vision to balance in dyslexia requires further investigation because there is

conflicting evidence as to whether visual deficits in cerebellar lesions arise from generalized impairment of the dorsal visual system (magnocellular) (e.g., Ivry and Diener, 1991; Thier, Haarmeier, Treue and Barash, 1999). Adults with ADHD would be predicted to demonstrate normal postural stability when only visual cues to information are removed because somatosensory information is predominant. However, when somatosensory information is modulated as in study 5 (Chapter 6), compensatory mapping between the visual, vestibular and motor postural systems would be impaired and postural sway would be exaggerated. This is precisely what was revealed in study 5 (Chapter 6). Postural sway was significantly associated with hyperactivity/impulsivity ratings that were also significantly associated with intra-participant variability of postural sway across trials.

Hyperactivity has been associated with both hypodopaminergic (Shaywitz, Yager, & Klopper, 1976; Cardinal, Pennicott, Sugathapala, Robbins & Everitt, 2001) and hyperdopaminergic animal models of ADHD (Giros, Jaber, Jones, Wightman & Caron, 1996). Either extremity of dopamine function may lead to disordered behaviour and cognition (Castellanos & Tannock, 2002). Castellanos and Tannock (2002) suggest that dopaminergic fibres in the posterior inferior vermis might form part of a cerebellar circuit that influences the ventral tegmental area and the locus coerulus. The ventral tegmental, a midbrain nucleus, is the main supplier of dopamine to the cortex and is important in initiating behavioural responses to stimulation via the cortex and basal ganglia. Insufficient dopamine arising from excessive uptake by DAT would hypothetically affect the ability to moderate the recovery response relative to the perturbation stimulus. The locus coerulus, a nucleus of the brain stem, is the main supplier of noradrenaline to the cortex and influences arousal, sensory perception and muscle tone (Pliszka, 2005). Release of noradrenaline that is disproportionate to the release of dopamine would hypothetically influence the ability to correctly perceive, adapt and repond to the perturbation stimulus. The stimulus itself may be perceived to be of greater or less salience than in reality. Pliszka (2005) suggests that although dopamine and noradrenaline are implicated in the pathophysiology of ADHD the main contribution may not be the total level of either produced but the amount produced relative to a particular task. The dysregulation of dopamine and noradrenaline is hypothesised to

play an important role in the pathophysiology of ADHD and involvement of lobules VIII-X within the regulation process has also been demonstrated (Castellanos & Tannock, 2002; Krain & Castellanos, 2005; Volkow et al., 2005).

Diamond (2000) reviewed the evidence for cerebellar structural abnormalities in developmental disorders and highlighted the correlation of the level of neural activation during performance of cognitive tasks between the dorsolateral prefrontal cortex and the neocerebellum (described in section 1.3.3). In view of the relatively late maturation of the cerebellum, development of this structure could be influenced by insult anywhere within the systems that underpin the perception-action-cognition cycle, during the process of neurogenesis (described in section 2.3) (Diamond, 2000; Thelen & Smith, 1994). The functional ability of reading is underpinned by neural systems elsewhere in the cerebral cortex and may therefore be related to the cerebellum by association and not by cause (Zeffiro & Eden, 2001). Weak connectivity between the prefrontal cortex and the cerebellum would likely influence both cognitive and motor development (Diamond, 2000; Hadders-Algra, 2000a, 2000b). Furthermore, impaired sensory information would likely influence the mapping of perception to action and impede the development of accurate internal models of stance and movement (Gurfinkel, 1994; Zeffiro & Eden, 2001). In this way, the cerebellum becomes the 'innocent bystander' proposed by Zeffiro and Eden (2001, p.512) rather than the causal mechanism claimed by Nicolson and Fawcett (1999).

The evidence presented in the thesis however, does not suggest that impaired postural stability arising from cerebellar impairment is a consequence of reading difficulty. The evidence suggests that impaired postural stability in dyslexia likely arises from co-occurring cerebellar impairment associated with ADHD but not dyslexia *per se*. The cerebellar deficit hypothesis is one of global, non-specific impairment. Until neuroscience methods, for example structural magnetic resonance imaging (MRI) that have been used to investigate ADHD, are applied to examine specific regions of the cerebellum and cortex in order to address hypotheses relating to postural control and reading, the premise of structural and/or functional

abnormalities that putatively underpin the range of deficits reported in dyslexia must remain hypothetical. At present, the hypothesis that impaired postural stability in dyslexia is a direct result of the abnormal structure and/or function of the posterior inferior vermis of the cerebellum associated with co-occurring ADHD appears to be the most promising explanation.

9.1.5 The implications for practice

The findings of the thesis suggest that tests of postural stability are useful indicators of general development in younger children but not specific indicators of risk of dyslexia. Children undergoing assessment for ADHD might also benefit from the literacy skill and postural stability measures included in screening batteries such as the DST-J. This is because their combination of deficits may well render them even more vulnerable to the negative psychological aspects associated with behavioural and specific learning difficulties. The usefulness of balance measures in predicting reading failure/dyslexia however is less apparent and therefore may be more beneficial in identifying co-existing attention or motor skill deficits than reading difficulties.

The findings also have implications for the training of motor function to remediate reading difficulties. Intervention techniques based upon the hypothesis of cerebellar impairment or delay use daily repetition of movement patterns to improve coordination and balance skills (DDAT, 2005). This repetition is intended to facilitate cerebellar development and lead to further expansion of cortical structures. Repetition is predicted to improve motor function and this improvement is hypothesised to automatically transfer to improved cognitive function including reading. While the cerebellar deficit hypothesis 'is silent on whether attempts to improve cerebellar function will generalise to reading' (Fawcett & Nicolson, 2004b, p.42), the commercial enterprises that market these treatment regimes are explicit in their claims for the effectiveness of their programmes (e.g., DDAT, 2005). If cerebellar abnormalities are associated with ADHD and not dyslexia, as the findings of the thesis suggest, then it is unlikely that hypothesised cerebellar remediation would impact upon literacy. There is no evidence to date that exercises to

specifically develop the posterior inferior vermis region of the cerebellum, the area most closely associated with the regulation of balance, would lead to cognitive development. Importantly, physical therapy has moved away from interventions based upon generalized repetition of movement patterns towards task and context specific interventions to improve motor and cognitive function (see section 2.2).

In dyslexia, the core deficits lie in phonological decoding and orthographic recognition skills. The reading difficulty is the behavioural manifestation of these cognitive deficits that are underpinned by biological bases in neural structure and function. The systems that underpin these reading processes have been shown to demonstrate reduced sensitivity to dynamically changing auditory and visual stimuli (Talcott et al, 1999; 2000b; 2002; Witton et al, 1997; 1998). Performance on these auditory and visual measures has been shown to predict the level of ability in phonological and orthographic reading component skills (Talcott et al., 2000a; 2002). If the cerebellum is just one component within the sensory motor systems that underpin reading, spelling and writing, then intervention techniques based on task-specific training of motor function are more likely to be of lasting benefit to the motor skills of a child with specific reading difficulties than programmes based upon generalised repetitive patterns of movement. Furthermore, learning has been shown to improve with the use of multimodal techniques (Lewkowicz, 2003; Rack, 2004). Control or systems theory predicts that interventions that involve visual and auditory presentation of a word followed by the articulation, visualisation and manual tracing of the word would be more successful in improving cognitive function and, in this case, reading. This is precisely the multi- sensory and motor rehearsal method prescribed for motor learning in developmental disorders by Hadders-Algra (2001b) (see section 2.2.2). With this method, the neural connections within the systems that underpin phonological representation, orthographic recognition and handwriting skills are strengthened. Performance of the task in multiple modalities is the key to developing the systems that are required to successfully accomplish the task within the every day environment. For example, these methods, originally devised by Samuel Orton, a pioneer of dyslexia teaching (Rack, 2004) are used successfully by the Dyslexia Institute (Rack & Hatcher, 2002; Rack & Walker, 1994) and currently are recommended within the UK National Literacy Strategy for children who

demonstrate literacy difficulties within Key Stages One and Two of the National Curriculum (Peer, 2004).

9.1.6 The key contribution of the thesis to dyslexia and deficits in motor control

The application of theory, method and analysis techniques taken from postural and balance research has enabled the thesis to bring a more objective and quantitative approach to the study of balance in dyslexia than had been accomplished by the studies reported in the meta-analysis in Chapter 3. The evidence presented in the thesis suggests that balance deficits are a correlate of reading difficulties but this association is mainly attributable to the effects of a third variable, related to hyperactivity/impulsivity or to ADHD-C symptoms. This supports the main finding of the meta-analysis, that the strongest predictor of balance deficits in dyslexia was whether samples had been screened for ADHD. The association between balance difficulties and ratings of hyperactivity/impulsivity or ADHD-C symptoms reported in the thesis suggests that impaired postural stability likely arises from impaired cerebellar function, hypothetically arising from abnormalities of the posterior inferior vermis identified by neuroscientific studies of ADHD. The evidence does not therefore support a direct relationship between balance deficits and reading difficulties.

Tests of postural stability therefore, may be useful indicators of potential motor difficulties in children that may be additionally disadvantaged by either dyslexia, ADHD, or both. Behavioural rating scales, such as those administered within the thesis, would be of equal benefit during initial dyslexia screening. The findings of the thesis consequently call into question the efficacy of intervention regimes that promote repetition of generalized balance and coordination exercises to advance cerebellar development in order to improve reading (DDAT, 2005; Nicolson, Hambly & Reynolds, 2003). This view is supported by current opinion and practice in postural and balance research and physical therapy, whereby the training of motor function to improve cognitive function is task and context specific. Multimodal intervention techniques, for example those employed by the Dyslexia Institute (e.g., Rack & Hatcher, 2002) and within the National Literacy Strategy (Peer, 2004) would

therefore, be of greater benefit to children with specific reading difficulties in the absence of motor deficits.

The most important contribution of the thesis to the broader sphere of developmental disorders is to provide support for authors who have called for motor assessment in ADHD (Piek, Pitcher and Hay,1999) and reciprocal assessment for ADHD in DCD (Pitcher, Piek and Hay, 2003). Given the extensive overlap between these disorders and dyslexia (Kaplan et al., 2001), I would further suggest that children who demonstrate early symptoms of either attention or motor deficits would benefit from the relatively short literacy and motor skills assessments provided by the DST-J and associated screening tests. In this way, the children most at risk of developing symptoms of a secondary or tertiary disorder would be identified early thus increasing their chances of achieving their potential. The co-occurrence of DCD symptoms and the planning, sequencing and coordination of movement in dyslexia, however, are yet to be examined.

9.1.7 What remains to be resolved?

Four further research questions and potential projects have been inspired by the findings of the thesis:

9.1.7.1 What is the nature of the relationship between the contribution of vision to balance, ocular-motor control and visual motion sensitivity in dyslexia?

The variability among the Romberg quotient percentages yielded by the children with dyslexia in our sample indicates that some children were less impaired in balance in the absence of vision. These findings support those of Moe-Nilssen et al. (2003) and Stoodley et al. (2005) and indicate that further research is warranted. The issues to be resolved are: 1) whether there is an association between eye movement and visual motion sensitivity in children with dyslexia; 2) whether balance difficulties are related to unstable eye movement (cerebellar function) or impaired sensitivity of the visual system to transient stimuli (magnocellular function). This group would be predicted to demonstrate greater inter-participant variability. The

children with impaired visual motion sensitivity and children with unstable eye movement may differ according to their relative contribution of vision to balance. This study would address the issues raised by Thier et al. (1999) and Ivry and Keele (1989). Measures of visual motion sensitivity (Random Dot Kinematograms) (e.g., Talcott et al, 2002) and eye movements (video eye tracking) during fixation and saccade would be compared with motion capture data in quiet standing in the tandem Romberg position performed with eyes open and wearing the opaque goggles described in section 5.1.2.1. This paradigm could be piloted with adults and then administered to children. All participants could also be assessed on measures of inattention and hyperactivity/impulsivity. Motion capture data would be analysed using linear and nonlinear kinematic techniques to examine individual variability of postural sway.

9.1.7.2 Do adults and children with dyslexia and dyslexia associated with hyperactivity/symptoms differ in the timing and sequencing of motor responses following provocation in dyslexia?

The association between hyperactivity symptoms and motion of segment 2 (hence COM) on the M/L axis across studies indicated subtle differences in the reactive pattern of motor activity in some adults and children following provocation. A force delivered on the A/P axis would generate a postural sway pattern that would be predicted to be collinear, equal and opposite (Hall, 2002). Different patterns of postural sway have been observed with different locations of cerebellar lesions. An association between postural sway on the M/L axis indicates an atypical response that is associated with comorbid hyperactivity. It would be predicted that these atypical responses indicate that the perceived threat to stability is greater for individuals with co-existing hyperactivity symptoms, leading to either use of a strategy previously employed in a similar context or the on line assembly of a new strategy for the current context. This would support the link between cerebellar function and balance and motor control deficits in dyslexia with ADHD. This could be further investigated using the provocation test and the simultaneous recording of motion capture and electromyography (EMG) data from the gastrocnemius, hamstring, quadriceps, paraspinals and abdominals. Linear and nonlinear analysis kinematic and kinetic techniques would be used to analyse the motion capture and

EMG data in order to examine individual variability of postural sway and reactive muscle activity.

9.1.7.3 What is the association between balance, motor skills and reading in the primary school population?

The National Curriculum Foundation Stage Profile teacher assessment affords the opportunity to retrospectively examine the relationship between balance and motor skills and the subsequent development of literacy skills and motor skills at completion of Key Stages One and Two (aged of 7 and 11) in a large population. Every government funded primary school will have a record of this profile assessment for every child within a reception class since September 2001. These children will be assessed for literacy skills within the National Framework for Qualifications at the end of Key Stages One and Two. This potentially presents an opportunity for a large, longitudinal study of the primary school population within the UK. Further motor skills assessments would be administered to assess balance, planning and coordination of movement at each stage. Teachers and parents would also be asked to complete behavioural rating forms. Cluster analysis would be used to examine the incidence of and rate of co-occurrence of developmental disorders in the primary school population. Multiple regressions would be used to assess the best predictor from the Foundation Stage Profile categories, of performance on literacy, numeracy and motor skill measures at ages 7 and 11 years. This study would potentially provide the largest assessment of literacy and motor skills in the primary school population since Haslum's (1989) review of the 1970 British Births Cohort Study survey data. It would consequently afford the best opportunity to date to resolve the question as to whether deficits in motor control are causes or correlates of reading disability.

9.1.7.4 Investigation of the cerebellum in adults with dyslexia and dyslexia with ADHD using structural MRI

Structural abnormality of the posterior inferior vermis of the cerebellum is the most robust finding in MRI studies in children and adolescents with ADHD. In this study adults would be assessed using the psychometric, literacy, behavioural and postural

stability measures employed in studies 4 and 5 (Chapters 5 & 6). Structural MRI methods would then be used to test the hypothesis that abnormality of the posterior inferior vermis of the cerebellum is revealed in adults who demonstrate reading difficulties and high ratings on the hyperactivity/impulsivity or combined dimensions of ADHD. Data from these participants would be compared with data from adults with dyslexia with low ratings on the ADHD dimensions. This study would not only aim to replicate the findings of the thesis but also more directly investigate the underlying cause of balance deficits in comorbid dyslexia and ADHD.

9.2 Relating the thesis to the broader sphere of developmental disorders

9.2.1 What have we learnt about dyslexia, postural stability and comorbidity?

Participants with symptoms of co-existing inattention, hyperactivity and combined attention deficits in excess of or at threshold of clinical cut-off scores in each of our dyslexia samples were found in each study sample. The proportions revealed were: from 40% (hyperactivity) to 60% (inattention) of adults and 18% (ADHD-C) to 27% (inattention) of children. These results are similar to those reported in previous studies (e.g., Kaplan et al, 2001; Willcutt and Pennington, 2000; Willcutt et al., 2005). Of these, both of the children above the ADHD-C threshold and one of those above the threshold for inattention symptoms generated *z* scores for postural sway in excess of 1.5SD above the sample mean. Three of the adults above or at threshold of clinical cut-off scores for hyperactivity/impulsivity symptoms also generated postural sway values above 1.5SD of the sample mean on the A/P axis. These adults also demonstrated the least efficient recovery strategy. This provides evidence that the most severe balance difficulties and greatest intra- participant variability across trials in dyslexia are found in persons with high hyperactivity or ADHD-C scores. Assessment of postural stability and motor skills may therefore be as relevant to the diagnosis of ADHD with dyslexia as the RAN tests of Digit Span and Naming Speed (e.g., Denckla & Rudel, 1976).

Castellanos et al. (2005; 2006) suggest that heterogeneity of symptoms in ADHD is the rule. Heterogeneity is also common in dyslexia and DCD. They further suggest

that because the diagnostic boundaries between developmental disorders, such as those in DSM-IV (American Psychiatric Society, 2000) in particular, are subjective and consequently undependable, the pathological physiology and related processes that underpin intra-individual variability may transverse diagnostic categories. Symptom based diagnoses may have in fact impeded research in ADHD (Castellanos & Tannock, 2002) and sampling criteria has certainly obscured research findings in studies of balance and dyslexia (see Chapter 3). Castellanos and Tannock (2002), Pennington (2003, 2005) Willcutt et al. (2005) have emphasised the importance of identifying not only genetic susceptibility for developmental disorder but also the influence of environmental aetiological factors, such as the effect of maternal smoking during pregnancy on dopamine dysregulation during neurogenesis. There is consensus between these research groups that diagnostic classifications do not easily enable mapping between genes and behavioural outcomes. Furthermore, recent evidence has suggested that genetic influences on developmental disabilities are likely to be generalist rather than specific in effect (Plomin & Kovas, 2005).

Castellanos et al. (2006) have also suggested that variability in performance across trials, with a basis in disorder of neurotransmission in state regulation, may be the key to understanding overlapping symptoms of developmental disorders. Response variability in ADHD may therefore share the same causal mechanism as response variability in dyslexia. Putatively, variability may also be an endophenotype for co-occurring ADHD with dyslexia (Pennington, 2005; Willcutt et al, 2005). The hypothesis that overlapping behavioural and cognitive symptoms arise from non-exclusive pathophysiology in developmental disorders would suggest that co-occurrence of developmental disorders is probable and categorical classification therefore, is inadequate. This approach challenges the convention of focussing research on the identification of a single core cognitive deficit in developmental disorders to provide a parsimonious causal account of the behavioural symptoms associated with a single disorder such as ADHD, dyslexia and DCD (Pennington, 2005). It also challenges the classification of diagnostic phenotypes by means of taxonomies of symptoms. Instead it promotes research into the identification of susceptibility genes, environmental influences and emergent endophenotypes or intermediate phenotypes that are related to deeper levels of functioning than

behavioural symptoms. Endophenotypes may be therefore, neurophysiological or cognitive predictors of behavioural symptoms (Castellanos & Tannock, 2002; Castellanos et al., 2005; Pennington, 2005; Pennington et al., 2005). Whilst the identification of endophenotypes is a relatively new approach to developmental research that is increasing in popularity it is not however, without challenge and traditional attitudes towards research into core deficits, categories of symptoms and single biological causal mechanisms persist (e.g. Ramus, 2003; Sergeant, 2005). The evidence presented in the thesis suggests that further research would be justified to investigate the potential of impaired postural stability as an endophenotype for dyslexia with co-occurring ADHD.

9.2.2 What framework can best describe the comorbidity of developmental disorders?

Frith's (1997; 2001) three levels framework for understanding developmental disorders (see section 1.2) is considered to be the most influential and effective model for describing the biological and cognitive underpinnings of symptoms of disorders at the behavioural level. The environment is seen to influence development at each level and it is relatively easy to map a linear causal chain from biological abnormality to behavioural atypicality within the framework. Biological, cognitive and behavioural characteristics of comorbid disorders sit side by side within each level and it less apparent how they interrelate. The main weakness of the model however, is its unidirectional linearity (Karmiloff-Smith, 1998). Furthermore, it is not clear how the development of the multiple connections between the neocerebellum and the dorsolateral pre-frontal cortex described by Diamond (2000) might unfold, how pathology in one set of structures could influence abnormality in another (e.g., Zeffiro & Eden, 2001) or how action might influence cognition in development (e.g., Hadders-Algra, 2000a, 2000b; Thelen and Smith, 1994). The Gilger and Kaplan (2001) view of a single underlying impairment that elicits patterns of characteristics according to individual brain development explains atypical development but also lacks explanatory power. As Frith (2001) points out, it does not provide an adequate explanation for the areas of relative strength, such as high level of intelligence, found in dyslexia. The ontogenetic causal model for the cerebellar deficit hypothesis is also linear and unidirectional (Fawcett & Nicolson, 2004b).

Only the bi-directional, neo-constructivist model proposed by Karmilloff-Smith (1998) takes into account individual differences arising from both the developmental timecourse and the interaction between genetic and environmental influences. What is required is a circular causal framework that provides an explanation within biological, cognitive, behavioural and environmental contexts that elucidates not only the process of development but also the progress of individual profiles of strengths and weaknesses.

Epigenetic dynamic systems theory (e.g., Lewis, 2000; Lewkowicz, 2000; Kelso, 1995; Thelen & Smith, 1994) underpinned by neurogenesis according to neuronal group selection theory (Edelman, 1989; Sporns & Edelman, 1993) are the most promising candidates for modelling the variability and comorbidity of developmental disorders. Within an epigenetic systems framework the characteristics of the phenotype emerge through a complex sequence of interactions between genetic and environmental influences (Forssberg, 1999; Gottlieb, 1991; Lewkowicz, 2000; Thelen & Smith, 1994). Neuronal group selection theory proposes that there is a collection of genetically specified neural networks available from the onset of development that are strengthened or attenuated according to dynamic modulation of neural division, growth and death (Forssberg, 1999; Sporns & Edelman, 1993). Neural development arises therefore, out of complex interactions between the individual and the environment from conception throughout the course of development. The epigenetic dynamic systems framework is extended here to explain comorbidity in developmental disorders in terms of the 'co-emergence' of behavioural symptoms from a complex system.

An epigenetic dynamic systems framework encompasses genetic, biological, cognitive, behavioural and environmental variables, which, at the same time, both influence and are potentially influenced by every other variable in the system. Behavioural traits emerge from the system as a pattern of attributes within a unique, individual profile. Heterogeneity, or the emergence of different levels of functional ability, is apparent at all levels of the system. Heterochrony, or the appearance of abilities at different times, is also apparent (Lewkowicz, 2000; 2003). In the search

for neurobiological causes of behavioural deficits developmental neuroscience has traditionally focussed on the identification of weaknesses. Developmental disorders however, are diagnosed in terms of a profile of scores that indicate areas of relative strength and weakness. To comprehend the co-emergence of these profiles and how task and context specific interventions can ameliorate symptoms of disorders and bring forth hitherto undeveloped strengths, three concepts need explanation: limitations, redundancy and degeneracy.

Limitations are the restrictions imposed on development by the immaturity of elements within a system (Turkewicz & Kenny, 1982; 1985; Lewkowicz, 2000). Sensory systems mature sequentially from the vestibular through to the visual system (Gottlieb, 1971). Limitations are beneficial as they prevent competition among modalities until maturation is complete (Lewkowicz, 2000). Weakness within one modality would be predicted to prolong limitations. Maturation of the sensory systems leads to sensory redundancy, whereby the same function is performed by numerous identical elements. In postural control, reduced sensory redundancy leads to greater variability in stability (Horak, Nashner & Diener, 1990). Abundant redundancy however facilitates stability by reducing variability in postural sway (Shumway-Cook & Woollacott, 2001). Weak temporal processing reduces the number of available degrees of freedom in selecting sensory cues to orientation. In turn, this reduces the number of available patterns of movement to adapt posture to a perceived threat to equilibrium or to a changed context. Intact temporal processing is therefore a pre-requisite for movement to proceed from a state of primary to secondary variability as Hadders-Algra (2000) described (see section 1.3.3). The multisensory intervention techniques described in section 9.1.6 provide redundancy of multimodal information to enhance learning.

The concept of degeneracy affords an opportunity for compensation for lowered redundancy. Degeneracy is the ability of elements within a system that differ structurally but that are able to perform similar functions. Degeneracy within a biological system indicates the complexity of that system (Edelman & Gally, 2001; Tononi, Sporns & Edelman, 1999). Degeneracy is apparent at all levels of biological

organization from genetic and neural encoding to patterns of motor unit coactivation in movement and behavioural strategies. Edelman & Gally (2001) propose that it is a pre-requisite for natural selection and the survival of evolutionary benefits. One important characteristic of degeneracy is that it is context dependent. I suggest therefore, that degeneracy offers one explanation of the heterogeneity and variability of profiles of relative strengths and weaknesses that co-emerge in developmental disorders described by Diamond (2000). Furthermore, degeneracy may provide a mechanism whereby multimodal task and context-specific learning may directly ameliorate co-emergent cognitive and behavioural dysfunction in developmental disorders when onset of intervention is early.

The dynamic systems framework illustrated in Figure 9:1 provides an account of strengths and weaknesses of equal probability according to genetic, environmental and experiential influences. Causality is viewed as circular and emerging properties of the system may also interact creating new cycles of mutually influencing variables (denoted by yellow loops). In terms of literacy, this is the process by which multimodal learning influences the acquisition of reading component skills, spelling ability and accuracy in handwriting. Furthermore, behaviours associated with developmental disorders such as ADHD and DCD co-emerge, thus providing an explanation for the range of characteristics associated with the broader phenotype of dyslexia. One anticipated criticism of circular causality within the dynamic systems framework is that it is non-deterministic. It could be argued however, that it may be more beneficial to predict the potential emergence and co-emergence of developmental disorders within families than to retrospectively attribute causation (e.g., Willcutt & Pennington, 2000; Willcutt et al., 2005). In the thesis sample of 30 children with dyslexia, 8 were siblings (26.7%) and a further 36.7% reported at least one immediate family member with a history of reading and or spelling difficulties. A further 20% reported literacy difficulties within the extended family.

Castellanos et al (2005), Pennington (2003, 2006) and emphasised the importance of considering environmental and cultural influences in addition to genetic predisposition in furthering our knowledge of developmental disorders. Scerif and Karmiloff-Smith (2005) have warned against simply mapping genes to behaviour

without taking into account the developmental process. Furthermore, an alternative view of dyslexia is that it is a genetically predisposed evolutionary gift that has been demonstrated at an individual level through genius and motivation to succeed (e.g., Albert Einstein, Richard Branson and Steve Redgrave). An epigenetic dynamic systems framework, with a neurophysiological basis in NGST, affords the opportunity to anticipate outcome and, as described in relation to control theory in section 9.1.4, specify early onset of task and context specific intervention to nurture ability and remediate disability. It may also provide an illustration of emerging endophenotypes that potentially might link or discriminate between developmental disorders.

Figure 9:1 An epigenetic dynamic systems view of the emergence of developmental strengths and weaknesses.

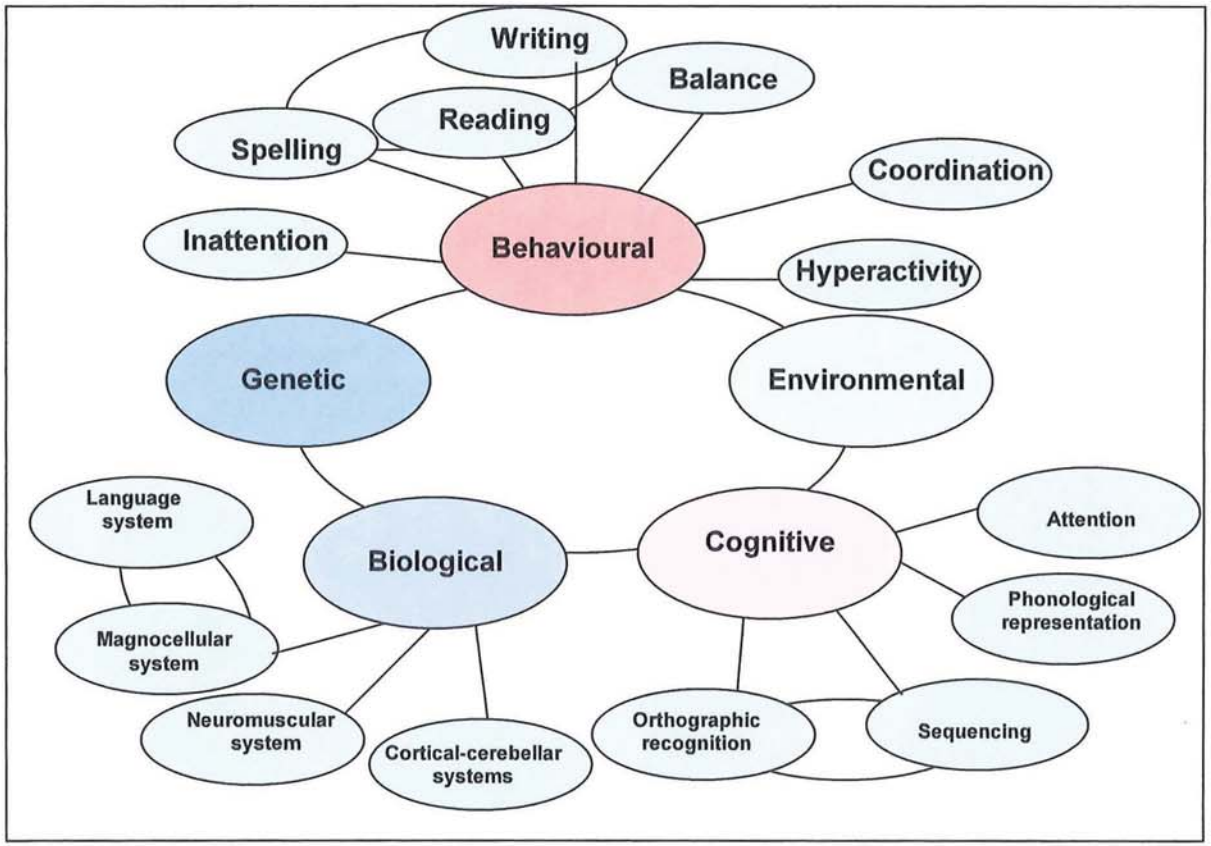

Figure 9:1 The diagram illustrates the multiple interactions between genetic, biological, cognitive, environmental and behavioural factors that influence development within all levels of the individual, self-organizing complex system. Cognitive and behavioural strengths and weaknesses emerge according to the ongoing post-conception cycle of interaction and the developmental time course. Yellow loops denote developing subsystems, impairment within which may signify emergent endophenotypes.

9.3 Conclusions

The thesis has provided a more comprehensive and quantitative analysis of posture and balance in dyslexia than any of the studies reported in the meta-analysis in Chapter 3. Digital optical motion capture and the application of both method and analysis techniques used in postural and balance research discriminated inter and intra-individual differences in postural stability more effectively than the DST-J/DAST Postural Stability component. Motor control deficits, as measured by the assessment of postural stability in the studies reported here, are correlates of reading difficulty related only by association with comorbid symptoms of hyperactivity or ADHD-C. Postural instability is therefore due to the probable abnormality of a specific region, the posterior inferior cerebellar vermis, of the cerebellum that has been robustly revealed in neuroimaging studies of ADHD, rather than mild global impairment of the cerebellum as predicted by the cerebellar deficit hypothesis. It is therefore unlikely that tests of postural stability can specifically predict risk of reading difficulty. They may be useful however, in highlighting the further needs of children with dyslexia who present additional motor and/or attention difficulties. In the broader sphere of developmental disorders, dyslexia screening tests that include tests of postural stability may be of value in the assessment of ADHD and DCD.

As the evidence presented here does not suggest a direct linear relationship between balance and reading, interventions that employ task-specific multimodal training to improve reading are more likely to be effective than regimes that employ generalized repetitive balance and coordination exercises. Finally, as developmental research moves away from the identification of single core deficit accounts of symptom-based diagnoses towards the identification of endophenotypes common to any number of developmental disorders, an epigenetic dynamic systems framework appears to be an appropriate model within which to study developmental disorders. In this framework evolutionary benefits as well as the genetic predisposition for deficits can be encouraged or ameliorated through task and context specific intervention techniques.

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***Denotes studies included in the meta-analysis**

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APPENDICES

Appendix 1

Impaired balance in developmental dyslexia? A meta-analysis of the contending evidence. ***Journal of Child Psychology and Psychiatry, (in the press).***

Appendix 2

BRITISH DYSLEXIA ASSOCIATION (BDA) INTERNATIONAL CONFERENCE (MARCH 2004). COVENTRY, UK.
Abstract: slide presentation.

Appendix 3

SOCIETY FOR NEUROSCIENCE ANNUAL MEETING (OCTOBER 2004). SAN DIEGO, CA, USA.
Abstract: poster presentation.

Appendix 4

INTERNATIONAL SOCIETY FOR POSTURAL AND GAIT RESEARCH (MAY 2005). MARSEILLES, FRANCE.
Abstract: poster presentation.