Experiments in Time: Exploring the components of motor timing behaviour in dyslexia

Emma Elizabeth Birkett

Doctor of Philosophy

Aston University

August 2013

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This investigation aimed to pinpoint the elements of motor timing control that are responsible for the increased variability commonly found in children with developmental dyslexia on paced or unpaced motor timing tasks (Chapter 3). Such temporal processing abilities are thought to be important for developing the appropriate phonological representations required for the development of literacy skills. Similar temporal processing difficulties arise in other developmental disorders such as Attention Deficit Hyperactivity Disorder (ADHD). Motor timing behaviour in developmental populations was examined in the context of models of typical human timing behaviour, in particular the Wing-Kristofferson model, allowing estimation of the contribution of different timing control systems, namely timekeeper and implementation systems (Chapter 2 and Methods Chapters 4 and 5). Research examining timing in populations with dyslexia and ADHD has been inconsistent in the application of stimulus parameters and so the first investigation compared motor timing behaviour across different stimulus conditions (Chapter 6). The results question the suitability of visual timing tasks which produced greater performance variability than auditory or bimodal tasks. Following an examination of the validity of the Wing-Kristofferson model (Chapter 7) the model was applied to time series data from an auditory timing task completed by children with reading difficulties and matched control groups (Chapter 8). Expected group differences in timing performance were not found, however, associations between performance and measures of literacy and attention were present. Results also indicated that measures of attention and literacy dissociated in their relationships with components of timing, with literacy ability being correlated with timekeeper variance and attentional control with implementation variance. It is proposed that these timing deficits associated with reading difficulties are attributable to central timekeeping processes and so the contribution of error correction to timing performance was also investigated (Chapter 9). Children with lower scores on measures of literacy and attention were found to have a slower or failed correction response to phase errors in timing behaviour. Results from the series of studies suggest that the motor timing difficulty in poor reading children may stem from failures in the judgement of synchrony due to greater tolerance of uncertainty in the temporal processing system.

Key words: Temporal Processing, Reading, ADHD, Error Correction, Motor Timing,

Acknowledgements

This doctorate was supported by an Open Competition Studentship award from the Economic and Social Research Council.

I would also like to acknowledge a number of people, without whom this work would not have been achievable. These people have provided a support and guidance in many different ways to enable and promote the research that I have undertaken.

I would like to thank my supervisor, Prof. Joel Talcott, who has encouraged and helped me to develop throughout this process. I also thank the clinical team at the Aston Dyslexia and Developmental Assessment Centre for their assistance and support over a number of years, including Dr. Kim Rochelle.

My thanks also go to the many children and adults who have participated in my studies over the last few years. The research would simply not have been possible without the support of teachers and parents and the enthusiasm of the participants.

For three years of moral support I am grateful to the postgraduate team in SW506 (members past and present). You have been an invaluable source of momentum for every peak and trough of the PhD journey. I also thank Rob Summers for his guidance and programming skills that helped my computer go beep at the right time. Similarly I thank Sarah Birkett for her artistic contribution to the computer programme used in Chapter 9.

Finally, I thank my family for always being at my side throughout this journey. Mum and Dad, thanks for always assuming I could achieve. David Birkett, thank you for your help in putting this document together. Your patience and belief in me is a continuing source of strength.

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1. Introduction to dyslexia

This chapter provides an overview of dyslexia and situates the thesis within the dominant epistemological position regarding the nature of risk factors which contribute to dyslexia. This provides the rationale for investigating a subtle underlying difficulty in dyslexia within the domain of temporal processing.

1.1. Defining dyslexia

Dyslexia is a developmental disorder, appearing in childhood which affects reading related skills, primarily word recognition ability. It is a complex disorder resulting in a heterogeneous array of behavioural outcomes which may become apparent in the classroom environment. In addition, subtle underlying deficits are present in children with dyslexia which may only be identifiable through carefully designed experimental paradigms. This variety means that different groups of professionals (such as educators, clinicians and researchers) have come to define dyslexia in different ways. A working definition of dyslexia encapsulating a number of perspectives was provided by Rose (2009) following a comprehensive review of the literature and consultations with these varied professional groups and people with dyslexia. Rose describes dyslexia as a learning difficulty primarily affecting the skills involved in accurate and fluent reading and spelling, that also features additional difficulties in phonological awareness, verbal memory and verbal processing speed. These difficulties in reading and spelling are accounted for by an impairment in word recognition rather than any particular difficulty in reading comprehension (Nation, 2005). In addition to these primary difficulties, Rose highlights frequently co-occurring difficulties in aspects of language, motor coordination, concentration, mental calculation and personal organisation. Other difficulties can also be identified through behavioural tasks, such as in non-word reading (pronouncing novel words to assess knowledge of sound-letter correspondence), phoneme deletion or spoonerism measures which assess phonological awareness, (e.g. Rack, Snowling, & Olson, 1992; Snowling, 2008) rapid naming tasks (e.g. Wolf, Bowers, & Biddle, 2000) and verbal memory tasks (e.g. Gathercole, Alloway, Willis, & Adams, 2006).

Although historically, the identification of dyslexia has relied on the presence of a discrepancy between a child's literacy skills and more general reasoning abilities, the Rose definition and other contemporary definitions recognise that dyslexia is best described as a continuum occurring across the range of intellectual abilities (Lyon, Shaywitz, & Shaywitz, 2003; Rose, 2009; Shaywitz, 2003; WHO, 2010). As such, children with dyslexia show phonological deficits regardless of whether their reading level is commensurate with their IQ level or discrepant from it (Fletcher et al., 1994).

It is also increasingly recognised that a diagnosis of dyslexia is frequently accompanied by a diagnosis of another developmental disorder such as Dyspraxia or Attention Deficit Hyperactivity Disorder (ADHD) (Pauc, 2005). Between 15 and 40% of children with a diagnosis of dyslexia also show symptoms of ADHD (Semrud-Clikeman et al., 1992; Willcutt & Pennington, 2000), a developmental disorder characterised by the presence of behavioural symptoms along the dimensions of inattention, impulsivity and hyperactivity (American Psychiatric Association, 2000; Barkley, 2006). These behavioural symptoms are underpinned by cognitive impairments in a variety of contexts including for executive function and reward-processing tasks which assess distractibility, vigilance and inhibition (Barkley, 2006). Evidence of overlap between clusters of symptoms in individuals with developmental disorders has contributed to the difficulty in arriving at appropriate definitions of such disorders, but is not unexpected given that diagnostic categories are imposed upon networks of symptoms by clinicians or researchers (Cramer, Waldorp, van der Maas, & Borsboom, 2010). In spite of such challenges, the presence of overlap between these disorders helps to limit the kinds of explanatory accounts used to explain these disorders (Pennington & Bishop, 2009; Pennington, 2006; Plomin & Kovas, 2005), as will be explained in more detail later in the chapter.

Estimates of the prevalence of dyslexia in the population vary between 3 and 20 percent depending on the definitional criteria used in the studies (Esser & Schmidt, 1994; Feeg, 2003; Miles, 2004; Peterson & Pennington, 2012; Shaywitz, Shaywitz, Fletcher, & Escobar, 1990; Shaywitz, 2003). The difference in these estimates is likely due to the differences in populations studied and the criteria used for defining dyslexia. For example, a re-examination of the British Births Cohort Study suggested that the UK prevalence rate was between three and six percent, depending on whether diagnosis criteria included children who were underachieving in reading or who showed positive signs of dyslexia (Miles, 2004). Dyslexia is found across different languages despite differences in orthography and phonology (reviews are presented by Caravolas, 2007 and Peterson & Pennington, 2012). Given the prevalence of the disorder and the literacy difficulties experienced by people with dyslexia, the costs of dyslexia for society are likely considerable: even low pass grades (D-G) in literacy at GCSE level are associated with 12% higher earnings in adults compared to those without such qualifications (Grinyer, 2005). In light of the challenges that dyslexia poses for individuals and society, educators and researchers alike are focused on identifying underlying risk factors that account for the presence of symptoms and may allow for earlier identification of dyslexia and more appropriate interventions.

1.2. Perspectives on dyslexia

In understanding the nature of dyslexia it is important to consider the elements of child development which contribute to reading proficiency. Although the primary deficit in dyslexia is in fluent word decoding, a range of other cognitive skills enable the development of reading and spelling abilities.

These cognitive skills are supported by various functional processes in the brain; processes which are dependent on the structural architecture and organisation of the brain and the functional communication that allows processing to occur. These cognitive and biological elements are influenced by genetic and environmental factors during development (with environmental influences including factors such as language exposure, schooling and nutrition). Dyslexia has therefore often been considered within the framework outlined by Morton and Frith (1995a, 1995b) in recognition of the interactions between the cognitive, biological and environmental levels of analysis that contribute to the observed behavioural symptoms which are used for diagnosis (Figure 1.1). Multiple risk factors for the development of dyslexia have been identified at each stage, particularly in relation to the route to fluent reading and research examining the genetic basis of dyslexia. These two streams of evidence are described below. Therefore, although dyslexia manifests in the classroom as a difficulty in reading and spelling, the recognition of these additional levels of analysis (biological, cognitive and environmental) means that research is often targeted at processes more distal to the core behavioural outcomes.

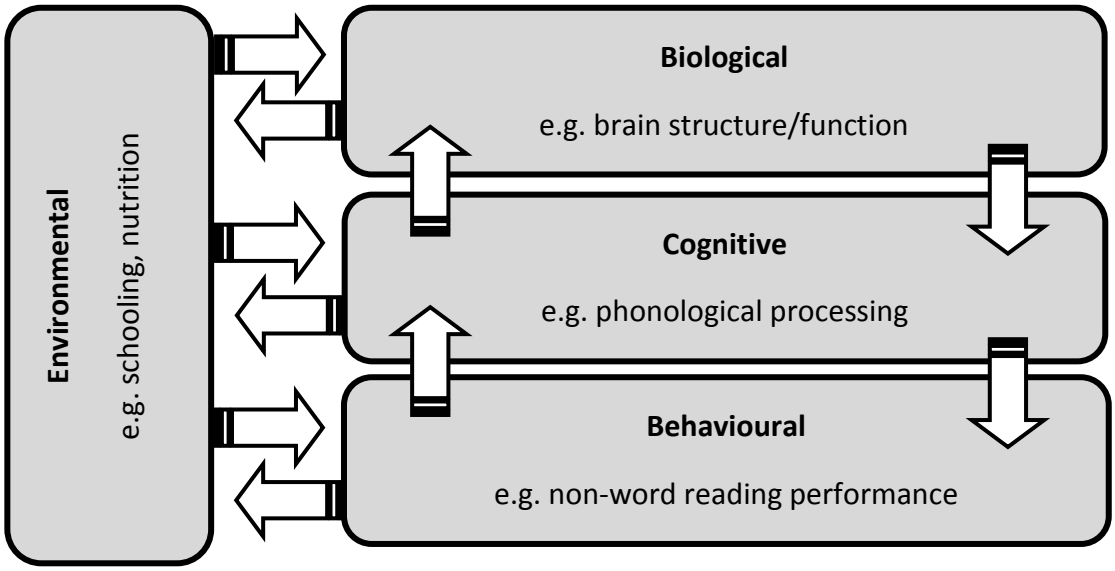

Figure 1.1: Levels of analysis framework for dyslexia
Figure adapted from (Morton & Frith, 1995a, 1995b) showing the various factors which can contribute to the development of dyslexia and examples of each level.

1.2.1. Evidence for multiple risk factors from the route to reading

The goal of literacy development is to be able to comprehend the written texts present in the child's environment and produce written texts in order to communicate with others. When learning to read, a child comes to understand that there are logical relationships between letters (graphemes) and sounds in spoken language which can be used to decode new words either through simple letter decoding and blending, or through analogy to words with similar spellings (Ehri & Mccormick, 1998; Kuhl, 2004). Children gradually become more proficient at decoding larger language units and over

time are able to decode words until reading becomes efficient and automatic (Ehri & Mccormick, 1998; Ehri, 1992). Spelling ability builds on these same phoneme-grapheme decoding skills and sight word vocabulary.

Reading and spelling are intimately linked to pronounced word forms, relying on the ability to segment spoken words into phonemes and map them onto graphemes, (Ehri, 1992). The ability to make sense of linguistically relevant vocal gestures (speech perception) develops long before reading or writing skills. At birth, babies can discriminate phonetic distinctions from any language (Kuhl & Rivera-Gaxiola, 2008) and over the first year of life, through exposure to the speech sounds and vocal gestures of their native language, infants lose the ability to discriminate sounds not present within their environment (Rivera-Gaxiola, Silva-Pereyra, & Kuhl, 2005). Children continue to learn speech vicariously, beginning to recognise words and associate them with events or objects in their environment (Kuhl, 2004). As well as developing word knowledge, this experience shapes the child's neural architecture so it is primed for further acquisition of native words comprising similar language units (Kuhl & Rivera-Gaxiola, 2008).

Particular sound-based features of language allow infants to identify likely word candidates, guided by previous experience of prominent features in the native language (Kuhl & Rivera-Gaxiola, 2008; Poeppel, Idsardi, & van Wassenhove, 2008). These features include the sounds, the gaps between sounds and the temporal and spectral characteristics of speech sounds. The speech recognition system is able to process these features in order to yield speech, despite the speech stream being perceived as a coherent stream of sound (Mann & Liberman, 1983; Whalen & Liberman, 1987). The units of speech are processed by neural areas specialised for speech processing and the meaning of speech is yielded without any need to overtly distinguish the segments that comprise words (Liberman, 1991). On an evolutionary timescale, this type of sound-based processing (of speech) has been used functionally over a much longer period than the analysis of visual graphemic word forms (for reading), allowing a higher degree of specialisation for such sound-meaning translations.

Speech perception therefore differs from the processes of learning to read and write, where the discontinuous sound stream has to be segmented at appropriate points which correspond to letters or letter strings (Davis & Johnsrude, 2007). This task is made more difficult because the phonemes represented by letters are different to the segments used to compute meaning during speech perception. In order to perceive words within this stream, regularities, such as stressed syllables and acoustic boundaries, are used in conjunction with lexical knowledge to derive segments which relate to meanings (Davis & Johnsrude, 2007). As such, when reading text, the distinctive features of speech that by convention relate to each letter have to be consciously extracted from the speech signal, a process which is more difficult for people who have poor phonological representations in their language specialisation (Leppänen et al., 2010; Liberman, 1991).

The building blocks of reading and spelling are therefore many and complex, requiring multiple stimulus-response pairings of sounds, word forms, and behavioural experiences, until skills become automated and proficiency develops. Consequently, the Morton and Frith framework shown in Figure 1.1 incorporates multiple cognitive mechanisms at the intermediate level which can interact with various environmental factors, giving rise to the behavioural literacy outcomes seen in a child in the classroom. This complex route to reading helps to explain why research is targeted at a variety of cognitive processes that at first glance appear somewhat removed from the core deficit in word recognition.

1.2.2. Genetic evidence

At the biological level, genetic analyses provide the principal level of explanation for the basis of disorders. At a macroscopic level genetic evidence can establish the likelihood that a disorder has a biological basis and help define the characteristic nature of developmental disorders, for example in helping to understand the overlap between dyslexia and ADHD. At a more local level, genetic analyses can identify and examine specific gene effects that contribute to disorders and subsequently help to identify candidate biological mechanisms that may be responsible for the functional brain differences that affect cognitive and behavioural development.

1.2.2.1. Heritability

The genetic basis of dyslexia has been well established with family studies showing that dyslexia clusters within families (DeFries, Singer, Foch, & Lewitter, 1978). Respectively, 50 and 75% of children with one or two parents with a reading difficulty have been found to be at risk of dyslexia (Wolff & Melngailis, 1994) and siblings show a greater incidence of reading difficulties than children without familial dyslexia (Pennington et al., 1991). Twin studies have confirmed that this familial clustering is influenced to a large extent by genes in addition to the contribution of the shared environment within families. Identical, monozygotic, twins have concordance rates for dyslexia of 68% compared to 38% in non-identical, dizygotic twins (DeFries & Alarcon, 1996). If the condition were influenced to a greater extent by environmental factors the heritability estimates for the different twin groups would be more similar.

1.2.2.2. Multiple and probabilistic overlap

In exploring the characteristics of developmental disorders, analyses from behavioural genetic studies indicate that it is unlikely that there is a single causal route leading from a single gene to the profile of dyslexia seen at the behavioural level (Fisher & DeFries, 2002; McGrath et al., 2011; McGrath, Smith, & Pennington, 2006). Instead dyslexia, like other developmental disorders, is thought to result from a number of small gene effects which each influence different pathways to behaviour development and convey risk for more than one disorder (McGrath et al., 2011). Quantitative heritability evidence confirms this hypothesis, with genes accounting for one disorder

being implicated in other learning disorders (Willcutt et al., 2002) which often share several of the cognitive risk factors associated with dyslexia (Gathercole et al., 2006; Wolf et al., 2000). Furthermore, genes accounting for variation within a disability are related to a number of cognitive or behavioural factors (Plomin & Kovas, 2005), giving rise to the heterogeneity observed within and across disorders (McGrath et al., 2011; Pennington et al., 2012).

Quantitative analyses of genetic relationships (Plomin & Kovas, 2005) also support the view that these developmental disorders form part of a continuum of ability across the population (Rosen, 2003), with genes being responsible for both normal variation and learning difficulties. Therefore, rather than being unique causes, genes interact with the environment, conferring probabilistic, rather than deterministic, risk for a disorder, such that not all children with dyslexia will show each deficit (Pennington & Bishop, 2009).

1.2.2.3. Risk loci

In addition to constraining explanations of developmental disorders, genetic analyses are able to direct research towards specific biological processes that may influence the development of reading difficulties. Not surprisingly, given the above discussion and the heterogeneity of symptoms present in dyslexia, the disorder has been linked to numerous quantitative trait loci across chromosomes (for reviews see Benítez-Burraco, 2010; Scerri & Schulte-Körne, 2010). By identifying genes at these loci and their biological effects, the potential routes through which neurobiological impairments lead to the profile of dyslexia are being distinguished. Notable examples of such loci are the KIA0319 and DCDC2 alleles located on Chromosome 6. In mouse models, these genes are implicated in key stages of brain development such as in axonal growth and migration (Galaburda, LoTurco, Ramus, Fitch, & Rosen, 2006; Meng et al., 2005; Paracchini et al., 2006), pointing towards potential biological mechanisms through which dyslexia develops.

The identification of risk loci also supports the notion described above that there should be both shared and unique components across co-occurring disorders (Plomin & Kovas, 2005). Several alleles are shared by children with dyslexia or ADHD (Bakker et al., 2003; Loo et al., 2004; Willcutt et al., 2002) and some alleles are found to be being unique to children with ADHD (for example those which regulate dopaminergic systems) (Hsiung, Kaplan, Petryshen, Lu, & Field, 2004). These shared genetic effects help to explain why developmental disorders such as dyslexia and ADHD co-occur more commonly than would be expected by chance.

The typical route to reading development described above and the evidence from genetic analyses serve to demonstrate how investigations at each level within the Morton and Frith model (Figure 1.1) help to constrain the types of hypotheses and research questions permissible at the other levels of analysis in regard to developmental disorders. As such, there is a need to account for the presence of symptoms of other associated developmental disorders when examining risk factors associated

with dyslexia. Furthermore, it is likely that the behavioural outcomes in dyslexia are influenced by a variety of cognitive and behavioural outcomes. This means that although the primary deficit in dyslexia is recognised as a difficulty with phonological processing affecting fluency of decoding, with some authors promoting the primacy of this cognitive deficit (Hulme, Snowling, Caravolas, & Carroll, 2005) other elements will contribute to the development of proficient reading and are worthwhile targets of research. The main theories that have been developed to attempt to explain the aetiology of dyslexia (summarised below) therefore go beyond the behavioural or cognitive levels of analysis to explain how domain general factors may contribute to the development of appropriate phonological representations. However, given the heterogeneity in genes and cognitive deficits described above, it is not surprising that there is a lack of consensus about the aetiology of dyslexia across these different theoretical positions, as discussed below.

1.3. Theories of dyslexia

1.3.1. The automatisation and cerebellar hypotheses

The automatisation hypothesis, described by Nicolson and Fawcett, suggests that children with dyslexia show reading difficulties because they fail to reach an appropriate level of automaticity on tasks which require fluency, such as reading (Fawcett & Nicolson, 1999; Nicolson & Fawcett, 2007, 2011). These authors found that children with dyslexia showed impaired performance on various motor fluency tasks, such as balance and speeded motor tasks, particularly when performing the tasks under dual task conditions. This general procedural learning deficit was put forward as a cause of articulatory fluency difficulties due to effortful articulation which would leave fewer cognitive resources available for sensory processing (Fawcett & Nicolson, 1999; Nicolson, Fawcett, & Dean, 2001; Nicolson & Fawcett, 1990). In turn, it was suggested that this profile of deficits would lead to inadequate processing of speech sounds with implications for the development of phonological awareness, working memory, and rapid naming skills.

Linking this theory to a biological level mechanism, Nicolson et al. (2001) proposed that the automatisation deficit arises from a difference in the cerebellum, leading to movement, timing and coordination differences that might affect writing and articulation in dyslexia. The cerebellum is involved in numerous facets of cognitive processing including language processing, receiving inputs from the majority of cortical areas allowing refinement of signals prior to them being relayed back to cortical areas (Booth, Wood, Lu, Houk, & Bitan, 2007; Ivry & Justus, 2001; Stoodley & Stein, 2012; Strata, Thach, & Ottersen, 2009). In support of this theory, children with dyslexia have been found to show reduced cortical volume in the right anterior lobes of the cerebellum and reduced grey matter asymmetry in the cerebellum (Eckert et al., 2003; Rae et al., 2002). Similarly, functional differences in the cerebellum are found in adults with dyslexia during literacy related and implicit learning tasks

of the kind implicated in the automatisation hypothesis (Brunswick, Mccrory, Price, Frith, & Frith, 1999; Menghini, Hagberg, Caltagirone, Petrosini, & Vicari, 2006).

The specificity of the cerebellar hypothesis has been questioned because the functional differences in the cerebellum may be the result of alterations in function in other brain areas, such as in the visual cortex (Zeffiro & Eden, 2000), or due to reduced articulatory preparation caused by the presence of impaired phonological representations (Ivry & Justus, 2001; Rae et al., 2002; Stoodley & Stein, 2012). Indeed, structural and functional brain differences in dyslexia are not limited to the cerebellum (Brunswick et al., 1999). Reservations about the theory have also been raised because the motor impairments in dyslexia may simply represent the overlap between dyslexia and other disorders such as ADHD (Buderath et al., 2009; Raberger & Wimmer, 2003; Rochelle, Witton, & Talcott, 2009; Viholainen et al., 2011; Viholainen, Ahonen, Cantell, Lyytinen, & Lyytinen, 2002) with some studies failing to replicate Nicolson and Fawcett's findings of motor impairments in children with dyslexia (Raberger & Wimmer, 2003; Ramus, Pidgeon, & Frith, 2003; Wimmer, Mayringer, & Landerl, 1998). Indeed, a meta-analysis of studies examining the balance deficits in dyslexia concluded that the deficits were unlikely directly related to reading ability, but more likely associated with the presence of ADHD (Rochelle & Talcott, 2006).

Such doubts have led recent reviews to conclude that the cerebellar differences in dyslexia more likely result from the reading difficulties themselves or from a secondary interaction with other disorders, rather than acting as a cause of such difficulties (Stoodley & Stein, 2012). Nicolson and Fawcett have also recently acknowledged the vast extent of connectivity between the cerebellum and other cortical and sub-cortical regions of the brain involved in language processing further challenging the specificity of the theory (Fawcett, 2011; Nicolson & Fawcett, 2007). There is therefore little consensus about the contribution of the cerebellum or an automaticity deficit to the phenotype of dyslexia.

1.3.2. The temporal processing hypothesis

A second theory which assumes that the difficulties in dyslexia stem from a domain general, non-linguistic deficit is the temporal processing hypothesis. This arose from evidence that children with dyslexia show behavioural deficits on tasks that comprise of temporal elements, such as rapidly changing or rapidly presented stimuli (Cohen-Mimran & Sapir, 2007; Farmer & Klein, 1995; Hood & Conlon, 2004; King, Lombardino, Crandell, & Leonard, 2003; Reed, 1989; Talcott et al., 2002; Tallal, 1980). The theory assumes that temporal processing is required for learning phoneme-to-grapheme correspondences in childhood (Farmer & Klein, 1995; Tallal, 1980), with a deficit in temporal processing leading to the development of inaccurate phonological representations that continue to affect phonological processing throughout childhood (Tallal, Miller, & Fitch, 1993). It is proposed that this deficit is not limited to the language domain, with children showing behavioural

impairments on both linguistic and non-linguistic temporal stimuli across different sensory modalities (Keen & Lovegrove, 2000; Lovegrove, Martin, & Slaghuis, 1986; Reed, 1989; Tallal, 1980; Vandermosten et al., 2011; Wright et al., 1997) as well as differences in the neural responses shown to these stimuli (Kujala et al., 2000; McAnally & Stein, 1996; Nagarajan et al., 1999). For example, children with dyslexia require longer stimulus intervals to individuate stimuli that are temporally separated or to discriminate the order of sequentially presented stimuli in both the visual and auditory domains (Farmer & Klein, 1995; Mcarthur & Bishop, 2001) and show difficulties in integrating information over time, such as in sensitivity to frequency modulated stimuli and discriminating visual motion coherence (Boets, Wouters, Wieringen, & Ghesquière, 2006; Everatt, Bradshaw, & Hibbard, 1999; Poelmans et al., 2011; Talcott et al., 2002, 2003; Witton et al., 1998).

A range of factors at the biological level have been proposed to account for the temporal processing deficit. One of the more dominant theories suggests that the difficulties in processing temporal stimuli stem a difference in the magnocellular layers of the auditory and visual relay nuclei of the thalamus which receive information from the primary sensory organs and project to the visual and parietal areas (Stein & Talcott, 1999; Stein, 2001). The theory was originally proposed in order to explain deficits shown by children with dyslexia in processing transient visual stimuli (Conlon, Sanders, & Wright, 2009; Everatt et al., 1999; Lovegrove et al., 1986; Slaghuis & Lovegrove, 1984, 1985; Talcott et al., 1998, 2000), because the magnocellular layers of the visual system which are specialised for processing spatial location, motion and depth, in contrast to the parvocellular layers which are specialised for non-temporal aspects of stimuli such as colour and object recognition (Stein & Walsh, 1997). The cells in these magnocellular layers have been found to be smaller and more disordered in children with dyslexia than in typically developing children (Galaburda & Livingstone, 1993; Livingstone, Rosen, Drislane, & Galaburda, 1991).

These differences in the visual system are proposed to relate to reading ability due to the need for rapid visual attention, visual search and eye movements during orthographic processing, with correlations found between motion coherence performance and orthographic sensitivity (Boets et al., 2006; Talcott et al., 2000). More recently, Stein and colleagues suggested that the cellular differences may explain difficulties across other sensory domains, including auditory and motor domains, with magnocells present in all sensory systems (Stein, 2001; Stoodley & Stein, 2011). Others have also linked the magnocellular deficit to attention regulation differences observed in individuals with dyslexia due to the attentional capture made possible by the magnocellular system, allowing rapid access to stimuli to facilitate sensory (and therefore phonological) representations (Hari & Kiesilä, 1996; Hari & Renvall, 2001).

Criticisms have, however been raised regarding the magnocellular theory because it only explains some of the variance in reading abilities in children (McLean, Stuart, Coltheart, & Castles, 2011) and

deficits on tasks thought to assess magnocellular function may be secondary to other kinds of brain differences that cause a range of processing difficulties across temporal processing tasks. For example, the magnocellular differences may result from their interaction with other disordered cortical or sub-cortical regions (Ben-Yehudah, Sackett, Malchi-Ginzberg, & Ahissar, 2001; Skottun, 2000; Stuart, McAnally, & Castles, 2001).

Taking a similar biological stance to the magnocellular theory, others have explained the temporal processing deficits found in individuals with dyslexia through reference to the efficiency of neuronal networks. For example, Llinas (1993) proposed that the clocking mechanism responsible for control of neural firing and binding cortico-thalamic cortical networks was disrupted in dyslexia, affecting the processing of rapidly occurring information. Tallal (2004) also suggested that the input/output activity of populations of neurons, which allow learning to occur through the plasticity of neuronal networks, may be affected in dyslexia. Any such brain based impairment is proposed to affect the development of appropriate neural representations of sounds in language due to a lack of binding within neuronal populations. Goswami (2011) more recently extended these ideas to a system-wide perspective, proposing that the impairment may lie in the rhythmic oscillations necessary for communication between populations of neurons, particularly the oscillations necessary for sampling the speech signal in the temporal domain.

Differences in the brain do support a difference in neuronal viability in dyslexia, with a decrease in the fractional anisotropy of white matter in the brains of individuals with dyslexia which is correlated with reading performance (Deutsch et al., 2005; Klingberg et al., 2000; Nagy, Westerberg, & Klingberg, 2004; Niogi & McCandliss, 2006; Steinbrink et al., 2008; Vandermosten, Boets, Poelmans, et al., 2012). Fractional anisotropy, measured by MRI based diffusion tensor imaging, indexes the diffusion of water molecules across neuronal pathways and is a surrogate measure for the myelination of neurons. The myelin sheaths surrounding neurons contribute to rapid communication across cortical networks. Although Llinas, Tallal and Goswami do not specify the areas of the brain which may be affected, a recent review indicates that the changes in white matter density may be specific to areas within the reading network (Vandermosten, Boets, Wouters, & Ghesquière, 2012). Moreover, from soon after birth, children at risk of developing dyslexia show differences in the way populations of neurons respond to stimuli (Schulte-Körne & Bruder, 2010) supporting a functional difference in the neuronal firing patterns in the dyslexic brain that may be present early in development before much exposure to language has occurred. Returning to the evidence from genetic studies, some of the risk loci that have been implicated in dyslexia are similarly associated with processes that contribute to neuronal migration and axonal development (Galaburda et al., 2006; Meng et al., 2005; Paracchini et al., 2006). Together these strands of evidence suggest a potential route from genes to behaviour in dyslexia, with a difference in the

efficiency of communication between neuronal networks indexed at the behavioural level by temporal processing deficits.

1.3.3. A multi-factorial perspective

There has been no general acceptance of any one of these theories, although different versions of the temporal processing hypothesis have somewhat dominated the research field. The difficulty found in describing the aetiology of dyslexia is not surprising given that children with dyslexia are a heterogeneous group due to the complexity of factors affecting the development of literacy skills (as detailed in Figure 1.). It is apparent that the presence of dyslexia is governed by a range of risk factors which interact with environmental factors and cognitive development to determine the outcome of the disorder (Thomas & Karmiloff-Smith, 2002). The resultant heterogeneity means that it is unlikely that all individuals with dyslexia will show the same impairments on all tasks that measure behavioural outcomes predicted by the theories (for reviews, see Ramus, 2003; Rosen, 2003). Thus, the theories regarding this developmental disorder can only attempt to explain particular aspects of impairment. More recently, conceptualisations of these developmental disorders take account of their multi-factorial nature and account for the fact that different disorders commonly co-occur with one another (Cramer et al., 2010; Snowling, 2008). This adapted perspective is in contrast to the position that single deficit models, such as the phonological deficit hypothesis, are alone sufficient explanatory models (Pennington & Bishop, 2009; Snowling, 2008). Instead, multiple accounts with overlapping effects are more likely (Pennington & Bishop, 2009), as highlighted by the evidence from behavioural genetics research (Cramer et al., 2010; Plomin & Kovas, 2005).

Despite this position, there is still merit in investigating risk factors that contribute to dyslexia at any level of the framework, providing investigations recognise this broader phenotype perspective and the convoluted route to development. Such research does not weaken the significance of any particular contributing risk factor within the framework of reading development (Hulme et al., 2005), but can contribute to our understanding of other levels of explanation. For example developing an understanding of which particular cognitive impairments are linked to phonological development and where such cognitive processes occur within the brain will help direct research when more information is gained about neural or genetic mechanisms of influence. Similarly, such investigations can provide more specific areas of functioning for remediation strategies to target.

In the present thesis, one particular cognitive risk factor is considered which is itself encompassed within the sphere of temporal processing. Specifically, the impairments in motor timing commonly found in children with dyslexia (Badian & Wolff, 1977; Klipcera, Wolff, & Drake, 1981; Thomson & Goswami, 2008; Wolff, Cohen, & Drake, 1984; Wolff, 2002) are investigated in detail. As will be described in greater detail in forthcoming chapters, motor timing is a task that involves processing in

the temporal domain, and so is most closely aligned with a broad version of the temporal processing hypothesis, in which a temporal processing impairment affects the appropriate development of sound based representations of language (although considering the summary above, a range of explanatory accounts may be appropriate). This impairment is of interest because it has been found to predict genetic risk for dyslexia (Wolff, Melngailis, & Kotwica, 1996; Wolff, Melngailis, Obregon, & Bedrosian, 1995) and deficits in motor timing predict reading and phonological ability across populations of good and poor readers (Thomson, Fryer, Maltby, & Goswami, 2006; Thomson & Goswami, 2008). As a risk factor associated with dyslexia, a temporal processing deficit is also attractive because it is consistent with the generalist nature of developmental disorders championed by the behavioural genetic evidence. Specifically, temporal processing difficulties are found in other developmental disorders such as autism and ADHD (Noreika, Falter, & Rubia, 2013a; Toplak, Rucklidge, Hetherington, John, & Tannock, 2003; Wimpory, 2002) and any biological mechanisms underlying temporal processing behaviour are likely contributed by a number of genetic risk factors.

The following two chapters provide an overview of motor timing that is not limited to the field of developmental dyslexia (Chapter 2) as well as a review of the literature on motor timing impairments in dyslexia (Chapter 3). The reviews in these two chapters motivate the research questions assessed in the later experimental chapters which are explained in full in Chapter 3.

2. Processing in time: An overview

2.1. Chapter overview

In subsequent chapters the relationships between aspects of motor timing and the symptoms of developmental disorders are investigated. Here I present an overview of research on time-based processing that will be relevant to these later investigations and which helps to motivate the data analysis strategy selected.

2.2. Processing in time

Temporal processing is ubiquitous across many domains of human behaviour. Time-based processing is as important as processing in other dimensions, such as spatial processing, because of the inherently temporal nature of our environment and the need for behaviour to occur in a timed and often predictable fashion. Moving, communicating and decision-making all involve judgements of time, whether to make movements occur synchronously with events occurring in our surroundings, to judge the length of a sound in perceived speech, or to make conditional decisions based on prediction of future events. The temporal limits of our behaviour and cognition are in turn constrained by the temporal limits of neural codes (Cohen, 2011) and different mechanisms have evolved to allow processing across the range of temporal scales necessary for survival (reviews are provided by Buhusi & Meck, 2005; Gibbon, Malapani, Dale, & Gallistel, 1997; Mauk & Buonomano, 2004). Circadian processes, such as sleep-wake, gestation or reproductive cycles, occur over long periods of days, months or years. Processes such as problem solving or decision making, occur over durations of minutes and seconds, and involve processing based on interval timing. At yet smaller magnitudes, timing for movement control for action and speech occurs on a sub-second scale and is commonly termed millisecond timing.

These distinctions are made on the basis that timing over these different scales is controlled by different cognitive or neural mechanisms. Qualitative differences in the properties of timing behaviour have been found using different magnitudes of temporal interval. For example, when humans perform sensorimotor synchronisation paradigms (finger tapping to a regular beat), the response intervals produced typically adhere to the 'scalar property' of timing when the pacing stimulus intervals are less than one second in length (Gibbon et al., 1997). This property is akin to Weber's Law, such that the coefficient of variation (standard deviation of response intervals divided by mean response interval) remains constant across intervals, because they are under control of a similar functional mechanism (Buhusi & Meck, 2005; Gibbon, 1977). Deviations from this scalar property and in temporal sensitivity or duration discrimination abilities are found for intervals longer than 1 or 1.3 seconds (Drake & Botte, 1993; Lavoie & Grondin, 2004; Madison, 2001a), indicating a change in the mechanism controlling time processing for intervals longer than about one second.

These differences in the precision of timing likely reflect the functional basis of behaviour, with a need for accuracy in behaviour that occurs over short intervals where the cost of making errors in behaviour is high (Madison, 2001b). In comparison, temporal precision is less critical for behaviour occurring over longer intervals. Generally uniform performance precision is found for intervals in the range of 250-1000ms (4-1Hz[1]) (Madison, 2001a), although the exact interval magnitude at which changes in the scalar property occur varies depending on the task used to measure performance (Buonomano, Bramen, & Khodadadifar, 2009).

As will be described in later chapters, studies assessing motor timing in developmental disorders have typically focused on intervals in the sub-second range. This review therefore primarily focuses motor timing tasks involving sub-second intervals, with reference to other types of tasks and scales to situate the research within existing understanding of timing behaviour. The terms "temporal processing" and "processing in time" are used in reference to the processing of sensory events which occur over time and/or the ability to produce events in time. The terms are not used to refer to neural processing of time per se, although it is assumed that neural mechanisms are responsible for these processes.

2.3. Neural basis of timing

A variety of research methodologies have helped to establish the neural mechanisms which contribute to timing control and have helped to demonstrate the differences between timing over millisecond and interval scales. There is a degree of overlap in the neural systems recruited for millisecond and interval timing, allowing refinement of temporal resolution for tasks where processing over multiple scales can be beneficial (Buhusi & Meck, 2009a; Wiener, Turkeltaub, & Coslett, 2010). Below, a summary is provided of the neural basis of timing, with reviews presented elsewhere (Buhusi & Meck, 2005; Gibbon et al., 1997; Grondin, 2010; Lewis & Miall, 2003; Pollok, Gross, & Schnitzler, 2006; Wiener et al., 2010); the main brain areas discussed are summarised in Figure 2.1 and Table 2.1.

Neuroimaging studies have shown that millisecond timing (typically measured using sensorimotor synchronisation tasks) recruits a network that operates without inputs from higher cognitive areas such as the frontal cortex. This network includes the posterior cerebellum, sensorimotor, pre-motor and auditory cortices and the subcortical structures of the basal ganglia (including the putamen and global pallidus) (Buhusi & Meck, 2005; Buonomano et al., 2009; Ivry, 1996; Lewis & Miall, 2003; Mauk & Buonomano, 2004; Meck & Benson, 2002; Rammsayer, 1999; Wiener et al., 2010). Interval timing,

[1] Inter-onset intervals are provided in milliseconds throughout this thesis to allow precise comparisons between study methodologies. They are also presented as Hz for ease of conversion because both milliseconds and Hz are regularly used in the timing and temporal processing literature. A conversion chart is also provided in the Appendix.

for tasks such as decision making or planning, engages overt cognitive processing to a greater extent, operating through a thalamo-cortical-striatal network which includes the basal ganglia, prefrontal and posterior parietal cortices (Buhusi & Meck, 2005; Buonomano et al., 2009; Grossberg & Merrill, 1996; Lewis & Miall, 2003, 2009; Meck & Benson, 2002; Rammsayer, 1999). The engagement of higher cognitive (especially frontal) areas allows for conscious awareness and control of supra-second interval processing and integration of functions such as attention, memory and decision making (Bueti & Walsh, 2009; J. L. Chen, Penhune, & Zatorre, 2008; Hinton & Meck, 2004; Meck & Benson, 2002; Rammsayer & Lima, 1991; Rammsayer, 2006).

The distinctions between millisecond and interval timing are borne out by investigations of the changes brought about by pharmacological treatments or brain injury or disease. Pharmacological agents that specifically target dopaminergic functions within the basal ganglia lead to specific impairments on timing tasks that involve the processing of millisecond duration intervals (Rammsayer, 1993, 1997). In contrast, agents that affect prefrontal executive functions alter processing of longer time intervals in the range of seconds (Rammsayer, 1992, 1997, 1999). Patients with Parkinson's disease, which damages cells of the substantia niagra and affects the secretion of dopamine from the striatum, also show difficulties with millisecond timing tasks (Harrington, Haaland, & Hermanowicz, 1998; O'Boyle, Freeman, & Cody, 1996) which are independent of any movement difficulties associated with the disease (Buhusi & Meck, 2005; Harrington et al., 1998). Similarly, patients with focal basal ganglion lesions have impairments on both timed motor tasks and tasks requiring the perception of changes in the tempo of sequences (Schwartze, Keller, Patel, & Kotz, 2011).

This evidence suggests that the basal ganglia are important for millisecond timing however the subcortical nuclei are also implicated in supra-second interval processing. The extensive projections from the basal ganglia through the thalamic nuclei to cortical areas, including through the dorsal putamen to the supplementary motor area (SMA) and through the caudate nucleus to the dorso-lateral prefrontal cortex (DLPFC) (Buhusi & Meck, 2005; T. K. Lee, Chau, & Leong, 1995) are thought to allow influence over a range of temporal magnitudes. Thus the neurons of the striatum can code different stimulus interval durations and may act as a reference to the temporal structure of stimuli for both the perception of time and the production of timed responses (Schwartze et al., 2011). The temporal coding provided by the striatum can therefore be used to monitor the synchronicity of events on a millisecond basis or to contribute to the prediction of future events through interactions with higher cognitive areas.

The cerebellum is also implicated in a range of timing tasks, including millisecond timing. The neurons of the cerebellum are more densely arranged than in any other area of the brain and the structure has an abundance of feed-forward and feedback connections to many areas of the cerebral

cortex, brainstem and spinal cord (Glickstein, 2007). The sensorimotor cortices are reciprocally connected to lobules IV, V and VI that are located laterally in the superior cerebellum and these areas are activated by bodily movements (Stoodley & Schmahmann, 2009). The prefrontal and association cortices are connected to the lateral portions of the posterior cerebellar hemisphere and are activated when processing emotions or conducting complex decision making (Stoodley & Schmahmann, 2009). The cellular architecture, with many inputs and synapses, makes the cerebellum well suited to helping refine signal patterns and improve the specificity of signal characteristics, such as those coding duration or intensity of a stimulus (Booth et al., 2007; Houk et al., 2007; Ivry & Justus, 2001; Mauk & Buonomano, 2004; Stoodley & Stein, 2012; Strata et al., 2009). Although involved in many types of cognitive processing, the cerebellum's role in timing is thought to be to optimising responses in temporal processing tasks, especially those involving movement (Houk et al., 2007; Stoodley & Stein, 2011). It is unlikely that the cerebellar contribution to timing stems purely from its involvement in motor functions, because the cerebellum is implicated in timing control regardless of the need for movement in tasks (Tesche & Karhu, 2000).

Damage to the cerebellum can affect both sub- and supra-second interval processing depending on the location of the damage to the cerebellar lobes (Mangels, Ivry, & Shimizu, 1998). Patient studies support a role for the posterior cerebellum (medial to lateral regions) in sub-second processing (Harrington, Lee, Boyd, Rapcsak, & Knight, 2004; Ivry, Keele, & Diener, 1988; Ivry & Keele, 1989) and for the superior lobules in motor timing (Kawashima et al., 2000; Penhune, Zattore, & Evans, 1998; Rao et al., 1997). When regional transcranial magnetic stimulation (rTMS) has been applied to the left or right lateral cerebellum, temporarily interrupting neural communication in each region, impairments are found in sub-second duration discrimination, duration reproduction and finger tapping synchronisation tasks with no comparative impairment found for supra-second processing (Koch et al., 2007). Focal lesions to the cerebellum also support a role in a variety of timing functions. Damage to areas of the posterior cerebellum have been linked to impairments in time reproduction (e.g. motor timing) but not duration judgement (Harrington et al., 2004) whereas lesions to the superior cerebellum, particularly the vermis, affect duration judgements but not judgements based on beat-based sequences (Grube, Cooper, Chinnery, & Griffiths, 2010).

The precise mechanism of cerebellar involvement in temporal processing behaviour is not yet well understood. It has been suggested that the cerebellum may act as an event timer or stop-watch for sub-second timing (Grube et al., 2010; Ivry & Richardson, 2002), taking advantage of the cerebellum's ability to extract temporal information from perceived sensory signals (Harrington et al., 2004; Penhune et al., 1998). Alternatively the cerebellum may act to signal the degree of synchrony between actions and perceptions (Grube et al., 2010; Houk et al., 2007), allowing feed-forward prediction of future events (Mauk & Buonomano, 2004) and working in conjunction with other areas of the timing network to achieve temporally based tasks. It is proposed that in the

cerebellum somatosensory inputs are compared to events such as motor responses and the degree to which these events concur with the anticipated outcome of behaviour is estimated (Doya, 2000; Miall & Reckess, 2002; Tesche & Karhu, 2000).

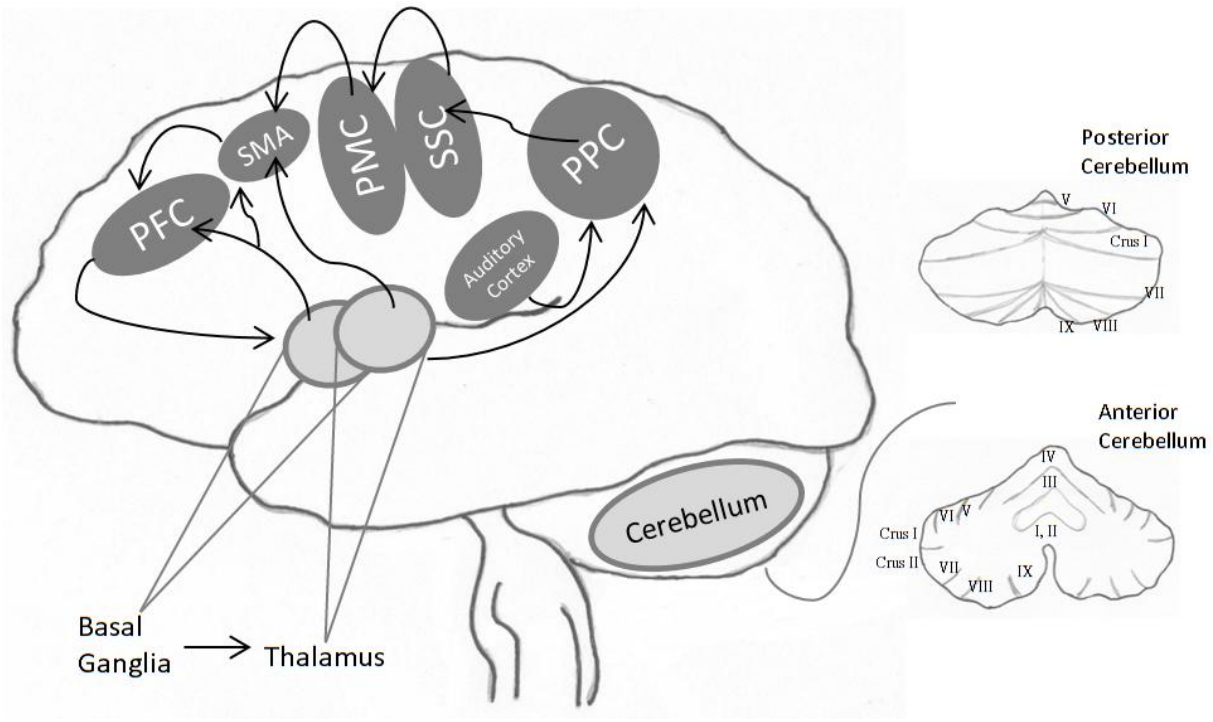

Figure 2.1: Network of thalamic, cortical, striate and cerebellar regions involved in timing functions
PFC (Prefrontal cortex), SMA (Supplementary Motor Area), PMC (Primary Motor Cortex), SSC (Somatosensory Cortex), PPC (Posterior Parietal Cortex). Black arrows represent direction of inputs to areas for timing. Cerebellum Lobules labelled I-IX with feed-forward and feedback connections to cortical and subcortical areas. Lobules IV, V, VI connect to motor cortices; Lobules VII and Crus I and II connect to prefrontal and posterior parietal cortices; Lobules I-V, VI and VIII connected to limbs via spinal cord and lateral areas related to timed motor responses.

Other brain areas which contribute to millisecond timing tasks have been recently summarised by Wiener et al. (2010) following a voxel-wise meta-analysis of neuroimaging studies, comparing the brain areas activated by various timing tasks. In addition to the basal ganglia and cerebellum, activations were consistently found during sub-second temporal processing in the right inferior parietal lobe (IPL), medial areas of the pre-central gyrus and left inferior frontal gyrus (IFG). The IPL likely contributes to timing control by monitoring synchrony between multisensory information, forming part of the cerebro-striatal network for timing with projections to the basal ganglia (Battelli, Pascual-Leone, & Cavanagh, 2007; Wiener et al., 2010). Areas of the pre-central gyrus contribute to both perceptual and motor timing tasks, and the role of this region is therefore thought to be not limited to motor control. Instead, Wiener et al. propose it has a role in information rehearsal, with the activation in sub-second tasks associated with the hand area of the motor homunculus and in the supra-second task associated with the mouth area, perhaps for vocal rehearsal or counting during performance. Activity in the IFG was found to be strongest when processing sub-second intervals and is therefore linked to the maintenance of a regular beat. Similar conclusions were reached by

Grahn and colleagues, suggesting the IFG supports beat-based processing such as synchronisation to or production of highly regular outputs (Grahn & Brett, 2007; Grahn & McAuley, 2009).

Table 2.1: Summary of brain areas involved in timing functions

Brain Area	Interval Duration		Temporal Processing Function Implicated
	Sub-second	Supra-second	
Cerebellum [A,B,C,D,E,F,G,H] (posterior/superior)	✓	✓	Optimisation of responses & predicting future stimuli or responses
IPL [H,I]	✓		Monitoring synchrony between multi-sensory stimuli
Sensorimotor cortices (PCG) [H]	✓	✓	Motor or vocal rehearsal
PMC [J,K,L]	✓	✓	Motor tasks and entrainment
SMA [H,M,N,O,P]	✓	✓	Accumulator of time
BG [H,Q,R,S,T] (via putamen & GP or via caudate) [U,V]	✓	✓	Monitor Synchrony & contribute to prediction of future stimuli
Thalamus [M,W,X,Y,Z]		✓	Relay for supra-second and decision based timing
Pre frontal [H,a,b,c,d]		✓	For temporal tasks requiring decision making, working memory or attention
IFG [H,M,e]	✓	✓	Processing regular beats

A Harrington et al. (2004); **B** Houk et al. (2007); **C** Ivry et al. (1988); **D** Koch et al. (2007); **E** Mangels et al. (1998); **F** Mauk & Buonomano (2004); **G** Stoodley & Stein (2011); **H** Wiener et al. (2010); **I** Battelli et al. (2007); **J** Arnal (2012); **K** Lakatos, Karmos, Mehta, Ulbert, & Schroeder (2008); **L** Rao et al. (1997); **M** Grahn & Brett (2007); **N** Konoike et al. (2012); **O** Lewis & Miall (2003); **P** Pouthas et al. (2001); **Q** Harrington et al. (1998); **R** O'Boyle et al. (1996); **S** Rammsayer (1993, 1997); **T** Schwartze et al. (2011); **U** Buhusi & Meck (2005); **V** Lee et al. (1995); **W** Dhamala et al. (2003); **X** Penhune et al. (1998); **Y** Pollok, Krause, Butz, & Schnitzler (2009); **Z** Schubotz, Friederici, & von Cramon (2000); **a** Bueti & Walsh (2009); **b** Chen et al. (2008); **c** Hinton & Meck (2004); **d** Meck & Benson (2002); **e** Grahn & McAuley (2009).

The insula and SMA are implicated in a range of timing tasks, both motor and perceptual, sub- and supra-second tasks, contributing to a more general timing network (Wiener et al., 2010). The SMA (most likely the pre-SMA) is active during tasks which require time intervals to be reproduced, with greater activation when processing long intervals (Pouthas et al., 2001). The role of the SMA is likened to an accumulator of time where the pulses used to represent intervals (which may be produced over time by a timekeeper-like mechanism) are stored. However, Macar et al. (2002) instead proposed that parts of the SMA work in conjunction with other parts of the timing network such as the striate cortex and thalamic nuclei to encode the passage of time, recognising that the SMA is also activated during other types of motor functioning, such as during planning or implementation of motor sequences (Nachev, Kennard, & Husain, 2008). The insula also contributes to a range of cognitive functions that are related to timing control, such as counting or learning, and by integrating physiological signals from the body the insula is thought to contribute to the appreciation of the flow of time (Wittmann, 2009). The auditory association cortices also assist in temporal processing by helping maintain auditory representations of temporal stimuli, with activations demonstrated in the superior temporal lobe during sub-second timing (Lewis & Miall, 2003; Rao et al., 1997). The additional demands of supra-second timing tasks are associated with

activity in frontal areas of the brain and these are thought to reflect working memory and other executive functions (Wiener et al., 2010).

2.4. Task parameters

2.4.1. Timing paradigms

As highlighted in the previous section, distinctions are commonly drawn between tasks that assess the processing of different time interval durations. In addition, the type of timing task employed influences the neural systems recruited for that task (Macar & Vidal, 2009; Wiener et al., 2010), with perceptual timing tasks activating different brain areas to motor tasks and differences also resulting from the degree of cognitive control, attention and decision making demanded (Wiener et al., 2010). A summary of the tasks often used to measure temporal processing is provided in Table 2.2. Similar to the division drawn between sub- and supra-second timing, the methodologies are separated into those involving explicit or implicit timing. Explicit timing tasks require attention to be directed towards the passage of time (e.g. verbal estimates of time periods, reproduction or production of interval durations, comparison of intervals and interval bisection tasks) whereas implicit tasks assess behaviours that have an implied need to encode the passage of time (e.g. judging temporal order or simultaneity, sensorimotor synchronisation, gap detection and anticipation of intervals; Grondin, 2010; van Wassenhove, 2009). In the table, implicit tasks are divided further, depending on the need for decision making to demonstrate, for example, the difference between temporal order judgements (overt decision) and motor timing tasks (production, without decision).

Explicit timing tasks typically assess the perception of durations greater than 1 second, using either prospective and retrospective methods of assessment (Grondin, 2010; Zakay & Block, 2004). In retrospective timing paradigms participants are unaware of the need to consciously attend to the interval duration. These tasks are similar to remembering event durations in real-life situations, such as how long it took to get dressed this morning, but are difficult to use experimentally because participants typically become aware of the nature of the task after the first trial. In prospective timing tasks, participants are informed of the need to attend to the passage of time. Intervals can be presented as single stimuli (e.g. a tone) or as an empty interval bounded by two stimuli. Estimates of temporal durations can be made through verbal reports (e.g. “ten seconds” or “one minute”) or through the production of an interval of the same length (e.g. via a button press). More precise perceptual thresholds for duration discrimination can be gained through use of two-alternative forced choice (2AFC) paradigms or bisection tasks where participants determine whether a stimulus is longer or shorter than an exemplar interval.

Table 2.2: Temporal processing tasks

Task	Description
Explicit: Attention directed to time or judgement	
Prospective judgements	Tasks where participants are aware beforehand that a judgement of temporal characteristics will be required
Retrospective judgements	Tasks assessing the memory for encoded time, where participants are unaware that a judgement of temporal characteristics will be required
Duration Estimation	Judging the size of an interval
- verbal estimation	Estimation of duration by verbally labelling the time elapsed
- single stimulus estimation	Estimating whether a single stimulus is, for example, "short" or "long"
- interval production	Estimation of duration using a movement e.g. moving a slide along a track
- interval reproduction	Pressing a button for the estimated duration
- comparison judgements	Comparing the duration of two intervals, often a 2AFC task
- bisection	Compare the length of an interval to the length of two previously learned probe intervals
Implicit-Judgement: Tasks relying on the encoding of time or processing of rate which include a judgement or decision element	
Individuation	Judgement of whether one or more items were presented
- gap detection	Detecting the presence of a gap in an ongoing auditory stimulus such as noise
- flicker fusion	Detecting whether a rapidly changing stimulus (e.g. visual LED) is flickering between two states or appears as constant
Ordering in Time	Judging the order in which stimuli are presented (requires individuation)
- temporal order judgements	Judging the order in which two or more stimuli are presented (requires individuation)
- sequencing	Producing sequences in the correct order e.g. rhythmic intervals with varied durations
Implicit: Tasks relying on the encoding of time or processing of rate without an explicit judgement being made	
Motor Production (discrete or continuous)	Producing intervals by making a bodily movement (related to interval production described above). Discrete tasks involve movements with distinct start and end points e.g. finger tapping. Continuous tasks involve oscillating movements such as circle drawing
- paced (synchronisation)	Producing movements in time with a stimulus, typically an isochronous stimulus with rapid intervals
- phase or period changes in paced timing	Producing movements in time with a stimulus, with alterations to the overall rate (period shifts) or to individual stimuli (phase shifts) requiring responses to be updated
- unpaced (continuation or self-paced)	Producing isochronous or rhythmically timed movements without a pacing stimulus, sometimes following a synchronisation phase (continuation) or self paced
Coincident Timing: Natural behaviours that rely on temporal encoding or production in time e.g. catching or hitting a ball	

With the need for an overt estimation of the passage of time or a judgement of time, these explicit tasks involve higher cognitive processing using attention, decision making and memory (Droit-Volet, 2013; Grondin, 2010). This makes it more difficult to determine whether behavioural performance on these tasks should be attributed to time based processing mechanisms or other cognitive processes. Imaging studies conducted with humans and primates support a role for more extensive cognitive processing in these explicit perceptual tasks with activity recorded in the insula, DLPFC and parietal cortices which is often absent in implicitly timed tasks (Lewis & Miall, 2003; Rao, Mayer, & Harrington, 2001; Wiener et al., 2010). In a review of primate studies Lewis & Miall (2003) compared brain activity for discrete judgements of temporal intervals to that for timing predictable continuous motor events (implicit tasks). The activity in these regions is thought to relate to the need for executive control, directed attention and planning in these explicit timing tasks (Lewis & Miall, 2006; Smith, Taylor, Lidzba, & Rubia, 2003).

The implicit tasks listed in Table 2.2 in comparison do not require attention to be directed to the passage of time. Thresholds for temporal processing are often measured via gap detection or stimulus individuation tasks which assess the smallest interval between two stimuli that can be reliably detected to yield a perception of two separate stimuli rather than a single continuous event. Forced choice tasks, employing judgements about which stimuli came first or which stimulus contained a gap are typically used to measure thresholds for these aspects of perception (e.g. order, gap detection). The use of psychometric functions in these choice tasks allows estimates of the threshold of perception to be calculated and these tasks are therefore rather more objective than the explicit decisions tasks described above. Other tasks in this group require the detection of stimuli within ongoing trains of stimuli or judgements about the temporal order of discrete stimuli.

Such tasks do, however, require a conscious judgement to be made about remembered stimuli and may often be unreliable in circumstances where participants have difficulties with attention or memory, such as in developmental or ageing populations or those with clinical disorders (McCormack, Brown, Maylor, Richardson, & Darby, 2002; Pouthas & Perbal, 2004; Witton et al., 2012). Indeed, when gap detection tasks have been used with children, discrepancies have been found between the thresholds estimated by psychophysical methods and those estimated through recordings of the evoked neural response to stimulus gaps using MEG (magnetoencephalography) (Diedler et al., 2009). As in the explicit tasks described above, the involvement of memory and decision making processes means that additional brain systems such as the prefrontal cortex and inferior parietal regions are recruited as well as regions contributing to temporal processing (Macar & Vidal, 2009; Rao et al., 2001; Wearden & Ferrara, 1993; Wearden, 2003; Wiener et al., 2010). The reliance on memory in these tasks may also mean that temporal processing is limited by the need for

stimulus or interval comparisons and storage or retrieval from memory (Mauk & Buonomano, 2004). Such processes will give rise to additional variability beyond the core timing mechanisms involved in sensorimotor temporal processing tasks.

The final class of implicit tasks that require encoding of time without overt judgements include a class of motor timing tasks where participants are required to produce responses repeatedly in time with an external stimulus. Responses are typically produced through movement of a motor effector, such as a finger, recorded through a touchpad. These timing tasks require production of a continuous stream of predictable movements over time, defined by the movement pattern. In comparison to the tasks described above, these motor timing tasks require relatively little higher cognitive control (unless very large the inter-stimulus-intervals are used e.g. >1.5 seconds) (Buhusi & Meck, 2005; Gibbon et al., 1997; Hinton & Meck, 2004). Attention may be initially involved to instigate the motor response, however, once a participant is entrained to the stimulus rate, synchronisation can continue with little attention to the task. Because entrainment can arise even in the absence of a conscious intention to synchronise, this type of response is thought to be controlled by automatic rather than conscious cognitive processing mechanisms (Lewis & Miall, 2003; Repp, 2002, 2005).

The automaticity of responses on these tasks is thought to stem from the relatively direct links between the timing control system and the motor output system (Lewis & Miall, 2003; Mauk & Buonomano, 2004). Many areas of the timing system (including the right cerebellum, pre-motor cortex, SMA and basal ganglia) are active during timing tasks irrespective of the requirement for physical movements (Grahn & Brett, 2007; Konoike et al., 2012; Lewis & Miall, 2003). Areas of the sensorimotor system, basal ganglia and the associated feed-forward loops through the cerebellum are also implicated in the preparation of timed movements, the anticipation of future events and for the monitoring of rhythmic stimuli (De Guio, Jacobson, Molteno, Jacobson, & Meintjes, 2012; Gibbon et al., 1997; Jantzen, Steinberg, & Kelso, 2004; Mauk & Buonomano, 2004; Meck, 2005). Furthermore motor timing tasks (tapping a finger to a beat or tapping unpaced) are found to activate a route to the dorsal dentate nucleus of the cerebellum (which projects to the pre- and primary motor areas) rather than to the ventral dentate nucleus (which projects to higher processing areas such as the DLPFC) (Rao et al., 1997).

It is thought that the motor system contributes to timing control by coding the temporal properties of stimuli using low frequency neural oscillations within the motor system (Arnal, 2012; Gerloff et al., 1998; Lakatos et al., 2008). Such mechanisms may account for the high temporal acuity that can be achieved by the motor system in comparison to the perceptual system (Repp, 2001a, 2002, 2005). To demonstrate this acuity Repp used a task in which participants tapped their finger in time to a beat and errors were created by adding a phase shift to one of the stimulus beats, thus making it

shift forwards/backwards in time with respect to the entrained beat. Participants were able to correct changes as small as 6ms, even in the absence of any conscious awareness of these changes, demonstrating that the motor system is capable of temporal processing at a smaller magnitude than the perceptual systems (Repp, 2002; Thaut, Tian, & Azimi-Sadjadi, 1998), which is able to detect temporal changes in the order of 20ms (when tested via temporal order judgement tasks) (Fink, Churan, & Wittmann, 2005). In the absence of the need for attention, memory or overt decision making for task completion, these motor timing tasks therefore provide more effective access to the central mechanisms controlling timing behaviour than the explicit or judgement dependent tasks.

Before examining motor timing tasks in more detail, it is notable that another distinction is often drawn in the timing field between "discrete" and "continuous" motor timing tasks. Discrete tasks involve a movement with distinct start and end points, such as the up and down movement of the finger tap. Continuous tasks, like repeated circle drawing, involve oscillatory forms of movement. There is some evidence that duration discrimination is correlated with timing ability on discrete motor timing tasks (finger tapping) but not with performance on continuous (circle drawing) tasks (Repp & Steinman, 2010a; Zelaznik, Spencer, & Ivry, 2002). The continuous timing tasks are therefore thought to incorporate a different mode of temporal processing to the discrete tasks, whilst elements of timing control are shared between the discrete-implicit and explicit tasks shown in Table 2.2. Some authors classify discrete tasks as "explicit" (e.g. Zelaznik, Spencer, & Ivry, 2002), although here I have referred to "explicit" tasks as those requiring overt decisions about time (in the upper rows of Table 2.2).

2.4.2. Motor timing paradigms

All of the temporal processing tasks described above help to inform about the mechanisms of control of timed behaviour. As the focus of this thesis is on the application of the motor timing paradigms to the analysis of temporal processing behaviour in children with developmental disorders, the remainder of this chapter focuses on methodological features of this particular paradigm that can affect performance. This review provides a basis for the examination of the research presented in subsequent chapters.

2.4.2.1. Presence of pacing stimuli

Synchronisation or synchronisation-and-continuation tasks have been frequently used to study motor timing behaviour (e.g. Bolbecker et al., 2011; Dhamala et al., 2003; Harrington et al., 1998, 2004; Jancke, Loose, Lutz, Specht, & Shah, 2000; Rao et al., 1997; Repp, 2005; Rubia, Noorloos, Smith, Gunning, & Sergeant, 2003; Schubotz, Friederici, & von Cramon, 2000; Wing & Kristofferson, 1973a, 1973b; Wolff, 2002). Synchronisation tasks require a repeated motor response to be generated at the same rate as the pacing stimuli. Typically a finger tap is used to respond to the stimulus, although other effectors can be used. The presence of the stimulus during responding

allows online updating of response timing with respect to the occurrence of the stimulus. In synchronise-and-continue tasks, the synchronisation phase is followed by a continuation phase. In the latter the stimuli are discontinued and the participant is required to maintain the same response rate until the end of the trial. In the absence of stimuli, this task relies more heavily on internal timekeeping systems to maintain control of the timed output responses.

Where stimulus (and therefore response) intervals are shorter than about 1.8 seconds, behaviour on both of these tasks is thought to be driven by the oscillatory activity of populations of neurons in the brain coupled to the occurrence of stimuli events, rather than by any cognitive judgement of elapsed time (Arnal, 2012; Gerloff et al., 1998; Lakatos et al., 2008; Macar & Vidal, 2009; Nozaradan, Peretz, Missal, & Mouraux, 2011; Nozaradan, Peretz, & Mouraux, 2012; Pollok, Gross, Müller, Aschersleben, & Schnitzler, 2005; Pollok et al., 2006; Repp, 2005). The oscillatory phase of neuronal population activity is coupled with the period of the stimulus, providing information about the temporal dynamics of the stimulus, allowing an automatic entrainment of responses to the stimulus interval (Engstrom, Kelso, & Holroyd, 1996). As such, the occurrence of each subsequent stimulus is predicted by the system so that synchrony can be maintained. This property of timing is evidenced by the presence of the "anticipation response", where the physical tapping response precedes the onset of the next pacing stimulus (by about 50-100ms), a feature which is regularly observed in studies of motor synchronisation (Aschersleben, 2002; Repp, 2005; Vos, Mates, & van Kruysbergen, 1995). This phenomenon demonstrates that synchronisation is achieved through the internal code which predicts the next stimulus rather than by a motor response to the stimulus (as in a reaction time task).

Several theories have been put forward to explain the anticipatory nature of the motor output (for reviews see Aschersleben, 2002; Repp, 2005). One hypothesis is that the differences in transmission times in the different sensory systems (e.g. tactile/kinaesthetic and auditory) result in a difference in the time at which the signals from the two systems arrive centrally in the brain. To overcome this difference the motor response is initiated before the auditory stimulus occurs to give rise to a perception of synchrony. Further analysis of this anticipatory response has shown that the degree of asynchrony is dependent on the detection of the "moment of occurrence" of the pacing stimulus (the perceptual centre or p-centre) (Vos et al., 1995). The "moment of occurrence" is temporally later than the physical onset of the stimulus and is the moment at which the stimulus is perceived to have occurred. As such, for auditory stimuli, participants do not synchronise their taps with the onset of the stimulus but with the perceptual centre of the stimulus, and the degree of anticipation is affected by the duration and the shape of the amplitude envelope of the auditory stimulus. Entrainment to stimuli allows prediction of future events and evaluation of the accuracy of timing.

For a synchronise-and-continue task, the entrainment achieved during the synchronisation phase is maintained during the continuation phase and leads to activity in the same thalamo-cortical-cerebellar timing network (including sensorimotor cortices) activated during the synchronisation phase (Jantzen et al., 2004). Typically during the continuation phase an increase is found in both the degree of activity (i.e. increase in power of EEG signals or increased oxygenated blood flow measured through MRI) and in the coupling between areas in the network (Gerloff et al., 1998; Jantzen, Steinberg, & Kelso, 2002; Pollok et al., 2006; Rao et al., 1997). In addition, activity in the inferior frontal gyrus during the continuation phase is thought to reflect the maintenance of the memory trace for the entrained tempo in auditory sensory memory (Rao et al., 1997). Performance during the continuation phase provides a measure of the characteristics of internal timekeeping mechanisms, in the absence of synchronisation stimuli to guide responses.

2.4.2.2. Form of movement responses

Most studies assessing behavioural performance on these synchronisation and continuation tasks have recorded the precision of intervals between finger tap responses. (e.g. Aschersleben, 2002; Aschersleben & Prinz, 1995; Harrington et al., 2004; Ivry et al., 1988; Madison, 2001a; Pollok et al., 2006; Rao et al., 1997; Vorberg & Wing, 1996; Wing & Kristofferson, 1973b). These responses have distinct start and end points and can be recorded on a response pad. This simple motor response is thought to have relatively efficient connections with the motor system (Mauk & Buonomano, 2004). As a discrete movement pattern it is more likely to engage the timing system associated with other forms of temporal processing (e.g. time perception) compared to continuous movements such as circle drawing (Spencer, Zelaznik, & Ivry, 2003; Zelaznik et al., 2002) which do not have an easily determined start point and result in less accurate and more variable timing responses (Elliott, Welchman, & Wing, 2009). The finger tap is also an over-learned movement response that is suitable for use with a range of participant populations. It is possible to measure timing using other motor effectors such as the arm or foot, however these limbs show slower and more variable movement times than finger movements (Aschersleben & Prinz, 1995; Hoffmann & Hui, 2010; Hoffmann, 1991).

Typically, where the characteristics of timing performance have been examined, one-handed, unimanual tapping tasks have been employed (Aschersleben, 2002; Patel, Iversen, Chen, & Repp, 2005; Wing & Kristofferson, 1973a, 1973b). However, the properties of bimanual, two-handed finger tapping tasks have also been investigated (Drewing & Aschersleben, 2003; Helmuth & Ivry, 1996). The latter typically require simultaneous tapping of one finger from each hand, either in synchrony or in an alternating fashion. Helmuth and Ivry (1996) found that variability of inter-response intervals was lower for simultaneous bimanual finger tapping performance than for unimanual movements and statistical decomposition of the timing variability suggested that this “bimanual advantage” arose from a process of averaging across multiple timekeeping mechanisms (one for each motor effector; Helmuth & Ivry, 1996). Later investigations did not support this model and instead

suggested that the bimanual advantage results from availability of additional sensory feedback from the two hands (Drewing & Aschersleben, 2003). The latter position is supported by evidence from neuroimaging studies, with bimanual timing tasks recruiting a similar neural network to unimanual tasks, including the SMA, primary and pre-motor cortices and the cerebellum, but with increased activity in the network when information is shared between effectors (Aramaki, Osu, & Sadato, 2010; Banerjee, Tognoli, Kelso, & Jirsa, 2012; Toyokura, Muro, Komiya, & Obara, 1999, 2002). As with any bimanual coordination task, some information is shared across the corpus callosum, however, callostomy patients retain the bimanual advantage suggesting that the coupling between effectors occurs in sub-cortical in structures such as the basal ganglia or cerebellum (Ivry & Hazeltine, 1999).

2.4.2.3. Isochronous and anisochronous stimuli

Another consideration in timing research is the type of rhythmic stimuli implemented within a task. Regular isochronous or metronome-like stimuli have typically been used in the synchronisation and continuation studies already described. An alternative is to use stimuli with a rhythmic structure that involve series of repeated phrases of stimuli and silence that may contain repeated elements at smaller temporal scales with varying tempos and metrical structures (strong and weak beats) (Geiser, Ziegler, Jäncke, & Meyer, 2009; Grahn & Brett, 2007; Grube & Griffiths, 2009; Konoike et al., 2012). Perception of rhythm in this type of sequence (or in music) is achieved through the hierarchical organisation and temporal coordination of these elements (Cummins & Port, 1998). Rhythmic stimuli have been used to assess temporal processing behaviour using temporal judgement paradigms (e.g. compare two phrases) or synchronisation and reproduction paradigms where participants match or repeat phrases through motor responses (e.g. Grahn & Brett, 2007). In synchronisation paradigms the goal can be to replicate all stimuli in the musical phrase or to synchronise to the beat-based structure (e.g. 4:4 time) or accents within the phrase. These different tasks involve a similar neural network to that used for isochronous timing tasks (described in 2.3), namely a network including the pre-motor and sensorimotor areas, cerebellum, thalamus and basal ganglia (Dhamala et al., 2003; Grahn & Brett, 2007, 2009; Penhune et al., 1998).

Compared to simple isochronous tasks, rhythmic tasks also, however, place demands on working memory to organise elements and maintain a representation of the rhythmic structure of phrases to facilitate prediction and production (Grahn & Brett, 2007; Konoike et al., 2012; Patel & Daniele, 2003). The inferior frontal gyrus and inferior frontal sulcus have been implicated in these memory functions during rhythm processing (Bengtsson et al., 2009; J. L. Chen et al., 2008; Grahn & Brett, 2007). Therefore, like some of the judgement tasks described earlier in Table 2.2 these tasks appear to give rise to additional processing elements beyond the basic timing network.

Analysis of musical performance has, however, been used as a measure of temporal processing with recognition of close affinity of music to movement, with the perception of a beat in music giving rise

to movement patterns synchronised to the tempo (e.g. foot taps or dance). Moreover, the importance of rhythmic cues or prosody within language has been recognised, with most languages comprising a recognisable rhythmic structure that supports the development of language in children (Cummins & Port, 1998; Patel & Daniele, 2003 and see editorial presented by Overy & Turner, 2009, and the associated special issue).

Tasks in which the detection and production of rhythm structures is beat-based (i.e. with a regular metrical structure) closely resemble the isochronous motor timing synchronisation tasks discussed above. Timed motor production is typically more temporally accurate for such beat-based rhythmic sequences where accents are regular than for irregular rhythms (where the beat is not metrical and accents are irregular) (Grahn & Brett, 2007; Trehub & Hannon, 2009). Performance on beat-based tasks is accompanied by an increase in activity in the basal ganglia and SMA (Grahn & Brett, 2007) and when these areas are affected by neurological conditions such as Parkinson's disease, this beat-based perception breaks down (Grahn & Brett, 2009). These neural correlates of beat perception appear to be active even in situations where behavioural responses to the rhythm are not required as well as for more complex rhythms providing they have a regular beat (Bengtsson et al., 2009; Grahn & Brett, 2007). Such evidence supports the existence of a beat-based timekeeping system which helps to encode the underlying temporal structure of stimuli with a regular meter (e.g. in music or speech). In summary, rhythmic tasks involving anisochronous temporal stimuli (e.g. music) assess processing in the same timing network as that measured by isochronous tasks. The additional processing requirements of rhythmic tasks tend to lead to recruitment of additional frontal brain circuits and irregular rhythms do not facilitate beat-based entrainment.

2.4.2.4. Stimulus modality

The perceptual modality through which temporal stimuli are presented has also been found to affect timing performance. In motor timing synchronisation tasks, stimuli can be delivered acoustically as tones or clicks; visually, via flashing LEDs or pictures on a screen; or through tactile stimuli such as periodically applying pressure to a fingertip on the opposite hand to that producing the synchronisation response. It has been established that the auditory system has higher temporal acuity than the visual system, enabling more accurate synchronisation of motor behaviour when pacing stimuli are presented acoustically (Grahn, 2012; Jäncke, Loose, et al., 2000; Jantzen, Steinberg, & Kelso, 2005; Kolers & Brewster, 1985; Loras, Sigmundsson, Talcott, Ohberg, & Stensdotter, 2012; Patel et al., 2005; Semjen & Ivry, 2001; Stauffer, Haldemann, Troche, & Rammsayer, 2012). Visual pacing stimuli give rise to greater variability in synchronisation responses than tactile stimuli with the least variability found with auditory pacing stimuli (variability operationalised as standard deviation of inter-response-intervals) (Grahn, 2012; Kolers & Brewster, 1985; Loras et al., 2012; Patel et al., 2005; Repp, 2003). These modality effects are independent of

participants ability to reproduce the inter-stimulus intervals in synchronisation paradigms in any one modality and are found even under bimanual tapping conditions (Semjen & Ivry, 2001).

The effect of stimulus modality is also apparent across other temporal processing tasks including duration discrimination (Beckmann, Grube, & Folta, 2010; Droit-Volet, Tourret, & Wearden, 2004; Penney, Gibbon, & Meck, 2000), judgement of rate (Fendrich & Corballis, 2001; McAuley & Henry, 2010; Recanzone, 2003) and rhythm reproduction tasks (Glenberg & Jona, 1991; Grahn, 2012). The modality of the stimulus also influences the response asynchrony or anticipation time (the time between the stimulus and response onsets) in synchronisation behaviour. As already described, asynchrony is typically negative for acoustically paced timing, with the tap occurring prior to the tone to achieve a perception of synchrony (Aschersleben, 2002). Visual pacing stimuli give rise to anticipation times that are significantly different in magnitude than auditory stimuli, with some studies finding smaller (or even positive) asynchronies for visual stimuli (Jäncke, Loose, et al., 2000; Kolers & Brewster, 1985; Pollok et al., 2009) and others finding larger asynchronies (Loras et al., 2012; Repp, 2003).

Differences in synchronisation behaviour across modalities may be accounted for by differences in the time taken to receive or process afferent sensory information from each sensory domain (Kolers & Brewster, 1985). The differences between the modalities have been measured using multi-modal stimuli in which both auditory and visual stimuli are presented together. In order to perceive multi-modal stimuli as being synchronous they must be presented close together in time, although if a visual stimulus is presented before an auditory stimulus then participants tolerate more asynchrony (112ms) between the stimuli before they judge them as being asynchronous, than when auditory stimuli precede the visual stimuli (65ms) (Lewkowicz, 1996). This phenomenon may occur because signals generated by the perception of visual stimuli take longer to arrive at central processing areas and have a more variable latency than auditory signals (Brebner & Welford, 1980; Ng & Chan, 2012) such that visual stimuli need to be presented prior to auditory stimuli in order for the two to be perceived as simultaneous (Arrighi, Alais, & Burr, 2006; Lewkowicz, 1996). An alternative proposal is that there are differences in the central timekeeping of different sensory signals, with the timekeeping system running faster in the auditory system than the visual timing system (Droit-Volet et al., 2004; Patel et al., 2005; Penney et al., 2000). If the timekeeper is thought of as a pacemaker that accumulates pulses until a count is reached and a response implemented, then under auditory conditions a greater number of pulses may accumulate over an interval period and give rise to longer perceived durations than for visual stimuli. Such timekeeping mechanisms are also influenced by attention which helps close the loop between the pacemaker and accumulation systems (Droit-Volet et al., 2004; Penney et al., 2000). The need for overt attention to be directed towards visual stimuli in order to perceive their temporal structure means that this switch may be less readily activated when viewing visual stimuli in comparison to listening to auditory stimuli.

The higher temporal precision of the auditory system is somewhat unsurprising given that music, with an inherent requirement for beat based processing, only exists in the auditory-motor domain (Patel et al., 2005; Repp, 2003). Equally, the need for high temporal precision in the auditory domain, for example for communication, may have driven the development of this acuity. With beat based processing being more ecologically valid in the auditory modality, performance may also be facilitated by the system's capacity for rhythmic processing allowing the chunking of beats (Glenberg & Jona, 1991; Grahn, 2012) and adaption to errors to occur (Kolers & Brewster, 1985; Repp, 2003). These mechanisms which give rise to accurate timing behaviour are not thought to available to the same extent in visual tasks. For example, when irregularities are presented in temporal sequences, they are not noticed or corrected to the same degree when presented visually as when presented acoustically (Kato & Konishi, 2006). One suggestion, to account for the reduced temporal acuity in the visual system, is that temporally presented visual stimuli have to be converted into an auditory representation (e.g. through echoic rehearsal of the stimuli) prior to processing their temporal nature (Glenberg & Jona, 1991; Grahn, 2012; Guttman, Gilroy, & Blake, 2005; Repp, 2003). However, such rehearsal may only occur when participants have been exposed to auditory stimuli prior to completing a visual synchronisation task (McAuley & Henry, 2010). Grahn (2012) attempted to improve visual synchronisation behaviour by incorporating motion and spatial cues into the pacing stimuli. She found that some beat based processing is possible in the visual modality if a rotating bar stimulus is used instead of a flashing light. Grahn suggested that the availability of spatial cues increased participants' ability to perceive temporal accents in the stimuli, although performance was still less accurate with the visual rotating bar than with auditory tones.

Visual and auditory tasks both activate the network, described above, that is typically associated with motor timing control, including the pre-motor cortex, somatosensory cortex, and the basal ganglia, thalamus and cerebellum (Jäncke, Loose, et al., 2000; Jantzen et al., 2005; Pollok et al., 2009; Schubotz et al., 2000). The improved temporal acuity with auditory stimuli is mirrored by stronger activations across this network in auditory compared to visually paced tasks (Jäncke, Loose, et al., 2000). In comparing the fMRI BOLD signal when completing visually and acoustically paced synchronisation and continuation tasks, Jäncke et al. found significantly greater activity in the SMA in acoustically paced tasks than with visual stimuli. This area is important for internally guided movements and contributes to beat-based guidance for timing (Jäncke, Loose, et al., 2000; Wiener et al., 2010). The comparative lack of activity in the SMA under visually guided conditions, together with stronger activations of occipital cortex and somatosensory areas such as M1 and S1 (Jäncke, Loose, et al., 2000; Pollok et al., 2009), suggest that under visual conditions, sensory stimuli are monitored to guide behaviour rather than relying on the operation of an internal timekeeping mechanism. A similar difference in activity between the two tasks was found in the cerebellum (Jäncke, Loose, et al., 2000). When presented as an auditory task, the right inferior cerebellum was

recruited. This area is connected to the secondary somatosensory areas and is important for collating information from temporal and parietal lobes in order to generate explicit motor behaviour. In comparison, for the visual task, the right superior cerebellum which connects to the primary sensory cortex, frontal and occipital lobes, was active. These areas are required for a more perceptually focused type of processing (Jäncke, Loose, et al., 2000).

Comparing across the synchronisation and continuation phases of the motor timing task, Jäncke et al. (2000) found differences across the two modality conditions. In the auditory condition the same brain areas were activated for both phases of the timing task. In comparison with visual pacing stimuli there was a change in the pattern of activity within the frontal-parietal timing network between the synchronisation and continuation phases. The activity in the dorsal pre-motor cortex in the visual synchronisation condition was in a right anterior area and changed to a more posterior and left lateralised focus when participants continued to tap their finger unpaced. The authors concluded that while auditory stimuli are processed by internal timekeeping systems across both synchronisation and continuation phases, sensory stimuli are relied upon under visual pacing conditions and additional internal guidance of behaviour is only recruited once the pacing stimuli are removed, guidance which was not in use during the visual synchronisation phase (Jäncke, Loose, et al., 2000).

Functional dissociations between auditory and visual tasks have been replicated in studies measuring the coherence of neuromagnetic brain activity between brain areas during task performance, using MEG. Pollok et al. (2009) found that the areas which showed functional interactions during auditory synchronisation tasks were different from those for visual tasks. The interpretation of these results again supported the idea that auditory timing is internally guided through prediction of the rhythmic beat-based sequences whereas visual timing is guided by monitoring the visual stimuli themselves and updating responses to match the stimuli.

The relative temporal superiority of the auditory system also means that auditory perception dominates over visual perceptions when simultaneous bimodal (auditory-visual) stimuli are presented during timing tasks. Under such conditions bimodal timing performance resembles that under auditory conditions alone (Repp & Penel, 2002). The visual component of the bimodal task is temporally captured by the perception of the auditory stimulus and the visual events are perceived as occurring at the same time as these auditory events, even if they do not occur entirely in synchrony (Elliott, Wing, & Welchman, 2010; Fendrich & Corballis, 2001; Recanzone, 2003; Roach, Heron, & Mcgraw, 2006; Wada, Kitagawa, & Noguchi, 2003). This temporal phenomenon is comparable to the spatial ventriloquism effect in which the comparatively high spatial acuity of the visual system means that auditory events occurring simultaneously, but originating elsewhere in space are captured by the visual perception and the two events are perceived as occurring in the

same location (Bertelson, Vroomen, Driver, & de Gelder, 2000; Recanzone, 2003; van Wassenhove, Buonomano, Shimojo, & Shams, 2008).

The auditory temporal advantage in bimodal timing tasks has been shown to be resistant even to large spatial disparity between the auditory and visual stimuli, and to reductions in the acoustic quality of the auditory stimulus (Kato & Konishi, 2006; Recanzone, 2003). To assess the source of this dominance tasks have been used in which uncertainty or jitter has been added to either the auditory or visual stimulus train. Under these conditions the stimulus which is perceived as more reliable is used for temporal processing (Heron, Whitaker, & Mcgraw, 2004; van Wassenhove et al., 2008; Wada et al., 2003), unless that stimulus is considerably temporally discrepant from the other stimulus (e.g. >50ms), in which case the two stimuli are perceived as temporally independent (Elliott et al., 2010). Typically the high temporal acuity of the auditory system means that auditory stimuli are more often relied upon to guide temporal responses. In light of such findings, it is proposed that bimodal stimuli are resolved by optimal weighting of the two sensory perceptions, with weights dependent on reliability, relevance and correspondence between stimuli. Where this weighting process gives rise to a simultaneous perception of the bimodal stimuli despite any temporal asynchrony between the two, adaptation to the asynchrony can occur, so that a coherent perception is maintained in future (Elliott et al., 2010; Roach et al., 2006; Roach, Heron, Whitaker, & Mcgraw, 2011). In summary, these behavioural effects and differences in neural activity under visual and auditory conditions indicate that temporal processing proceeds via different routes under the two stimulus modalities. The highest temporal acuity is shown in the auditory modality on standard motor timing tasks leading to auditory dominance when synchronising to bimodal visual-auditory stimuli.

2.5. Perspectives on timing

Many of the studies, described above, that have assessed the properties of timing behaviour have taken a neuropsychological stance towards the description of the causes of behaviour. This perspective derives from a cognitive perspective, describing distinct neural loci that are relevant to particular timing functions which work as a network to process temporal stimuli. Such models provide hypotheses about control mechanisms which adequately represent behavioural data (Wearden, 2003). This dedicated or modular approach to timing control contrasts with “intrinsic” models that view timing as the product of the oscillatory activity of the neuronal assemblies themselves (Buonomano et al., 2009; Ivry & Schlerf, 2008). Oscillatory activity in the brain results from the simultaneous firing of groups of neurons and is thought to allow communication across modules of the brain (Fries, 2005). This can be measured directly or indirectly through neuroimaging tools with high temporal resolution such as MEG and EEG. Intrinsic models propose that the oscillatory characteristics of neuronal populations, distributed across the brain, give rise to the

control mechanisms that enable temporal processing. Such models are supported by evidence demonstrating that the phase of neuronal oscillations, for example in the auditory cortex, are coupled with the temporal dynamics of environmental stimuli and change when different tempos are presented (Luo, Liu, & Poeppel, 2010; Nozaradan et al., 2011; Tecchio, Salustri, Thaut, Pasqualetti, & Rossini, 2000). This means that neural activity can code a variety of temporal properties of stimuli and allow for temporal prediction of future events (Arnal, 2012; Fujioka, Trainor, Large, & Ross, 2012).

There are differences between these two perspectives of timing control and the intrinsic and dedicated models are yet to be resolved into a single account of temporal processing. Intrinsic processing is typically modelled computationally, for example through the State Dependent Network model (Karmarkar & Buonomano, 2007) in which temporal relationships are represented through spatial patterns of neural activity. Such models, however, currently fail to represent timing behaviour under task conditions that place constraints on timing, such as the differences in timing with short (sub-second) versus long temporal intervals (Spencer, Karmarkar, & Ivry, 2009). Nevertheless, there is recognition that *both* provide useful accounts of particular strands of evidence (Ivry & Schlerf, 2008). Both perspectives are able to account for behavioural performance in more complex timing tasks that require attention or memory by recruiting additional modules (dedicated) or supplementary neuronal activity (intrinsic) that are independent of the mechanisms necessary for timing control (Spencer et al., 2009).

Some of the most influential cognitive models of timing control are aligned with the modular approach. The Scalar Expectancy Theory (Church, 1984; Gibbon, 1977) has been the basis for many models of temporal behaviour. It proposes that processing in time stems from a clock-like mechanism, which generates pulses at regular intervals, and an accumulator in which pulses are stored until the end of the interval is reached. The length of the interval is then determined by the count of the stored pulses such that longer intervals are coded by a larger number of pulses. The intervals can be stored in memory for later reference or decision making. In such models cognitive factors (e.g. arousal and attention) are proposed to affect timing by influencing the ongoing generation of pulses or by preventing the activation of a switch which starts and stops the accumulation process, particularly for longer stimulus intervals (Macar & Vidal, 2009). Other clock based models have been described by Wing and Kristofferson (1973a) and Wearden (1991, 2003). Such models are not incompatible with the intrinsic approach to timing because the pacemaker and accumulation components can be represented as distributed networks across the brain and because accumulation could occur via spatio-temporal summation across populations of neurons (Buonomano & Laje, 2010; Ivry, 1996; Macar & Vidal, 2009).

2.6. Analysing timing performance

Working from a dedicated cognitive approach to timing behaviour, many studies have used motor timing performance on synchronise-and-continue tasks to explore the behavioural characteristics of timing and to model the mechanisms of control that underlie behaviour. These methodologies have helped to establish the characteristics of typical timing behaviour and the models derived from such data are useful for understanding the cause of atypical timing performance in clinical populations.

Early descriptions of motor timing behaviour highlighted the presence of characteristic patterns within response intervals when a finger was tapped in time with isochronous stimuli. Stevens (1886) found that the variability in the inter-response intervals (IRIs) recorded showed meandering patterns which were not random and contained elements that increased linearly with the inter-stimulus interval (ISI) size. In his experimental study Stevens found that IRIs rarely deviated more than 5% from the target interval and that any abnormally long intervals were followed by deviations in the opposite direction (i.e. shorter intervals) in order to return the mean IRI to the target rate. Because these aberrant intervals were corrected after they had occurred, Stevens proposed that the errors came from the execution of the response rather than from the interval standard "carried in mind" and so these observations provided some insight into the properties of the system controlling timed responses (Stevens, 1886, p. 401). Since then similar observations have led to the development of models of timing to account for the mathematical properties of these fluctuations in motor timing behaviour.

The models of timing behaviour fall into two categories, with distinctions drawn between linear and dynamic models. These two categories reflect the intrinsic and dedicated perspectives on timing described above (Section 2.5). Linear models often take the form of a stochastic equation, using a probability distribution based on past events in order to predict the values of future events. Dynamic systems models assume that temporal regularities in behaviour emerge from the properties of the biological systems involved in temporal behaviour production. A brief overview of these perspectives is provided below; a number of reviews of these models are provided elsewhere (Grondin, 2010; Heuer, 1996; Ivry & Richardson, 2002; Ivry, 1996; Krampe, Engbert, & Kliegl, 2002; Macar & Vidal, 2009; Mauk & Buonomano, 2004; Schöner, 2002; Vorberg & Wing, 1996).

Models approaching timing behaviour from a dynamic systems perspective assume that timing is derived from the properties of neurons or properties of the motor system (Buonomano & Laje, 2010; Miall, 1989; Pressing & Jolley-Rogers, 1997; Schöner, 2002). When many thousands of synchronisation responses are recorded, distinct long term trends have been found which resemble 1/ƒ noise (i.e. low frequency dependencies), as are found in many other biological systems (Madison, 2001b; Wagenmakers, Farrell, & Ratcliff, 2004; Wing, 1980). By analysing the emergent properties of these trends over long time series, equations can be derived which model the state of the system.

These equations code for the properties of a large number of biological components of the system that are hypothesised to be relevant and the models allow prediction of the typical properties of timing behaviour, often in greater detail than is provided by linear models (Buonomano & Laje, 2010; Wing & Beek, 2002).

Linear models of timing suggest that an internal timing mechanism generates pulses to represent time intervals and these can be used to guide behaviour, for example through implementation in the motor system (Church, 1984; Hinton & Meck, 1997; Wing & Kristofferson, 1973a). Most models in this class conceive the timekeeping mechanism as either a clock-counter (event-based) or an hourglass timer (interval-based). The former assume that intervals are timed by waiting until a given count of internal (e.g. neural) events ("pulses") has been achieved before a response is implemented (Wing & Kristofferson, 1973a). This accounts for the observation that variability in performance on finger tapping tasks increases as the length of ISIs increase; there is greater opportunity for random variability to be introduced over extended wait times (Wing & Kristofferson, 1973a). The alternative interval timer approach suggests intervals of different durations are timed by distributed timers each with different periods. These timers are often likened to a series of hourglasses, each timing intervals of different lengths (Hinton & Meck, 1997; Ivry, 1996; Schöner, 2002). This idea is supported because it (a) allows a large number of interval durations to be represented and (b) accounts for evidence of timing being accurate over a limited range of interval durations (Ivry, 1996).

Ivry (1996) reviewed the evidence that suggests that different neural loops are responsible for the hourglass function. In comparison, other reviews suggest that there is currently no compelling evidence at present to accept or falsify either the event- or interval-based hypotheses and they both provide useful approximations of the same neural processes and therefore may not be wholly incompatible (Madison, 2001b; Schöner, 2002; Wing & Beek, 2002). For example, in order for stochastic models to account for more complex behaviours such as bimanual tapping or synchronisation to poly-rhythms additional terms can be included to allow for a form of coupling between multiple timers, akin to the multiple components of dynamic models (Schöner, 2002). Data from dynamic systems models have also been shown to reflect the variance structure predicted by stochastic models (Pressing, 1999; Wing & Beek, 2002). Thus the two perspectives likely provide solutions which are able to model behavioural characteristics at different scales of analysis (Wing & Beek, 2002) or for different types of task (Delignières, Lemoine, & Torre, 2004; Zelaznik et al., 2002). For example, dynamic timing models are found to be more appropriate for the description of the complexities of continuous oscillatory movements (e.g. circle drawing) whereas stochastic clock models are better suited to the analysis of discrete movement tasks like finger tapping (Delignières et al., 2004).

In order to apply dynamic systems modelling considerable quantities of behavioural data must be gathered (e.g. thousands of finger taps). In contrast, stochastic models can be applied to short time series of 30 or more responses and also allow identification of component processes that contribute to behaviour. For these reasons, the linear models have been more extensively applied in studies of typical timing behaviour as well as in research assessing timing data from populations who show timing deficits. As with any reductionist model of a biological system, linear models need to be applied in the context of their limitations for predicting all forms of a complex evolved behaviour. Despite this it is thought that they provide useful approximations of behaviour that help to quantify elements of timing behaviour (Beek, Peper, & Daffertshofer, 2000; Madison, 2001b).

2.6.1. The Wing-Kristofferson model of time series analysis

One of the most influential linear models of timing behaviour that has been frequently applied to motor timing data was described by Wing and Kristofferson (1973a, 1973b). In the Wing-Kristofferson model the timing system is represented as a stochastic process comprising of independent random variables, including a clock-like timekeeper mechanism that generates regularly timed outputs and an implementation (motor) system which enables timed physical responses to be output. The variability recorded in timed movements is therefore thought of as a combination of the variability in these two systems. Wing and Kristofferson (1973a, 1973b) demonstrated that when isochronous timed responses are recorded, the statistical dependencies within time series allow the two components to be separated, such that the variance attributable to each system can be measured directly from observed behaviour. A full derivation of the statistical parameters of the model is provided in Chapter 5 of this thesis.

Wing and Kristofferson (1973a, 1973b) collected synchronisation finger tap responses from a group of adults and demonstrated that performance over a number of trials matched the predictions of the model. Dissociations between the timekeeper and implementation components were found when performance was measured across response rates (up to 1 second intervals). As response rates increased, the timekeeper variance gradually decreased whereas the implementation variance remained roughly constant (Wing & Kristofferson, 1973a). These findings were taken as evidence for the existence of a clock counter mechanism in which the number of clock generated pulses determines the interval between responses. When fewer pulses are required for shorter intervals at faster tapping rates, timekeeper variance was reduced because of the decreased opportunity for variability to arise in the count of pulses. Implementation variance, in comparison, did not systematically vary with implementation rate, due to the random and independent nature of the delays generated in the motor system (Wing & Kristofferson, 1973a). Further support for the implementation variable as representative of peripheral motor systems comes from studies which find that this variance component (but not timekeeper variance) is affected by the type movement

(hand or arm; left or right limb) and by the loss of sensory sensations through nerve damage in the arm (Ivry & Keele, 1989; Sergent, Hellige, & Cherry, 1993; Wing, 1977a). Congenital hypothyroidism in children has also been found to lead to increased implementation variance compared to controls with no concomitant increase in timekeeper variance (Kooistra, Snijders, Schellekens, Kalverboer, & Geuze, 1997). This disorder affects the development of efficient communication across the central nervous system, leading to particular impairments in motor control. The implementation component of the Wing-Kristofferson model is therefore taken as a representation of the generation of movements in the motor system, downstream of the timekeeping mechanism (Ivry & Keele, 1989; Sergent et al., 1993).

The development of a method to decompose aspects of timing control has also allowed the efficacy of these two systems to be assessed in patient groups where motor timing deficits present as part of the pathology of the disorder or disease. Such models allow analyses to expand understanding of the components of timing behaviour beyond simple analysis of the deviation of response intervals from the mean response rate (i.e. standard deviation of inter-response intervals). Studies which have used the Wing-Kristofferson method (see Table 2.3) support the validity of the two components as being representative of different parts of the timing system.

Diseases affecting the basal ganglia, an integral part of the timing system, appear to have pervasive effects on both the timekeeper and implementation components estimated from the Wing-Kristofferson approach (Freeman et al., 1996; Harrington et al., 1998; O'Boyle et al., 1996). Parkinson's disease and Huntington's disease both affect the neurons of the striate cortex and are associated with differences in both components of the timing system as well as with a faster rate of finger tapping (smaller IRIs). When patients have received pharmaceutical treatments to improve the dopaminergic functions in the striatum, implementation variance, timekeeper variance and mean IRI were found to be normalised compared to pre-treatment levels (Harrington et al., 1998; O'Boyle et al., 1996; Pastor, Jahanshahi, Artieda, & Obeso, 1992). The evidence for damage to the basal ganglia leading to increases in the timekeeper component as well as in the rate of responses (IRI) suggest that the basal ganglia is important in forming a representation of time as described in Section 2.3. The reasons for the changes in implementation variance are less well understood. It has been proposed that the increases may result the presence of difficulties in the production of movements with appropriate force (Pastor et al., 1992) or the downstream effects of the diseases on the indirect and direct connections between the striate cortex and the motor cortex in Huntington's and Parkinson's patients (Freeman et al., 1996).

Participants with lesions to the cerebellum show increased timekeeper variance without any corresponding increase in implementation variance (Harrington et al., 2004; Ivry et al., 1988). The participants studied by Ivry et al. and Harrington et al. varied in the degree of damage to the

cerebellum (extensive cerebellar atrophy compared to more specific damage to medial and lateral areas of the middle-to-superior cerebellar lobules), but with similar effects. In comparison to the studies with patients with diseases affecting the striatum, these elevated levels of timekeeper variance in the cerebellar patients were found in the absence of any differences in mean IRI. This indicates that the cerebellum does not influence the rate of the clocking mechanism but acts as a downstream regulator of the timekeeping signals. Therefore the timekeeper component calculated in the Wing-Kristofferson model must incorporate elements of timing control beyond the pulse generator. Indeed, timekeeper variance also increases when participants are required to complete a distracter task alongside the motor timing paradigm (Sergent et al., 1993). By examining this component alongside the typical measures of performance (e.g. mean and deviation of IRIs) the efficacy of the different elements of the timing system can be measured in patient populations.

Table 2.3: Examples of use of the Wing-Kristofferson model to examine timing characteristics in patient groups.

Area of timing network implicated	Cause of Impairment	Effect on Mean IRI	Component showing increased variance	Authors
Cerebellum	Lesions / atrophy	No change	TK	Harrington et al. (2004); Ivry & Keele (1989)
Wide range of cortical areas especially motor	Congenital Hypothyroidism	No change	IMP	Kooistra et al. (1997)
Basal Ganglia	Parkinson's Disease, Huntington's Disease	Increased IRI	TK and IMP	Freeman et al. (1996); Harrington et al. (1998); O'Boyle et al. (1996)
Basal Ganglia / Cerebellum	ADHD	Adults: no difference Children: trend for Increased IRI	Adults: TK Children: TK & IMP	Valera et al. (2010); Zelaznik et al. (2012)
Basal Ganglia / Cerebellum	Bipolar Disorder	Increased IRI	TK	Bolbecker et al. (2011)
Cortical-striatal-cerebellar circuits	Schizophrenia	Increased IRI	TK	Carroll, Donnell, Shekhar, & Hetrick (2009)
Right Auditory Association Cortex, MTG, STG, IPL	Stroke damage	Not reported	TK	Wilson, Pressing, & Wales (2002)
Assorted regions of central and peripheral nervous systems	Foetal Alcohol syndrome	No Change	TK and IMP	Simmons, Levy, Riley, Madra, & Mattson, 2009

TK – Timekeeper variance, IMP – Implementation variance, IRI – Inter-response interval

Studies of participants with Attention Deficit Hyperactivity Disorder (ADHD) and bipolar disorder also reinforce the different roles of the cerebellum and basal ganglia in timing behaviour (Bolbecker et al., 2011; Valera et al., 2010; Zelaznik et al., 2012). These disorders were investigated because the neuropathology of these disorders involves a degree of impairment in the basal ganglia and/or cerebellum (although other brain areas are also affected). The profile of increased timekeeper variance together with smaller IRIs in adults with bipolar disorder (Bolbecker et al., 2011) and children with ADHD (Zelaznik et al., 2012) indicate a similar presentation to Parkinson's patients, with an impaired clock mechanism which affects the rate of IRIs. In contrast, Valera et al. (2010) found that adults with ADHD had higher timekeeper variance than controls, but showed no difference in the rate of tapping. The absence of a difference in IRIs in adults but not in children may be accounted for by the delayed development of the basal ganglia in adolescents with ADHD which is remediated by adulthood (Castellanos & Tannock, 2002). Comparing these results with those from previous patient studies, it would appear that the children with ADHD show a profile like the patients with striatal impairments, whereas adults with the disorder show a profile like the cerebellar patients, once the striate cortex has matured.

These studies demonstrate the insight that can be gained in applying a time-series analysis model to timing data from particular populations, not only to establish the origin of any group differences (in terms of timekeeper or implementation variance) but also to allow comparative analysis with other populations who have known areas of brain dysfunction. The original model described by Wing and Kristofferson (1973a, 1973b) has been most widely applied in timing research, although extensions to the model have been proposed. For example, Buhusi and Meck included additional components to account for the roles of attention and memory in accumulating timekeeper pulses for longer duration tasks (Buhusi & Meck, 2009b). Similarly, the model has been extended to take account of feedback mechanisms which account for performance under different stimulus modalities or under bimanual responding (Drewing & Aschersleben, 2003; Elliott et al., 2010; Semjen, Schulze, & Vorberg, 2000). Such models have not yet been widely applied to timing data from patient populations.

2.7. Chapter summary

Motor timing tasks such as finger tapping provide a measure of the limits of the timekeeping system and have a close affinity to the mechanisms of timing control in the motor system. These motor timing tasks operationalise the same timing mechanisms assessed by perceptual timing tasks, but the absence of a requirement for overt decision making, attention or memory makes motor timing paradigms ideal for studying temporal processing capacities in both normal and atypical populations of interest. Such tasks have allowed investigation of the conditions under which temporal performance changes, such as with stimulus modality, interval size and response type. The behavioural response generated in motor timing tasks gives rise to a well controlled response

pattern with characteristic elements of variability. These patterns have been modelled and give clues to the organisational principles of the timing system allowing comparisons of performance across populations with apparent difficulties in timing. The Wing-Kristofferson model in particular has been used to demonstrate that timekeeper and implementation processes can be dissociated through statistical analyses of time series data. Such analyses are useful in the study of timing in populations where variability in timing has not been previously investigated beyond simple analysis of deviation around the mean. The model is attractive in that the components can be estimated from the relatively small samples of data which are readily gathered from child or clinical populations.

3. Motor timing in dyslexia and related disorders

3.1. Chapter overview

As described in Chapter 2, motor timing tasks provide a measure of the functioning of components of the timekeeping system and have been applied to a variety of clinical populations. Such tasks have also been applied to populations of children and a deficit in motor timing has been found to be associated with the presence of dyslexia. The difficulties experienced by children and adults with dyslexia in producing timed motor outputs are reviewed presently. Secondly, this chapter highlights the similar motor timing difficulties shown by children with ADHD. In conjunction with the previous chapter this discussion leads to the development of the research questions addressed in the thesis.

3.2. Motor timing and dyslexia

The ability of motor timing tasks to reveal underlying features of timing control (as described in Chapter 2) has prompted these tasks to be variously applied to populations of children and adults with dyslexia. These studies are summarised in Table 3.

Peter Wolff and his colleagues were instrumental in applying motor timing tasks to investigate how children with dyslexia process temporal stimuli (Badian & Wolff, 1977; Klipcera et al., 1981; Wolff et al., 1984, 1995; Wolff, Michel, Ovrut, & Drake, 1990; Wolff, 2002). This series of studies was motivated by findings that boys with learning difficulties had poorer performance than typically developing boys on observer rated measures of rhythmic motor performance (Hurwitz, Bibace, Wolff, & Rowbotham, 1972). To extend these findings Wolff et al. employed a range of motor timing tasks providing objective measurements of timing behaviour which typically required participants to tap their finger(s) in time to an auditory metronome or beat with regular intervals (isochronous synchronisation; as described in Chapter 2). In some experiments the pacing beat was subsequently removed and participants continued tapping at the same speed in the absence of the pacing stimuli (a continuation task). The mean inter-response interval (IRIs) produced and the standard deviation of IRIs were recorded to determine the precision and variability of temporal reproduction in the participant groups of interest.

Using this type of paradigm Badian and Wolff (1977) assessed a control group of typically developing children (n=392) and a group of 28 boys with reading ages below that predicted by their chronological age and IQ score. A unimanual (one-handed) tapping task with stimulus inter-onset intervals (IOIs) of 500ms (2Hz) was used alongside a bimanual alternation task at 833 and 500ms (1.2 and 2Hz). The alternation task required participants tap alternately with each hand on subsequent beats (i.e. Left-Right-Left-Right). On this task, the discrepant readers showed greater performance variability (standard deviation of IRIs) than the control group, and especially so for left-hand

responses. On the unimanual task the boys with reading difficulties showed less variability than controls, although significance values were not reported for this comparison. Badian and Wolff concluded that the motor timing difficulty in the poor readers was limited to bimanual performance. The performance data on the bimanual trials were, however, collapsed across the two response speeds and across the synchronisation and continuation phases of the tapping task so it is unclear whether the poor readers timing were equally impaired in all elements of the task. In a follow-up study, Klipcera et al. (1981) introduced an additional bimanual condition which demanded responses with the left and right fingers in synchrony (bimanual unison tapping) and performance was assessed only during the continuation phase of the different task. The children with reading difficulties showed greater performance variability than controls on the bimanual alternation task, but not on the unimanual or bimanual unison tasks.

This evidence suggested that poor readers have a timing difficulty limited to bimanual tasks where left-right responses need to be coordinated to occur alternately. Subsequent studies have challenged this, finding impairments in timing across both unimanual and bimanual tasks in children with reading difficulties (Wolff et al., 1984; Wolff, Michel, Ovrut, et al., 1990). Wolff et al. (1984) found that children who had IQ-discrepant reading scores had greater variability and reduced accuracy in matching the target intervals during the continuation phase of tapping tasks irrespective of whether they were tapping in synchrony to a beat under unimanual or bimanual unison conditions (at 650ms; 1.53Hz) or under bimanual conditions (at 333ms (3Hz), unison or alternating). Similarly, Wolff et al. (1990) demonstrated that adults and adolescents with reading scores below the predicted level had more variable performance across both unimanual and bimanual tasks (with IOIs of 650, 500 or 333ms; 1.53, 2 or 3Hz) than typically developing children or children with learning difficulties (i.e. low IQ). These two studies also implemented a complex asynchronous tapping task in which participants tapped one hand at the same time as the pacing stimulus and the other hand twice as fast as the stimulus rate such that this tap segmented the stimulus interval. The poor readers again showed more variability and made more errors on this task than the controls groups at IOIs of 500 and 333ms (2 and 3Hz) (Wolff et al., 1984; Wolff, Michel, Ovrut, et al., 1990). Among the poor readers, Wolff et al. (1984) found that synchronous and asynchronous bimanual performance, but not unimanual performance, was correlated with measures of literacy, such as reading achievement, spelling and rapid naming and a measure of the ability to produce rapid sequences of spoken syllables. Wolff et al. (1990) did not examine such associations in the study conducted with adults and adolescents. The authors interpreted these results as supporting a primary difficulty in bimanual coordination linked to reading ability despite the presence of the deficits on the unimanual tasks, particularly at the faster tapping rates.

Table 3.1: Studies investigating motor timing performance and dyslexia

Authors	Paradigm[A]	Synchronisation Phase	Continuation Phase	Stimulus Rate (ms)	Response Type[B]	Mean Age Years (SD)	Type of Reading Difficulty Groups	Control Participants	ADHD[C]	Group Difference	Description
Badian & Wolff (1977)	S&C (data collapsed across both conditions)	15 s	30 s	500	Unimanual	12 (1.9)	Reading age discrepancy, n=28	School children n=392, aged 5-13	Not controlled	*	Controls had larger IRI SD (significance unknown)
				500	Bimanual Alternation					*	RD Group larger IRI SD
				833	Bimanual Alternation						
Klipcera, Wolff & Drake (1981)	S&C (data from C phase analysed only)	15 s	45 s	650	Unimanual	13 (±0.8)	>1.5 year delayed in reading, n=30, boys	Boys n=15, aged 13(±0.8)	Not controlled		
					Bimanual Alternation					*	Greater variability and larger deviation from target ISI rate
					Bimanual Unison						
Wolff, Cohen & Drake (1984)	S&C (data from C phase analysed only)	30s	30s	650	Unimanual	12 (1.1)	Average of 2.7 years below grade level in reading, n=20, boys	Children matched for social class and intelligence, n=15	Included 3 informally identified boys with hyperactivity symptoms	*	Greater variability & deviation from target ISI rate in RD group
				650	Bimanual Unison						
				333	Bimanual Unison					*	Greater deviation from target ISI & associated with measures of literacy
	S only			333	Bimanual Asynchronous Alternation (2:1)					*	Greater variability in RD group especially when left hand was leading
Wolff, Michel, Ovrut & Drake (1990)	S&C (data from C phase analysed only)	20s	60 taps	650, 500, 333	Bimanual Unison	Adolescents aged 13-18 and adult men aged 18-21	Adolescents: >2.5 grades below expected reading level and normal IQ, n=50 Adults: reading below 9th grade, n=41	Adolescents: learning difficulty group (n=50) & typically developing group (n = n.d.) Adults: typically developed group (n=41)	Not controlled	*	Increased variability at 500 & 333ms intervals in adolescents with RD than both control groups
					Bimanual Alternation					*	Increased variability for adolescents with RD
					Bimanual Asynchronous					*	Increased variability overall and more errors at 500 and 333ms interval rates for both adolescents and adults with RD than controls

Authors	Paradigm[A]	Synchronisation Phase	Continuation Phase	Stimulus Rate (ms)	Response Type[B]	Mean Age Years (SD)	Type of Reading Difficulty Groups	Control Participants	ADHD[C]	Group Difference	Description
Waber et al. (2000)[D]	S&C (C data analysed only)	10s	20s	666	Unimanual	9.6 (1.6)	Learning Impaired (but IQ>80), n=100	243 non-learning impaired, aged 9.4 years (SD 1.2)	Assessed behavioural disturbances (including attention) using parent ratings		Learning Impaired had greater variability but not significant once IQ controlled. Motor timing performance predicted variance in reading
				333 & 250	Bimanual Alternation					*	Learning Impaired had greater variability
				500, 333, 250	Bimanual Asynchronous					*	Learning Impaired had greater variability
Wolff (2002)	S only	n.d.	n.d.	500, 670	Unimanual and Bimanual Unison	13.6 (1.8)	Students with dyslexia (n=12) with normal or above average intelligence	12 school children (age & gender matched)	Controlled for other learning difficulties	*	Impaired readers had longer anticipation times
				400-500-670	Keep in time to beat as rate changes					*	Impaired readers took longer to match the new stimulus rate
Thomson & Goswami (2008)	S&C	20s	20s	666	Unimanual	10.6 (1.3)	Clinical diagnosis of Specific Reading Difficulties (n=25)	Typically developing school children, IQ>80 (n=23)	Only diagnoses of specific reading difficulties included		
				500						*	SRD group greater variability and different mean asynchrony in synchronisation phase only
				400						*	SRD group greater variability and different mean asynchrony in synchronisation phase & increased variability in continuation phase
Thomson, Fryer, Maltby & Goswami (2006)	S&C	20s	20s	400	Unimanual	22.3 (3.3)	Adults with clinical diagnosis of dyslexia (n=19)	Controls with no history of LD or other disorder aged 22.3(2.9) (n=20)	Controlled for additional learning disabilities		
				500						*	Adults with dyslexia had increased variability in synchronisation & continuation phases
				666						*	Adults with dyslexia had increased variability synchronisation phase

*In all experiments listed, stimuli were presented acoustically as tones or beeps. [A]Motor Timing Paradigm: S-Synchronisation C-Continuation. [B]Response Type: all studies measured finger tap responses either one- (unimanual) or two-handed (bimanual). [C]Whether the presence of ADHD symptoms was controlled for. [D]Did not specifically sample for reading difficulties, rather children with learning impairments. n.d. - not described. *Significant difference between groups.*

The evidence presented by Wolff et al. (1990) of greater timing variability in the group with dyslexia compared to control groups with and without learning difficulties suggests that the temporal deficit may be limited to cases with specific literacy difficulties rather than more general learning problems. However, a relationship between timing performance and more general ability has been highlighted by Madison, Forsman, Blom, Karabanov and Ullén (2009) who assessed timing over a range of stimulus intervals and found performance variability was associated with intelligence when tapping at intervals of 400-900ms (2.5-1.1Hz). Similarly, Holm, Ullén, Madison (2011) found a single timing factor was related to intelligence in a group of young adults. Such associations may be accounted for by the fact that timing performance follows a similar age-related trend as reasoning ability, with low scores in childhood and old age and higher scores in adolescence and adulthood (McAuley, Jones, Holub, Johnston, & Miller, 2006).

It is difficult to establish the specific contribution of general ability to the results described above because the studies did not assess other cognitive skills. A follow-up study by Waber, Wolff and colleagues (2000) attempted to address this by examining motor timing in children aged 7-11 with general learning difficulties not specifically in reading, who were failing to meet the demands of schooling (n = 100), and a control sample (n = 243). Unimanual and bimanual tasks were again used to assess timing across a range of IOIs from 666ms (1.5Hz) on the unimanual task to 250ms (4Hz) on the bimanual tasks. Variability was operationalised using the coefficient of variation (CV) measure which eliminates the variability attributable to the tapping rate (calculated as standard deviation of IRIs divided by mean IRI). The group with learning impairments showed greater variability on all the tasks, although group differences on the unimanual task were not significant once IQ was included as a covariate. Learning impairment group membership also accounted for variance in timing ability after controlling for reading ability, IQ and gender. This indicates that some variance in timing performance was related to more general learning ability. Across the entire sample, uni- or bimanual motor timing performance predicted reading, spelling and numerical operations abilities and these relationships were not accounted for by variance attributable to non-verbal reasoning. Together these results support the association between literacy and motor timing suggested elsewhere (Thomson & Goswami, 2008; Wolff et al., 1984) as well as a relationship between motor timing and more general ability factors.

The greater variability shown by children and adults with reading difficulties on unimanual tasks has been replicated more recently in studies assessing motor timing performance alongside literacy skills and other measures of temporal processing (Thomson et al., 2006; Thomson & Goswami, 2008). The additional measures (e.g. amplitude envelope detection) are discussed in detail later in this chapter where the link between motor timing and language development is described (See 3.2.1.2.b). Adults with dyslexia were found to have significantly greater timing variability during synchronisation tasks with 500 or 666 ms IOIs (2 or 1.5Hz) and during the unpaced continuation phase with 500ms IOIs

(2Hz) (Thomson et al., 2006). Children with dyslexia likewise had greater response variability than controls for the synchronisation task with 400 and 500ms IOIs (2.5 and 2 Hz) and for unpaced motor timing with 400ms IOIs (Thomson & Goswami, 2008). Performance on these tasks was again found to be predictive of literacy ability (with better performance associated with higher literacy scores), although after controlling for factors such as age and IQ, only the children (but not the adults) showed associations between measures of reading or spelling and motor timing performance. The strongest relationships were found between a composite measure of paced motor timing variability (collapsing across response rates) and written language measures such as spelling (Thomson et al., 2006; Thomson & Goswami, 2008). The authors suggested that the difference in the associations found for adults and children likely represents a change in motor timing ability over development such that performance decrements in motor timing are more strongly associated with literacy components in childhood. The adults were still unable to produce well controlled timing outputs but this did not correlate with written language measures. Nevertheless, these results confirm that timing difficulties associated with dyslexia are not limited to bimanual tasks, but are also found for single-handed tapping either when following a beat or maintaining an interval rate in the absence of a stimulus. The strength of the correlations between literacy measures and the paced timing measures suggests that the synchronisation difficulties present the most difficulties for these children. By controlling for general reasoning abilities, Thomson and colleagues were also able to confirm that the difficulties associated with literacy are not accounted for by general ability alone.

Additional support for the importance of motor timing as a risk factor in dyslexia comes from research examining the extent to which motor timing deficits are shared amongst families with a history of reading difficulties (Wolff et al., 1996, 1995). The behavioural data from these family studies demonstrated that proband children, selected on the basis of below expected reading and spelling abilities, had higher variability and lower accuracy on motor timing tasks (with ISIs of 200-500 ms; 5-2Hz) (Wolff et al., 1996). In addition, the children with dyslexia with motor timing difficulties (about half of the sample with dyslexia) also tended to have relatives with the same timing difficulties, whereas the relatives of probands who did not show timing difficulties had normal motor timing performance. The children with dyslexia who had relatives with dyslexia and motor impairments also made more phonologically unacceptable errors on a spelling task (Wolff et al., 1996). It was therefore proposed that temporal resolution and dysphonetic spelling deficits form part of the same phenotype, at least in half of the cases of familial dyslexia (Wolff et al., 1996, 1995).

In summary, these studies point towards motor timing performance being a behavioural marker for literacy difficulties. Children show difficulties on both bimanual and unimanual finger tapping tasks across a range of tapping rates from 650 to 200 ms IOIs (1.53-5Hz). The associations found between performance on these tasks and literacy skills are present across both children with reading difficulties and control groups with these skills representing part of a single phenotype. The reasons

why there may be a connection between motor timing and literacy abilities are not necessarily obvious given that motor timing and literacy tasks are somewhat different in form. A number of potential explanations have, however, been proposed.

3.2.1. The link between motor timing and dyslexia

If motor timing performance is associated with reading difficulties, it is likely that a component of timing behaviour relates to a cognitive or neural risk factor that contributes to the development of reading ability. For instance, the previous chapter described how timing performance can act as a marker of the functioning of particular areas of the brain (Chapter 2). In the dyslexia field, a number of suggestions have been proposed to account for the co-occurrence of motor timing difficulties and dyslexia. These are motivated by consideration of either the types of motor timing impairments present in dyslexia, the components of motor timing control or by considering the relationship between timing and language development. A short overview of these theories is presented here to demonstrate why it is useful to investigate motor timing in dyslexia, as motor timing may provide a metric of functioning in processes that contribute to learning to read.

3.2.1.1. Inter-hemispheric communication

Some of studies of motor timing described above indicated that children with reading scores discrepant from their general ability scores typically have greater performance variability on bimanual tapping tasks, especially when tapping the left and right hands in an asynchronous 2:1 ratio (Badian & Wolff, 1977; Klipcera et al., 1981; Wolff et al., 1984). The authors hypothesised that this was due to a delay or disruption in the neural communication between the two hemispheres of the brain in these children which lead to a failure of coordination between the responses of the two hands (Badian & Wolff, 1977; Klipcera et al., 1981; Wolff, Michel, Ovrut, et al., 1990).

Two-handed timing tasks do involve greater inter-hemispheric communication than single-handed tasks due to the exchange of sensory feedback from the two hands under such conditions. This enables performance to become more stable in these tasks than under unimanual conditions (Drewing & Aschersleben, 2003; Helmuth & Ivry, 1996). However, it is likely that such bimanual coordination occurs subcortically because severe disruption of the corpus callosum, which provides the main connections between the two hemispheres, does not affect the stability of bimanual tapping (Ivry & Hazeltine, 1999). Despite the requirement for additional coordination of sensory information, bimanual motor timing tasks recruit similar left and right localised neural networks to those recruited by unimanual tasks, and the coordination of these networks gives rise to the same stabilities in performance across both unimanual and bimanual tasks (Banerjee et al., 2012; Semjen & Ivry, 2001). Such evidence means it is difficult to attribute the impairment shown by participants with dyslexia in bimanual performance to an inter-hemispheric communication problem. Indeed, a degree of inter-hemispheric communication occurs even on unimanual tasks, such as between the

secondary somatosensory cortices (Jantzen et al., 2004). Therefore poor performance on both bimanual and unimanual tasks could be indicative of either an inefficiency in inter-hemispheric communication or a problem in the networks controlling unimanual timing which contribute to both tasks.

Robust group differences were also found in Wolff's studies for the asynchronous alternating bimanual paradigm where participants tapped left and right in a 2:1 ratio (Wolff et al., 1984; Wolff, Michel, Ovrut, et al., 1990). This type of task involves additional planning, memory and attention and when compared to simple isochronous rhythms, resulting in additional bilateral neural activity across the timing network (as described in Chapter 2; Jantzen, Steinberg, & Kelso, 2004). Difficulties with memory and attention are core symptoms of dyslexia and so group differences on this task might be expected for the asynchronous task irrespective of a timing difficulty. The increased timing variability found on unimanual as well as the bimanual tasks (Wolff et al., 1984) may be indicative of the same specific timing difficulty, but insufficient research has been conducted to compare between the tasks to allow the relative importance of the bimanual deficits to be determined.

3.2.1.2. Motor timing and language development

An alternative perspective regarding the relationship between motor timing and reading difficulties is that motor timing performance reflects the ability to synchronise to auditory sensory events; an ability that is important for language functions. This perspective must be considered in light of the extended route to reading (described in Chapter 1) through which speech perception and production abilities contribute to the eventual goal of learning to read, write and spell. Two hypotheses are situated within this perspective. Firstly, that which builds on the motor-theory of speech, suggesting that the motor system (and therefore the timing of motor functions) plays a role in the development of language abilities. A second hypothesis focuses on the temporal structure of language which demands successful temporal processing in order to develop an appropriate language processing capacity. As such, both hypotheses consider that motor timing tasks are measures of the capacity for temporal processing which contributes to language development during childhood. Wolff (e.g. 2002) highlighted both hypotheses in his discussions of motor timing impairments, although he did not explicitly distinguish between them. The distinction is, however, useful for evaluating the evidence for the relationship between motor timing and the development of literacy difficulties in dyslexia.

3.2.1.2.a. Motor functions associated with language processing

The strength of links between motor and language control systems has been widely debated since Liberman's proposal that the perception of speech is affected by motor commands for speech production (Liberman, Cooper, Shankweiler, & Studdert-Kennedy, 1967; Liberman & Mattingly, 1985). These claims continue to receive support with evidence that speech perception and discrimination are mediated by activity in the motor cortices (for reviews see Devlin & Aydelott,

2009; Galantucci, Fowler, & Turvey, 2006; Kelso & Tuller, 1984). An evolutionary perspective predicts the recruitment of the motor system in language tasks to ensure that communication production systems are linked to systems required for perception of language (Galantucci et al., 2009). The timing of language related gestures often becomes synchronised (or entrained) with speech patterns and this entrainment is thought to assist with the organisation of information within these communicative systems (Allen, 1975; Browman & Goldstein, 1989; Cummins, 2009; Ojemann, 1984; Rusiewicz, 2010; Tzeng & Wang, 1984; Westermann & Miranda, 2004; M. Wilson & Wilson, 2005). Others oppose a strong version of the motor-theory of speech because language abilities can develop in the absence of speech production abilities (as in the case of dysarthria), although such opponents concede that the motor system may play a role in detecting syntactic structure by enabling prediction and anticipation of speech and speech-related gestures, allowing coordination and communication between two speakers (Scott, Mcgettigan, & Eisner, 2009; Werker & Tees, 1999).

The process of forming speech sounds begins in infancy with babbling at around 6 months, giving the child the opportunity for feedback from caregivers about the quality of the child's own vocalisations. A child also receives feedback from the perceptions of the auditory and somatosensory sensations created by the utterances (MacNeilage & Davis, 2001; Westermann & Miranda, 2004). Communicative synchrony between speakers and listeners allows the speaker to shape sounds into a functional form recognised by others in the child's environment (Doupe & Kuhl, 1999; Kuhl & Meltzoff, 1996; MacNeilage & Davis, 2001). The motor system is thought to assist in formulating predictions about this synchronisation to help process the speech signal. Neural feedback within the sensorimotor system has been shown to act as top-down modulation for the auditory system during passive listening tasks to auditory tone sequences (Arnal, 2012). By enabling prediction and anticipation of sounds in a rhythmic stream, such feedback is likely used to process other periodic stimuli like sound-stimuli in speech perception (Arnal & Giraud, 2012; Westermann & Miranda, 2004).

With this foundation in the motor-theory of speech in mind, it has been suggested that the temporal deficits in non-linguistic motor behaviour may be associated with impaired literacy development in children with dyslexia (Wolff, 2002). Any disruption in the perceived synchrony of sensorimotor events (as described in the studies of motor timing tasks cited above) or dynamics of co-articulated speech would be expected to affect the development of appropriate motor-speech relationships (Thomson et al., 2006; Wolff, 2002).

Additional analysis of the characteristics of motor timing performance in Wolff's (2002) sample showed that the group with dyslexia were able to match the 670 and 500ms stimulus intervals, but had larger and more variable anticipation times than the age matched controls. Similar results were found by Klipcera et al. (1981) and Thomson et al. (2008), although in the latter study the group

difference in anticipation times did not reach significance but was in the same direction. As described in Chapter 2, during motor synchronisation tasks, motor responses occur before the physical onset of the pacing stimulus (anticipation), a phenomenon thought to give rise to a synchronous perception of the two events (Aschersleben & Prinz, 1995; Aschersleben, 2002; Repp, 2005). When synchronising to a beat, anticipation times can be experimentally altered by changing the characteristics of the pacing stimulus or the response criteria. For example, synchronisation to visual pacing stimuli results in longer anticipation times than synchronisation to auditory stimuli (Kolers & Brewster, 1985; Repp & Penel, 2002) and producing paced responses with the hand gives rise to shorter anticipation responses than with the foot (Aschersleben & Prinz, 1995). The transmission times for sensory information from the stimulus are thought to vary under these different conditions such that the asynchrony between the response and stimulus has to be altered in order to achieve a perception of synchrony (Jäncke, Loose, et al., 2000; Kolers & Brewster, 1985; Repp & Penel, 2002; Repp, 2005).

The longer anticipation times recorded in children with dyslexia (Klipcera et al., 1981; Wolff, 2002) therefore may be due to a difference in the time these children require between the action and the perception in order to achieve a perception of synchrony. This could reflect a failure to track or make use of feedback from the sensory or motor systems that would allow prediction of future stimuli and enable accurate synchronisation. It is proposed that the difficulty in achieving simultaneity between responses and the environment has the potential to affect the development of linguistic abilities earlier in childhood when speech production and perception rely on temporal synchrony and stimulus-response pairings (Thomson et al., 2006; Wolff, 2002). In summary, a lack of information about the synchrony of stimuli and responses may disrupt the prediction and anticipation of future stimuli, affecting the quality of the feedback generated within the motor system (whether for motor tasks or linguistic stimuli).

3.2.1.2.b. Timing ability related to language functions

Alongside these suggestions that the motor system has a direct involvement in communicative behaviours, there is also support for the idea that the ability to process the temporal characteristics of language contributes to language development (a theory aligned with the temporal processing hypothesis described in Chapter 1). The evidence described above, supporting the top-down influence of temporal oscillations in the motor cortex on auditory processing (Arnal, 2012; Westermann & Miranda, 2004), highlights a role for temporal processing in functions that rely on the changes occurring over time, such as language. More specifically timing abilities are thought to be important in processing the temporal aspects of speech (Goswami et al., 2002; Goswami, Huss, Mead, Fosker, & Verney, 2013; Huss, Verney, Fosker, Mead, & Goswami, 2011; Leong, Hämäläinen, Soltész, & Goswami, 2011; Wolff, 2002) which are essential for extracting the segments of the speech stream.

Prosody in speech comprises the prominent elements of the acoustic stream such as the patterns, contrasts, and units (accents, syllables, words, phrases) which allow organisation and segmentation of the speech signal, providing information about syntax and semantics (Shattuck-Hufnagel & Turk, 1996; Speer, Kjelgaard, & Dobroth, 1996). Prosody in speech is often likened to rhythm in music, requiring analysis of complex patterns to generate a stable perception (Cummins & Port, 1998; Patel & Daniele, 2003). Each language has a unique prosodic structure and pre-linguistic infants are sensitive to this rhythmic property of their native-language (Byers-Heinlein, Burns, & Werker, 2010; Petitto, Holowka, Sergio, & Ostry, 2001; Ramus, 2002). This structure is thought to help cue infants to word and syllable boundaries within their native language, scaffolding language acquisition (Petitto et al., 2001; Ramus, 2002; Wood & Terrell, 1998) and the development of appropriate phonological representations (Beattie & Manis, 2011; Holliman, Wood, & Sheehy, 2010). In light of the importance of prosody in language development, researchers have examined the relationship between rhythmic abilities and literacy development.

Rhythmic processing and dyslexia

As described in Chapter 2, rhythmic temporal processing is not identical to processing isochronous stimuli because the former typically involves stimuli characterised by accented elements of varying durations and temporal qualities. However, such stimuli may be closer in form to speech stimuli (Ramus, Nespor, & Mehler, 1999 but see Arvaniti, 2009) and so rhythmic processing has been investigated to further explore the relationship between rhythmic timing and literacy development.

Two longitudinal studies of rhythmic performance support a link between rhythm reproduction ability and later reading skills in both good and poor readers (David, Wade-woolley, Kirby, & Smithrim, 2007; Dellatolas, Watier, Le Normand, Lubart, & Chevrie-Muller, 2009). David and colleagues followed school children from grades one to five, measuring rhythmic timing skills along with phonological awareness, reading ability and rapid naming skills. The results indicated that rhythm abilities at grade 1 were predictive of reading ability in grade 5 after controlling for phonological ability and naming speed. They suggested that rhythmic skills improve segmentation and sequencing skills as well as phonological processing and naming speed. Segmentation ability continued to be important for reading development from grade one to five for both the initial learning of sounds and the later acquisition of polysyllabic words. Similar results were found by Dellatolas et al. (2009) who showed that rhythm processing at grade one was predictive of reading at grade two in good and poor readers. A link between early phonological ability and later reading ability was not confirmed in this study, although this result may have been due to the limited range of phonological measures employed. Both studies assessed rhythm reproduction with rather insensitive hand clapping, foot tapping or walking tasks, judged for success by observers. This methodology therefore precludes analysis of the types of temporal errors made by the children and

whether these are similar to those found in studies of motor timing that used objective recording of isochronous finger tapping.

To establish the extent of timing difficulties in dyslexia a rhythm reproduction task was also included in the study conducted by Wolff (2002). This assessed the ability to keep in time with an anisochronous rhythm, either paced by a metronome or unpaced. The timing of finger tap responses was measured with millisecond accuracy. In contrast to the control groups who were able to match the target rhythms, the reading impaired sample were much less accurate at producing appropriately stressed rhythms especially when performing in time to the metronome, often failing to represent short and long intervals within the sequence or their order. These results indicate a difficulty with anisochronous as well as isochronous rhythm production in children with reading difficulties. However, the extent to which this rhythmic performance was related to reading problems was not established.

Similar group differences were found by Overy, Nicolson, Fawcett and Clarke (2003). A range of tasks assessing rhythm, tempo and beat perception as well as rapid perception tasks (note order or note number discrimination) were administered. On the individual tasks, there were no significant differences between boys with and without dyslexia. The boys with dyslexia did, however, show generally poorer performance across the tasks involving rhythmic timing, and tapping to a rhythm was strongly correlated with spelling ability. The lack of groups differences for the individual tasks, such as finger tapping to a beat, may have resulted from the small number of responses collected for these tasks, with only eight finger taps recorded in comparison to the thirty or more recorded in many of the studies of motor timing behaviour (Klipcera et al., 1981; Wolff, Michel, Ovrut, et al., 1990).

A further longitudinal study assessed melody and rhythm discrimination skills in children receiving musical training (Forgeard, Schlaug, Norton, Rosam, & Iyengar, 2008). Many of the children assessed already had a high level of musical experience prior to the study and were tested before and after further musical training. Even at baseline, the authors found that melody and rhythm discrimination abilities measured using a same-different judgement task were associated with phonological awareness in typically developing children. Similar correlations were found for a group of ten year olds with dyslexia. The children were followed up after 14 and 31 months of training and rhythm continued to play a role in word attack skills until age 8. After this time the amount of variance predicted decreased which may have been accounted for by the loss of participants from the sample at the later time points. More recently Lathroum (2011) also found that music perception (operationalised through measures of rhythm, pitch and melody) accounted for unique variance in phonological awareness after controlling for age and general intelligence. The association between rhythm and music perception was as high as for the other variables of pitch and melody.

A study which brought together a number of measures of literacy, rhythmic meter perception, beat detection and syllable stress (which will be discussed further below) similarly found that the ability to detect the metrical structure of rhythms was important for reading, spelling and phonological awareness (Huss et al., 2011). Employing two control samples to assess the effect of development, Huss et al. found that the children with dyslexia performed below the level of the chronological age matched controls, but at a similar level to children matched for reading level. The group differences on the same-different rhythm judgement task were found across a number of variables such as time structure, accent position and note number and length. The results indicated that metrical perception is an important predictor of reading development across the spectrum of reading abilities, with associations present even after controlling for memory and phonological awareness. A follow up of the children in this study a year later showed that the detection of beat structure remained a strong and significant predictor of single word reading, reading comprehension and non-word reading even after controlling for earlier phonological ability (Goswami et al., 2013). The authors highlighted the similarities between rhythmic structure of music and language and concluded that sensitivity to temporal events in language has an effect on the perception of structure in language and contributes to the appropriate development of phonological awareness.

Further support for the link between timing ability and language development comes from a study of temporal discrimination skills in a group of 11 year old children including both typically developing and discrepant readers (Grube, Kumar, Cooper, Turton, & Griffiths, 2012). Grube et al. found that the ability to detect short isochronous sequences with deviant tones or deviant metrical structure was related to phonological processing ability, but the detection of temporal discrepancies in longer stimulus trains was not predictive of literacy skills to the same degree. They proposed that rhythmic processing for short sequences reflects the short prosodic components in language that are especially important for linguistic development at this age. Because the recognition of prosodic patterns in speech relies on the ability to process components in time, these temporal difficulties may contribute to the impaired phonological representations that underlie the profile of dyslexia. Indeed a recent training study comparing phonological and rhythm based interventions (targeting prosodic-like beat based temporal processing) for children with reading difficulties found that both types of programme were equally as successful in improving phonological awareness (Thomson, Leong, & Goswami, 2013).

This group of studies suggest that children with reading difficulties have a rhythm processing deficit and that difficulties in this area of processing are predictive of literacy development. The parallels between rhythmic processing and prosody in speech mean that deficits in this area may influence the ability to appropriately organise, segment and predict speech sounds with consequences for development of phonological ability (Holliman et al., 2010; Holliman, Wood, & Sheehy, 2012). Both isochronous and anisochronous (e.g. rhythmic) motor timing tasks require skills such as beat

detection but rhythm tasks also involve order judgement and memory and it is therefore unclear whether rhythm related deficits are due to a simple timing problem or problems with serial order or memory. Indeed brain imaging studies show increased recruitment of brain areas across the time-processing network when more complex rhythms are processed (including in the primary motor cortex, supplementary motor area, basal ganglia, thalamus and cerebellum; Dhamala et al., 2003; Grahn & Brett, 2007). Huss et al. (2011) controlled for memory in the correlation between rhythm processing and reading so the presence of rhythmic deficits is unlikely to be wholly due to any co-existing memory deficits. Performance on the isochronous motor timing and rhythmical tasks may therefore be reflective of a similar temporal difficulty in children with dyslexia. Grahn and Brett (2007) compared behavioural and neural responses for simple versus complex rhythmic tasks and found that even stimuli with a simple rhythmic structure activated the beat-based temporal processing system required for isochronous sequence processing. This beat-detection system may therefore scaffold processing of a range of temporal stimuli including rhythms and speech. In dyslexia the performance difficulty on simple isochronous tasks may reflect an impairment in the beat-detection or timekeeping system that is required for processing the metrical structure of both simple and complex rhythmic stimuli.

A route to the phonological deficit

In a similar way to the established hypothesis regarding temporal processing in dyslexia (discussed in Chapter 1), a route has been proposed which links the timing deficit to disruption in phonological representations frequently seen in dyslexia (Wolff, 2002). The evidence of difficulties with isochronous and anisochronous timing tasks suggests that children with dyslexia fail to achieve appropriate stimulus-response representations for elements occurring in time. For rhythm and prosody the relationships between the stressed and unstressed units in a stream are important for the perception of the rhythmic forms or phrases in music or speech (Arvaniti, 2009; Dauer, 1983; Holliman et al., 2010; Villing, Repp, Ward, & Timoney, 2011; Werker & Tees, 1999; Wood & Terrell, 1998) and so a failure in temporal representations would affect prosodic processing. The perceptual-centre (or p-centre) of acoustic stimuli has been examined as a potential marker of the stressed components within speech (and non-speech) sounds (see Section 2.4.2.1). The p-centre is a term used to describe the moment at which an acoustic event is perceived to occur (Morton, Marcus, & Frankish, 1976) and is therefore crucial for achieving synchronisation to prominent components of acoustic stimuli. In isochronous timing, the p-centre (rather than the onset of a stimulus) is used to produce regularly timed responses in synchronisation tasks (Vos et al., 1995) and in speech it is used to detect the peaks of syllables (de Jong, 1994; Marcus, 1981). Any impairment in the recognition of p-centres due to the difficulty in synchronisation to perceptual events may therefore affect the sorts of rhythmic processing described above.

Goswami et al. (2002) formalised this theory, proposing that the difficulty seen in dyslexia with rhythmic timing may extend to the detection of syllabic rhythms or prosody in speech by impairing the acquisition of speech sounds with subsequent effects of the quality of phonological representations. Equally, they proposed that *superior* rhythmic ability may scaffold the development of phonological processing. In support of this theory, Goswami and colleagues have found that children with reading difficulties are less sensitive to changes in a particular acoustic element that signals p-centres, namely amplitude envelopes or rise time (Goswami et al., 2002), and have associated difficulties with prosodic sensitivity (Goswami, Gerson, & Astruc, 2010). Rise times are defined as the rate of change of the intensity of a stimulus such as a speech sound and the onset of this energy component in a speech sound affects the location of its p-centre (Scott, 1998).

Goswami (2011) proposed that the temporal deficits in dyslexia arise from inefficient phase locking of neuronal oscillations to the frequencies important for the recognition of stress or prosody in speech, with the inefficiency reflected in both rise time and motor timing tasks. In the same way that neuronal oscillations have been found to track pacing stimuli for motor synchronisation (Gerloff et al., 1998), components of the speech signal are processed by different sampling windows which operate through neuronal networks oscillating at particular frequencies (Luo & Poeppel, 2007). Goswami builds on this schema to suggest that particular sampling windows, essential for segmenting speech into syllabic and prosodic components, may be affected in dyslexia. As such, neuronal oscillations in the Theta (4-10Hz) and Delta (1.5-4Hz) frequency bands are implicated because the intervals between speech syllable have, on average, a period of 200ms (5Hz) and incorporate stressed syllables every 500ms (2Hz) (Dauer, 1983). Any inefficiency in the oscillatory networks processing these frequencies may lead to failures in appropriately tracking the dynamical form of the speech stream with subsequent effects on phonological processing. Speech comprehension is known to be supported by monitoring the rhythmic nature of speech via neuronal phase locking to envelope cues, both for intelligible speech or speech where linguistic cues have been degraded (Peelle & Davis, 2012; Peelle, Gross, & Davis, 2012). This hypothesis builds on previous evidence that children with dyslexia fail to detect amplitude and frequency modulations in the 2-10Hz range (Talcott et al., 2000; Witton et al., 1998). Goswami (2011) intimates that disruption of the low frequency oscillations will lead to reliance on parallel high frequency oscillatory activity in the Gamma range thought to be important for phonemic segmentation. This altered method of processing in turn contributes to the cognitive profile seen in children with dyslexia.

To test these ideas an amplitude modulation detection task has been used to assess rise time sensitivity (e.g. Goswami et al., 2002; Thomson & Goswami, 2008). This task allows manipulation of the amplitude envelope of an auditory stimulus and the repeated stimuli give an impression of an isochronous beat (with ISIs of 150ms or larger) when tones have short rise times. Participants are required to decide whether a beat is present as the rise time duration is changed. In a series of

studies, adults and children with dyslexia have been found to show lower sensitivity to these modulated beats (Goswami et al., 2002; Goswami, Fosker, Huss, & Mead, 2011; Huss et al., 2011; Leong et al., 2011; Thomson & Goswami, 2008). This sensitivity has also been found to be correlated with key symptoms of dyslexia such as reading, spelling, phonological awareness, phonological short term memory and rapid naming, even after controlling for general ability levels. The associations have also been found for precocious readers who show improved sensitivity compared to matched controls (Goswami et al., 2002) and across different languages (Goswami, Wang, et al., 2010; Hämäläinen, Leppänen, Torppa, Müller, & Lyytinen, 2005; Muneaux et al., 2004), pointing to the potential universal importance of these rhythmic processing skills across the ability range and across different languages.

In the study conducted by Huss and colleagues, rise time sensitivity was measured alongside the rhythmic processing tasks and rise time detection was found to be predictive of literacy skills and was associated with musical meter perception (the discrimination of strong and weak beats in short musical trains) (Huss et al., 2011). Similar associations between rise time and stress have been found for speech stimuli in adults with dyslexia (Leong et al., 2011) which may explain the difficulties children with dyslexia have with discriminating the stress patterns in sentences (Wood & Terrell, 1998) and in two-word object names (Holliman, Wood, & Sheehy, 2008). Thus, the difficulty in setting up an internal representation of rhythmic stimuli for both linguistic and non-linguistic acoustic streams with temporal qualities may be explained by a failure to locate the prominent features of stimuli with knock on effects for the development of appropriate phonological representations of speech.

With evidence for beat detection or synchronisation problems in both motor and speech domains in children with dyslexia, it is possible that these difficulties stem from a similar underlying temporal processing difficulty. When the overlap between these factors has been assessed, groups of adults and children with dyslexia have shown reduced sensitivity to amplitude envelope onsets alongside motor timing impairments, with significant associations found between the two measures (Thomson et al., 2006; Thomson & Goswami, 2008). For the adults, unpaced motor timing variability on finger tapping tasks was related to rise time sensitivity and phonological short term memory, and paced motor timing variability was related to reading ability. When other factors such as age and reasoning ability were controlled, only rise time and duration discrimination scores were unique predictors of reading and spelling (Thomson et al., 2006). In the study of children, paced and unpaced motor timing performance was related to rise time detection as well as measures of literacy skill including reading, spelling and phonological awareness. Further mediation analyses indicated that the relationship between paced motor timing and literacy skills was not governed by rise time performance in these children (Thomson & Goswami, 2008). The adults appeared to no longer show a consistent link between motor timing and literacy, but rather the route between timing and literacy

was mediated by rise time detection ability, essential for the temporal segmentation of speech. In contrast a direct relationship between motor timing and literacy skill was present in children, alongside associations between rise time detection and literacy skill. In light of the important contributions of the motor system in scaffolding temporal aspects of speech and language development early in life this maturational difference is not surprising.

It is worth noting that across both these studies, the groups with dyslexia showed increased variability in motor timing in both the paced and unpaced conditions. However, for the adults the timing measure that was related to reading ability was the unpaced (continuation without the beat) task whereas in the children the strongest relationships were found for the paced (synchronise to the beat task). The factor that is most important for timing in children may therefore be the synchronisation component of the task. This result is predicted by the rise time hypothesis described above (Goswami et al., 2002) and given the importance of p-centre location for synchronisation performance (Vos et al., 1995). However, it is not conclusive that a p-centre deficit is the core basis for temporal difficulties because many of the earlier studies of motor timing in dyslexia found group performance differences for unpaced tasks (Klipcera et al., 1981; Waber et al., 2000; Wolff et al., 1984; Wolff, Michel, Ovrut, et al., 1990). Unpaced, continuation motor timing paradigms only require the registration of the participants own tap, without any need to monitor p-centres within stimulus trains. Therefore, unless the perceptual deficit extends to the registration of the participants own finger tap responses, timekeeping mechanisms may be the site of impairment, playing a role in monitoring the on-going response output (Lewis & Miall, 2003; Torre & Delignières, 2008; Vorberg & Wing, 1996).

Further evidence that the difficulty may be limited to the perception of synchrony comes from the different timing tasks administered by Wolff (2002). He measured the ability of adolescents to produce a timed rhythmic response (unpaced or paced) in time with a metronome set to either the participants preferred rate of tapping or a faster rate. In contrast to the control groups who were able to match target rhythms, the reading impaired children were much less accurate at producing appropriately stressed rhythms especially when performing in time to the metronome, often failing to represent the short or long intervals within the sequence or their serial order. Finger tapping at the participants preferred rate should improve performance because it should reflect the underlying period of a person's internal timekeeping mechanism or a multiple of this period (Vanneste, Pouthas, & Wearden, 2001). Thus, the timing difficulties reported may all stem from a problem with the monitoring of synchrony to the internal timekeeping mechanisms themselves.

An additional paradigm implemented by Wolff (2002) required participants to synchronise to one stimulus rate for ten seconds (at 666ms ISIs; 1.5Hz) and then match the rate when it changed (to 500ms, then 400ms, then 500ms intervals for ten seconds each; 2, 2.5 and 2Hz). This task, with large

step changes, requires the conscious detection of a change in the period of the stimulus and subsequent updating of the period of the internal timekeeping system to match the new rate (Repp, 2001a, 2005). Badian and Wolff (1977) also implemented this task in their study of timing in discrepant readers but did not report any results for the task. Wolff (2002) found that the group with dyslexia took longer to readjust to the new rate than control participants, who only needed two or three beats to return to synchronous tapping. This again may indicate a deficit either in detecting synchrony or in updating the internal representation of the stimulus within timekeeping systems to match the new stimulus rate. Both of these failures could be described within the temporal sampling deficit put forward by Goswami (2011), as a failure to represent the stimulus rate with the appropriate neuronal oscillations which also contributes to failures in setting up appropriate phonological representations when learning speech and language.

The review presented here in Section 3.2.1, demonstrates the relevance of a motor timing deficit as a measure of temporal processing in dyslexia, with timing contributing to language development, through the functions of either the motor or language systems that allow appropriate sound based representations of language to be acquired. It is not the purpose of this thesis to test any of these claims about the contribution of temporal processing, but to establish more about the specific nature of the timing deficit that is associated with dyslexia.

3.2.2. Motor timing in the context of other deficits in dyslexia

The motor timing task involves stimuli which occur repeatedly and (typically) rapidly in time. Participants are required to attend to and recognise the temporal features of the stimulus train and entrain to the tempo. In both the synchronisation and continuation phases of the task responses need to be synchronised to either the stimulus or an internal representation of the stimulus, with tactile feedback being received from the finger when responses are made. In addition to the problems described with motor timing tasks, other temporal processing difficulties experienced by children and adults with dyslexia can be considered alongside the motor timing deficits already described.

3.2.2.1. Simultaneity and order

In order to judge whether responses produced are in time with external stimuli or internal representations of stimuli in a motor timing task, participants must examine the simultaneity between stimuli and responses. As already noted, the increased anticipation times found on motor timing tasks in children with dyslexia (Wolff, 2002) suggest that these participants may have a difficulty in judging the coincidence between stimuli and responses. The ability to judge simultaneity of two stimuli has been previously measured in populations with dyslexia using gap detection, individuation or fusion tasks and there is evidence that children with dyslexia require longer ISIs to be able to perceive two stimuli as being separate rather than one (for reviews see Farmer & Klein, 1995;

Rosen, 2003). Children with dyslexia struggle to repeat the order of pairs of rapidly presented sounds regardless of whether the stimuli are linguistic or non-linguistic in form (Tallal & Piercy, 1973, 1974; Tallal, 1980). Tallal suggested that these difficulties could disrupt the ability to decode the rapid dynamic aspects of speech leading to the profile of difficulties seen in dyslexia (Tallal & Piercy, 1978; Tallal, 2008) via the route from temporal processing difficulties to language difficulties described in Section 3.2.1.2.b. An impairment in judging simultaneity may prevent children from noticing any discrepancy in the occurrence, or order of, stimuli and as such, the larger anticipation exhibited by these children may not give rise to the same impression of a stimulus-response discrepancy as it might to a child without dyslexia.

3.2.2.2. Rapid and dynamic nature of stimuli

There has been some debate in the literature about whether the above difficulties are only present at particular stimulus presentation rates. In contrast to Tallal's finding of an order judgement difficulty with rapidly presented stimuli, other research has found that the inability of children with dyslexia to judge the order of two stimuli is also present when longer ISIs were employed (Chiappe, Stringer, Siegel, & Stanovich, 2002; Share, Jorm, Maclean, & Matthews, 2002; Waber et al., 2001). Although the ISIs tested (100-400ms; 10-2.5Hz) all fall within the range that would be considered millisecond timing for a motor timing task (Lewis & Miall, 2003; Mauk & Buonomano, 2004) it is thought that judging the order of stimuli at rapid ISIs (around 100ms) is governed by automatic perceptual processes compared to longer ISIs (around 400ms) which require judgements based more on memory (Waber et al., 2001). As such, the deficit with the more rapid stimuli is more likely relevant to the motor timing tasks under consideration here, where processing is performed automatically through perceptual or motor systems.

Another related factor discussed by Tallal that may be relevant to motor timing behaviour is the dynamic nature of temporal stimuli. Processing dynamic stimuli has been termed the "perception of rate" (Farmer & Klein, 1995; Tallal, 1980; Witton et al., 1998), i.e. processing stimuli with features that change over time. Children with dyslexia show impairments in processing a range of visual and auditory stimuli with features that change in this manner, including coherent motion and frequency or amplitude modulation tasks (Hämäläinen, Leppänen, & Lyytinen, 2012; Stein & Talcott, 1999). A deficit in this type of processing is thought to affect processing of the dynamic aspects of speech. Such findings have been grouped together by Stein and colleagues within the magnocellular deficit hypothesis (see Chapter 1) which assumes that cellular differences in the magnocellular layers within sensory systems lead to an insensitivity to the dynamic aspects of stimuli (Renvall & Hari, 1997; Stein & Walsh, 1997; Stein, 2001). Although originally intended to explain deficits in the visual modality in dyslexia, the theory has been expanded to the auditory and tactile domains (Stein, 2001). The likelihood that the primary source of temporal processing deficits in dyslexia lies in the magnocellular system is still debated (see Chapter 1 and Hulme, Snowling, Caravolas, & Carroll, 2005; Ramus, 2003;

Skottun, 2000), but such work has focused attention on the importance of brain based mechanisms in contributing to temporal processing behaviour.

A difference in the neural efficiency of the brain has for a number of years been put forward as a potential source of the temporal processing difficulties recorded in dyslexia, although not all authors have been as explicit as Stein in identifying a single responsible brain pathway. For example, Llinas (1993) suggested that the clock which determines the rate of neural firing may be affected in dyslexia, leading to ineffectual binding of the different cortico-thalamic networks required for efficient temporal processing. Similarly, it is proposed that the deficit in dyslexia stems from the slower rate at which sensory buffers are filled and subsequently processed, due to differences in neuronal oscillations (either cortically or subcortically) (Hari & Kiesilä, 1996) such that rapidly presented stimuli are divided into longer chunks which reduce the precision of representations for rapid temporal stimuli (Hari & Kiesilä, 1996; Hari & Renvall, 2001; Renvall & Hari, 1997). This view is supported by Merzenich et al. who recognised that the temporal integration periods of the neural system are essential for determining the temporal characteristics of stimuli, such as stimulus asynchronies (Merzenich, Schreiner, Jenkins, & Wang, 1993). As highlighted in Chapter 1, differences in neuronal viability are present in the brains of individuals with dyslexia (Deutsch et al., 2005; Klingberg et al., 2000; Nagy et al., 2004; Niogi & McCandliss, 2006; Steinbrink et al., 2008) as well as differences in the manner in which populations of neurons respond to stimuli (Schulte-Körne & Bruder, 2010) supporting the presence of a functional difference in the neuronal firing patterns. Furthermore, some of the genetic risk factors associated with dyslexia are implicated in processes that contribute to neuronal migration and axonal development (Galaburda et al., 2006; Meng et al., 2005; Paracchini et al., 2006).

It is not entirely clear how these brain-based differences might affect performance on motor timing tasks. For example, they could result in general slowness in the transmission of signals across the whole timing system (from sensory registration, through to timekeeping entrainment and response generation). Under the magnocellular hypothesis, the difficulties might only be expected within the processing of sensory information from stimuli and tactile responses, with knock on effects for timing output. Hari and Renvall's (2001) theory would suggest that children take longer to disengage from each stimulus resulting in longer sensory input chunks or cognitive windows, with subsequent effects on the output of timed responses. It is possible to reconcile such theories with the more contemporary analysis presented by Goswami (2011) such that children fail to sample the temporal stream of information appropriately due to failures to phase lock neural oscillations to temporal stimuli, with special reference to the sampling of rates relevant for language.

3.2.2.3. Motor impairments

A major component of motor timing tasks is the implementation of timed responses carried out by motor effectors such as the finger. Within the Wing-Kristofferson model of timing (described in Chapter 2), this motor implementation process is separated from the central timekeeping process that monitors the stimulus rate and produces a timed output to be implemented through the motor system. Previous evidence has indicated that some children with dyslexia show difficulties with performance on an array of motor tasks, not just those overtly involving timing. For example impairments have been found across measures of postural control and balance, fine motor movements (e.g. peg-moving or rapid finger movements) and eye movement control (for reviews see Raberger & Wimmer, 2003; Stoodley & Stein, 2011). Such evidence has primarily been discussed in relation to the automaticity-cerebellar theory of dyslexia (e.g. Fawcett & Nicolson, 2004; Nicolson & Fawcett, 1990, 2011) described in Chapter 1. Nicolson and Fawcett proposed that an impairment in procedural learning and in the automatisation of skills affects articulation alongside fine and gross motor control, leading to disruption of the learning pathway for normal language acquisition. As described in Chapter 1, the specificity of this hypothesis has been questioned, yet such evidence it highlights an alternative route by which a motor timing deficit might arise, via a failure of motor control rather than of central timekeeping.

Results from the studies examining motor timing in dyslexia argue against this position, however. For example, Thomson and Goswami (2008) measured motor dexterity on a peg moving task alongside the auditory and motor timing measures and found that dexterity did not explain any of the variance in the relationships between motor timing, auditory rhythm processing and language development. Using a principle components analysis, Stanford and Barratt (1996) examined the performance dimensions associated with motor tasks in typically developing male adolescents and reached a similar conclusion. Finger tapping performance formed part of a "temporal information processing" factor alongside language measures and time judgement ability. In contrast, motor dexterity on a pegboard task was related to a separate "speed and accuracy" factor along with reaction time performance. Such findings indicate that any motor deficit in dyslexia is unlikely to explain the variable or inaccurate performance found on motor timing tasks.

A further argument against any motor deficit accounting specifically for the timing deficit associated with dyslexia is that the presence of motor difficulties in dyslexia may be accounted for by presence of symptoms of co-occurring disorders such as Attention Deficit-Hyperactivity Disorder (ADHD) (Denckla, Rudel, Chapman, & Krieger, 1985; Raberger & Wimmer, 2003; Ramus et al., 2003; Rochelle & Talcott, 2006; Rochelle et al., 2009). With extremely high rates of overlap between symptoms of dyslexia and ADHD (Willcutt & Pennington, 2000) the motor timing impairments found in children and adults with dyslexia may result from an additional moderator variable which accounts for either the presence of ADHD or the overlap between the two disorders in an individual. If ADHD were to

account for the any motor implementation difficulties it would be expected that the motor implementation component derived from the Wing-Kristofferson model would be more strongly associated with symptoms of ADHD. Children with ADHD do experience motor timing difficulties that resemble those found in children (as detailed below in Section 3.3), however some studies have concluded that these difficulties should be attributed to centralised timing difficulties rather than a motor impairment (Toplak, Dockstader, & Tannock, 2006).

Evaluating the extent to which the presence of ADHD might account for motor timing impairments in children and adults with reading difficulties is challenging due to the lack of reporting of the presence of ADHD in many of the studies of timing in dyslexia (see Table 3.; Badian & Wolff, 1977; Klipcera, Wolff, & Drake, 1981; Wolff, Cohen, & Drake, 1984; Wolff, Michel, Ovrut, & Drake, 1990). More recently, increased awareness of the complex and overlapping aetiologies of developmental disorders (Plomin & Kovas, 2005; Willcutt et al., 2010) has led to the use of criteria which exclude children with co-occurring diagnoses (Thomson et al., 2006; Thomson & Goswami, 2008; Waber et al., 2000; Wolff, 2002). Studies which have limited their samples to children with specific reading disorders suggest that some difficulties with motor timing performance are specifically related to dyslexia (Thomson & Goswami, 2008) although mild symptoms of ADHD may have been present in participants in such studies. Such symptoms may contribute to the variance in reading explained by timing performance. In order to provide a comparison on motor timing difficulties in dyslexia and ADHD, the evidence for timing impairments in ADHD is considered below (Section 3.3).

3.2.3. Summary of the motor timing difficulty in dyslexia

Research has demonstrated that children with reading difficulties show impairments on timing tasks that employ either isochronous or anisochronous rhythms and with both synchronisation and continuation motor timing paradigms. The differences shown are typically increased variability in the IRIs produced together with extended anticipation times. The evidence described suggests that there may be some alteration in the registration of stimuli or a lack of awareness of discrepancies between stimuli and responses. The presence of greater variability may indicate that the timekeeping system generates more variable outputs or that the variability in responses compared to stimuli is not recognised. Alternatively, the motor implementation component of timing behaviour may not be consistent over time, although this is less likely. Such performance difficulties have been linked to language development through hypotheses suggesting that aspects of temporal processing are required for appropriate development of speech production and perception mechanisms with downstream effects for the development of language components such as phonological processing. Anisochronous timing tasks may be closer in form to the prosodic features of speech and require timing for prediction and anticipation of future intervals, although the complex rhythms involved utilise a more complex neural system for timing control. The presence of impairments on

isochronous tasks however, suggests the presence of a more basic impairment in temporal processing that may affect the ability to judge synchrony between simple actions and events or to maintain timed responses in the absence of stimuli.

In synchronised performance, a timekeeper produces pulses to be implemented by the motor timing system and these can be updated with reference to the pacing stimuli. The location of the perceptual centre (p-centre) is used to guide synchronisation rather than the onset of the stimulus. Despite the involvement of the stimuli, the timekeeping system also plays a role in timing control, evidenced by the occurrence of responses prior to the stimulus onset: the system anticipates the occurrence of stimuli rather than responding to its presence. In contrast, continuation tasks place greater demands on the timekeeping system to maintain a timed output in the absence of guidance from stimuli. Thus, the difficulty experienced by children with dyslexia on synchronisation tasks may arise from the perception of the guiding stimulus, the central representation of action-perception synchrony or from the timekeeping system. Given the difficulties children with dyslexia have with registering p-centres, or making judgements about dynamic stimuli it is possible that a perceptual difficulty explains the failure to appropriately synchronise to the beat. Nevertheless, children with reading difficulties also show impairments on continuation paradigms where entrainment and internal timekeeping are required and so these mechanisms may be responsible for the difficulties reported.

The associations between performance on synchronisation tasks and literacy skills appear to be stronger than those found for continuation performance. For example, Thomson and Goswami (2008) found significant correlations between literacy skills and synchronisation performance but not for continuation performance. Unfortunately these analyses collapsed across three different rates of performance, selecting the most reliable pairs of scores as determined by Cronbach's alpha for the measures of accuracy and variability for the paced and unpaced tasks meaning that it is difficult to evaluate which pairs of scores were responsible of the associations reported. Studies that have collapsed across both synchronisation and continuation phases of the tapping task in their analyses also make it difficult to compare across the two paradigms (Wolff et al., 1984). In spite of such problems, the group differences found between good and poor readers on both tasks suggest that children have a difficulty in achieving synchrony and/or a problem with the timekeeper that affects performance on both paced and unpaced tasks.

A third system involved in both tasks is the motor output system, referred to in the Wing-Kristofferson model as the implementation system. There has been some debate about the specificity of motor impairments linked to an automatisation deficit in dyslexia which would point towards a failure in the implementation or motor control elements of timing behaviour. However, it

is unlikely that the implementation system is responsible for the variability in timing seen in dyslexia given that motor dexterity does not typically account for variance in timing performance.

In relation to the response paradigm, children with reading difficulties typically showed performance decrements compared to controls on one- and two-handed, bimanual tapping tasks (bimanual synchronisation or alternation). The more complex bimanual tasks require additional planning for response generation and so whilst early studies focused on the deficit on these bimanual tasks, the additional processing demands of such tasks may have confounded assessment of the existence of more basic timing deficits. The evidence for difficulties on simpler single handed tapping tasks such as increased anticipation time, greater mean response intervals and greater response variability, implicates a problem in timekeeping which may or may not be accompanied by a difficulty with coordination between the left and right hands, such as an absence of the bimanual advantage (the reduced variability that typically accompanies bimanual tapping).

Associations have also been found between motor timing ability and measures of general intelligence which may be the result of temporal processing abilities contributing to a range of general learning capacities in other domains. For example, action-perception timing is important for future prediction, anticipation and learning of associations within the environment and may therefore scaffold a range of functions (Cohen, 2011). Such evidence indicates that there is a need to control for reasoning abilities in studies of motor timing, but where reasoning abilities have been accounted for results have indicated that the general intelligence does not account for the relationship between timing and literacy skills.

Across the studies using finger tapping tasks to assess motor timing, a number of different rates of finger tapping have been assessed, with ISIs ranging from 333ms to 1000ms (3-1Hz). The higher rates used in some of the research conducted by the Wolff group (e.g. 200-333ms i.e. 5-3Hz; Wolff et al., 1995) typically involved bimanual alternation with one hand tapping half as fast (i.e. with 400-666ms; 2.5-1.5Hz)). In contrast, the studies of unimanual tapping have focused on tapping intervals of between 400 and 666ms with children with dyslexia consistently showing motor timing impairments at these output speeds. The studies by Thomson, Goswami and colleagues suggest that for paced synchronisation tasks children and adults with reading difficulties show performance decrements across a range of rates of performance (from 400-666ms) and at faster rates for the unpaced continuation tasks (e.g. 500ms for adults and 400ms for children) (Thomson et al., 2006; Thomson & Goswami, 2008). That significant impairments are found in the 400-600ms range is especially relevant for the theories linking temporal processing and language development. The rates of syllable production and stress timing occur at a similar frequency of 2Hz and any disruption in this range is likely to have implications for the ability to follow the prosodic features of speech that

are known to scaffold the development of speech production, perception and later phonological processing.

Distinctions have been drawn between sub- and supra-second timing mechanisms, with sub-second tasks being controlled by more automatic timing systems that supra-second tasks (as explained in Chapter 2). The evidence above therefore implicates the automatic system for millisecond timing control in dyslexia. This system has close links with the motor system and is controlled through a network involving the basal ganglia, cerebellum, pre-motor cortex and SMA. Patients with lesions or disruptions in this area show similar performance decrements on motor timing tasks.

3.3. Motor timing and ADHD

ADHD and dyslexia are known to co-occur at such a high rate (>25%) (Pauc, 2005; Willcutt & Pennington, 2000) that they almost certainly share underlying cognitive, neurological and genetic risk factors (Pennington, 2006; Plomin & Kovas, 2005). Although independent clusters of symptoms are used to define these two disorders, recent models of the different routes through which co-morbidity can occur indicate that a categorical definition of developmental disorders should be disregarded in favour of dimensional models of symptom groups (Cramer et al., 2010). Hence, the likelihood of finding risk factors which overlap between disorders such as dyslexia and ADHD is high. At a neurophysiological level, a risk factor might explain an array of disorder-specific cognitive and behavioural symptoms, as well as symptoms that overlap between disorders. Examples of such risk factors have been discussed elsewhere, with phonological difficulties conveying risk for both dyslexia and language impairment, and serial naming deficits increasing the risk of developing dyslexia alone or in combination with ADHD (Pennington & Bishop 2009). It is therefore not surprising to find an area of cognitive impairment like motor timing that overlaps between dyslexia and ADHD. The disturbances of motor timing found in dyslexia may therefore be due to the presence of symptoms of ADHD or a feature of the overlap between the two disorders. In order to establish whether the motor timing difficulties are indeed reflective of an overlapping risk factor it is helpful to compare characteristics of performance at the behavioural level in these two disorders and to examine the putative mechanisms by which these difficulties are thought to arise in ADHD.

Timing is an ideal candidate for explaining the overlap between disorders as it is a basic functional element within the nervous system at so many levels of functioning. Wimpory (2002) proposed that timing may explain many of the variations that occur within child development, depending on the severity of timing failures, location in the processing system or age at which any difference occurs. Furthermore, recent genetic evidence has implicated generalist genes in the aetiology of developmental disorders, meaning that symptoms are likely to follow continuums of impairment across the population and that similar genes are likely to be related to several developmental

disorders (Plomin & Kovas, 2005). This multifactorial nature of disorders means that overlapping risk factors are likely to be common.

Motor timing (finger tapping) paradigms of the kind used with populations with dyslexia have also been employed in studies of ADHD. Such research has shown that children with ADHD fail to generate or sustain movements in time with stimuli and that this temporal processing deficit is linked to their symptoms of impulsivity and behaviour management (Barkley, Murphy, & Bush, 2001; Rubia, Halari, Christakou, & Taylor, 2009; Smith, Taylor, Rogers, Newman, & Rubia, 2002). As in dyslexia, difficulties are also found across a range of other temporal processing tasks such as duration discrimination, duration reproduction and duration production tasks (Toplak et al., 2006), although this review concentrates on the evidence from motor timing paradigms specifically. The studies reviewed below are summarised in Table 3.2.

Using visually paced finger tapping tasks to measure motor timing synchronisation abilities, Rubia and colleagues found larger IRI variability in children with ADHD and this was associated with symptoms of hyperactivity (Rubia et al., 2003; Rubia, Taylor, Taylor, & Sergeant, 1999). In this task participants tapped in time to a visual pacing stimulus (a repeated picture of an aeroplane) occurring at different stimulus rates and higher variability was found in the children with ADHD when stimuli were presented with ISIs of 700ms or smaller (less than 1.4Hz). A further study conducted by Rubia and colleagues using a similar visually paced synchronisation task and ISIs of 600ms (1.6Hz), did not find any behavioural differences between children with and without ADHD, but did find task related differences in brain activity between the groups when the blood-oxygen level dependent response was measured with fMRI (Rubia, Overmeyer, et al., 1999). In particular, the children with ADHD showed increased activity in parts of the timing network, such as in the SMA and putamen during the timing task.

Another examination timing in children with ADHD examined performance characteristics across movement speeds using bimodal stimuli (combined acoustic and visual stimuli) (Ben-Pazi, Gross-Tsur, Bergman, & Shalev, 2003). Seven different response rates were implemented (intervals of 166-1000ms; 6-1Hz), although none of the children were able to complete the task at the fastest rate. The children with ADHD tapped with more errors and greater variability than control children and did not appear to recalibrate their response speed when different stimulus rates were presented, producing a relatively constant mean response rate across trials. A follow up study found that timing errors were more common in children aged 6-7 than children aged 8-11 and that errors were related to the presence of hyperactivity and impulsivity symptoms (Ben-Pazi, Shalev, Gross-Tsur, & Bergman, 2006). Higher performance variability in both adults and children with ADHD has also been replicated in two recent studies (Valera et al., 2010; Zelaznik et al., 2012).

In contrast to these studies that used visual or bimodal pacing stimuli, Toplak and Tannock (2005a) compared performance across auditory and visual tasks. Participants with ADHD and a control group synchronised to a tone or a visual dot stimulus and then continued tapping without the stimulus for 11 taps. No group differences were found for performance variability in the continuation phase, although an interaction was present between interval rate and stimulus modality, with higher performance variability on the visual task with 1000ms intervals (1Hz). Toplak and Tannock (2005a) reported that 24% of their sample also had symptoms of reading disorder and discussed the potential importance of the overlap between timing deficits in ADHD and dyslexia although they did control for this in their analyses to establish the effect of any overlap.

In one of the only studies in the field to specifically assess the contribution of both ADHD and dyslexia to timing performance, Tiffin-Richards et al. (2004) found no significant differences in variability or mean IRI on synchronisation and continuation tapping tasks between groups with either dyslexia alone, ADHD alone or both dyslexia and ADHD. The only group difference reported by the authors was poorer performance on a complex rhythm reproduction task in children with dyslexia regardless of the presence or absence of a co-occurring diagnosis of ADHD. Although such timing difficulties are expected in children with dyslexia, the lack of a timing difficulty in the children with ADHD is in contrast to the earlier evidence for increased timing variability in children with ADHD in the absence of any other clinical conditions.

In a review of temporal processing difficulties in ADHD, Toplak and colleagues highlighted the inconsistent application of stimulus parameters, such as the modality of the pacing stimuli, in the research on motor timing and developmental disorders (Toplak et al., 2006). Such criticisms stem from the findings of an earlier study of duration perception where group differences for children with ADHD in duration perception were larger for a visual duration task than for an auditory task, suggesting that the relationship between attention and timing may be modulated by the sensory modality of the task stimuli (Toplak & Tannock, 2005a).

Considering these studies together, there appear to be inconsistencies in the findings which may be accounted for by methodological differences between these studies. The studies collecting only a small number of synchronisation or continuation responses (i.e. Tiffin-Richards et al., 2004; Toplak & Tannock, 2005a) may have collected insufficient data to give an accurate account of timing control, especially if children with symptoms of hyperactivity or inattention take more time to adjust to task conditions. Greater variability in participants with ADHD has been consistently found in the studies with a larger number of data points in the time series (Ben-Pazi et al., 2006; Rubia et al., 2003; Zelaznik et al., 2012). Secondly, the studies using acoustic pacing stimuli (Tiffin-Richards et al., 2004; Toplak & Tannock, 2005a) do not report significant group differences that are found using visual pacing stimuli (Rubia et al., 2003; Rubia, Taylor, et al., 1999), although a recent study employing

acoustic stimuli did find group differences in performance variability (Zelaznik et al., 2012). Because visual stimuli have been found to increase synchronisation variability in adults (see Chapter 2; Kolers & Brewster, 1985; Repp & Penel, 2002) the difference in the modality of pacing stimuli across these studies may account for the discrepancies in the group related results.

Table 3.2: Studies investigating motor timing performance and ADHD

Authors	Paradigm[A]	Synchronisation Phase	Continuation Phase	Stimulus Rate (ms)	Stimulus Modality (Stimulus Type)	Age Group	Type of ADHD group	Control Description	Group Difference	Description
Rubia, Taylor, Taylor & Sergeant (1999)	S&C	n.d.	n.d.	400, 700, 900, 1200, 1800	Visual (aeroplane)	6-12	Over-activity & inattention cut off scores, n=11	Without symptoms, n=11	*	Increased variability at slow speeds
Rubia, Noorloos, Smith, Gunning & Sergeant (2003)	S	n.d.		700, 900, 1200, 1800	Visual (aeroplane)	7-11	Clinical diagnoses, n=35	Without symptoms, n=11	*	Increased variability
Rubia, Overmeyer et al. (1999)	S	30s		600, 5000	Visual (aeroplane)	12-18	Clinically referred, n=24	Without symptoms, n=9		No behavioural group differences. Significant group differences in brain activation
Ben-Pazi, Gross-Tsur, Bergman & Shalev (2003)	S	40 taps		166, 200, 250, 285, 333, 400, 500, 1000	Combined visual (black diamond) and auditory (tone)	6-14	Clinical diagnoses, n=27	Age and gender matched, n=33	*	Faster response rate and increased errors
Ben-Pazi, Shalev, Gross-Tsur & Bergman (2006)	S	40 taps		200, 222, 250, 285, 333, 400, 500, 1000	Combined visual (black diamond) and auditory (tone)	6-12	DSM-IV criteria, n=64	Without symptoms, n=60	*	ADHD: faster and more variable tapping which was mediated by methylphenidate treatment
Toplak & Tannock (2005b)	C		11 taps	400, 1000	Separate trials: visual (dot) and auditory (tone)	13-18	Clinical diagnoses, n=46	Without symptoms, n=44	*	Group differences with visual stimuli at 1000ms intervals
Tiffin-Richards, Hasselhorn et al. (2004)	S (&C)	12		263, 500, 625, 750, 875, 1000	Auditory (tone) (continuation phase with own responses as pacing stimuli)	10-13	Clinical diagnoses, n=16	Control (n=18), Dyslexia (n=17) and Dyslexia+ADHD (n=17) groups, Dyslexia measured with IQ-Spelling discrepancy criteria, combined group had spelling discrepancy and parent reports of ADHD symptoms		No group differences in paced or unpaced variability in tapping. The dyslexia group had poorer performance on an additional complex rhythm reproduction task
Valera et al. (2010)	C	30 taps	30 taps	500	Auditory (tone)	18-55	Clinical diagnoses, n=21	Without symptoms, n=19	*	Group differences in overall variability and timekeeper variance
Zelaznik et al. (2012)	C	10 taps	40 taps	500	Auditory (tone)	7-12	Clinical diagnoses, n=27	Without symptoms: parent & teacher ratings, n=24	*	Group differences in overall variability, timekeeper and implementation variance

*In all experiments listed finger tapping was completed with one hand (unimanual). [A]Motor Timing Paradigm: S-Synchronisation C-Continuation. n.d. - not described. *Significant difference between groups.*

3.3.1. Explanations of motor timing deficits in ADHD

The difficulties shown by children and adults with ADHD on motor timing tasks and other experimental measures of temporal processing (reviewed by Toplak et al., 2006) have been explained through reference to the traditional theories of ADHD, such as in relation to executive function impairments or to the brain areas that are important in both temporal processing and ADHD. For example, a deficit in working memory may affect the ability to maintain a representation of interval durations prior to making a judgement about the duration (Barkley, 1997). This explanation accounts for findings of impaired duration discrimination with duration intervals in the order of magnitude of seconds or tens of seconds, but does not explain the performance differences found in millisecond timing tasks such as motor timing where an online memory of interval durations is not required (Buhusi & Meck, 2005; Lewis & Miall, 2003). Indeed it has been found that working memory ability does not predict performance on short duration time perception tasks (Smith et al., 2002).

Failures of inhibitory mechanisms may also affect temporal processing by preventing accumulation of the timekeeper pulses until the required delay threshold for a particular duration prior to responding (Barkley et al., 2001). This hypothesis stems from the delay-aversion account of ADHD which suggests children with ADHD are impaired at delaying responses or gratification, leading to impulsivity (Smith et al., 2002; Sonuga-Barke, Saxton, & Hall, 1998). It is unclear whether these mechanisms are sufficient to explain performance on short duration tasks where timekeeper pulses likely drive responses without the need for accumulation in memory. The failure to inhibit executive functions may also divert attentional capacity from timing, resulting in the performance differences shown across supra- and sub-second motor timing tasks (Zelaznik et al., 2012). The associations found between motor timing performance and the hyperactivity-impulsivity dimension support this viewpoint (Ben-Pazi et al., 2003; Rubia et al., 2003; Rubia, Overmeyer, et al., 1999). It is proposed that the temporal processing deficits in ADHD are sufficient to explain several behavioural characteristics of the disorder such as inattention and inhibition, by causing a lack of behavioural persistence and cognitive foresight on a range of tasks (Barkley et al., 2001; Rubia et al., 2009). Such theories imply that a primary impairment in ADHD is an insensitivity to temporal events or the ability to encode them (Smith et al., 2002; Toplak et al., 2006; Toplak & Tannock, 2005b).

The temporal processing impairments in ADHD have also been explained with reference to the neural substrates of timing which appear to overlap with the neural areas typically affected in ADHD. Timing tasks activate a network of brain areas including the basal ganglia, cerebellum and pre-frontal cortex, each with specific roles in timing control (as described earlier in Chapter 2). These areas each contribute to motor timing: the basal ganglia are implicated in the output of intervals due to the action of dopamine which suppresses noise and enhances useful signals (Grahn & Brett, 2007, 2009; Harrington et al., 1998; Hinton & Meck, 2004; Lewis & Miall, 2003; O'Boyle et al., 1996); the

cerebellum is important for accurate control of timing with damage to this region affecting accuracy on both millisecond and longer duration timing tasks (Ivry et al., 1988; Ivry & Keele, 1989; Nichelli, Alway, & Grafman, 1996; Penhune et al., 1998; Smith et al., 2003); and the pre-frontal and parietal cortices implicated in tasks with longer duration intervals that require higher-order cognitive processing, such as memory, decision making and attention (Lewis & Miall, 2003; Meck & Benson, 2002; Rammsayer & Lima, 1991; Rammsayer, 2006; Smith, Taylor, Brammer, Halari, & Rubia, 2008; Smith et al., 2003).

Several parts of this timing network are implicated in the brain pathology of ADHD. Processing in the pre-frontal cortex (PFC) is commonly found to be altered in children and adults with ADHD during tasks that assess executive functioning as well as during timing tasks (Barkley, 1997; Pennington & Ozonoff, 1996; Rubia et al., 2001; Rubia, Overmeyer, et al., 1999). However, the primacy of a frontal lobe deficit in ADHD is debated because differences in the functioning of the frontal cortex may result from abnormal development in other brain areas with which the frontal cortex is connected and/or compensatory effects (Boonstra, Oosterlaan, Sergeant, & Buitelaar, 2005; Halperin & Schulz, 2006; Sergeant, Geurts, & Oosterlaan, 2002). Metabolic over-activity in the striatum and reduced volume in the caudate is found in ADHD especially in late childhood with some recovery to typical levels by later adolescence (Castellanos & Tannock, 2002; Castellanos et al., 1996; J. S. Lee et al., 2005; Rubia, Overmeyer, et al., 1999). ADHD is also treated with pharmacological agents (e.g. methylphenidate) that reduce over-activity in the dopaminergic circuits of the basal ganglia (J. S. Lee et al., 2005) and the administration of methylphenidate is found to reduce errors and variability on timing tasks (Ben-Pazi et al., 2006). Finally, reduced cerebellar volume and differences in functional processing in the cerebellum are reported in cases with ADHD (Castellanos & Tannock, 2002; Castellanos et al., 1996; Durston et al., 2004; Mostofsky, Reiss, Lockhart, & Denckla, 1998; Valera et al., 2010). Indeed reduced activity has been found across the cerebellar-cortico-striatal timing network during motor timing tasks in adults with ADHD compared to controls (Valera et al., 2010).

Some studies have used models of timing behaviour to examine the statistical properties of motor timing performance. For example, the statistical method described by Wing and Kristofferson (1973a, 1973b), described in Chapter 2, has been used to show that lateral lesions of the cerebellum contribute to an increase in the variability that is attributed to timekeeping mechanisms (Harrington et al., 2004; Ivry et al., 1988). In contrast patients with Parkinson's or Huntington's disease, which affect the basal ganglia, show an increase in the variability attributed to both the timekeeping and implementation components (Freeman et al., 1996; Harrington et al., 1998). With evidence for structural and functional impairments in the basal ganglia and cerebellum in ADHD, it is likely that both of these statistical components of timing behaviour (timekeeper and implementation variance) may also be affected in ADHD (Toplak & Tannock, 2005a).

Two studies that assessed performance on an acoustically paced synchronise-and-continue finger tapping task in participants with ADHD applied the Wing-Kristofferson model to examine performance variability. As well as showing the common profile of greater within-participant variability, adults with ADHD had larger estimates of timekeeper variance than control participants but showed no differences in implementation variance (Valera et al., 2010). A study of children with ADHD, in contrast, found elevated levels of both timekeeper and implementation variance compared to controls (Zelaznik et al., 2012), results that are in agreement with Toplak and Tannock's (2005a) proposal that both components should be affected. Further analysis of the statistical properties of time series data revealed that children with ADHD employed a different timing strategy to control children, tending to update responses with respect to the stimulus (i.e. closed-loop timing which makes use of the stimuli to correct responses) rather than becoming entrained to the stimulus and following an internally generated timekeeper pulse. The difference between these the studies by Valera et al. and Zelaznik et al. may be the result of maturational effects on ADHD symptoms, such as the development of the basal ganglia by adolescence (Castellanos & Tannock, 2002) which may ameliorate the difference in implementation variance by adulthood.

3.4. Contrasting motor timing deficits in dyslexia and ADHD

In light of the evidence above, both dyslexia and ADHD appear to share difficulties in motor timing performance. In ADHD, the behavioural difficulty is commonly associated with symptoms of hyperactivity, whereas in dyslexia, motor timing is associated with key indicators of reading development. In the dyslexia field the explanations for motor timing disruption focus on cognitive level phenotypes that might explain the difficulties whereas in the ADHD field research focuses on cognitive level explanations that are linked to the biological basis of ADHD. This probably stems from the medical focus within ADHD research, given that ADHD is remediated through pharmaceutical measures and contrasts with dyslexia where remediation is primarily cognitive. This is not unexpected, but since both disorders have been shown to have a genetic basis, biological level explanations might be expected for dyslexia.

Except for the study by Tiffin-Richards et al. (2004), most of the research examining motor timing in ADHD did not specifically control for literacy abilities (e.g. Ben-Pazi et al., 2003, 2006; Toplak & Tannock, 2005a; Valera et al., 2010; Zelaznik et al., 2012), and similarly the studies of dyslexia often did not assessed the contribution of symptoms of ADHD (e.g. Thomson et al., 2006; Wolff et al., 1984; Wolff, Michel, & Ovrut, 1990). The studies that did control for this factor used screening questionnaires or required a diagnosis of a specific developmental disorder in the absence of other learning difficulties (Rubia et al., 2003; Rubia, Taylor, et al., 1999; Rubia, Overmeyer, et al., 1999; Thomson & Goswami, 2008; Waber et al., 2000). Without measuring symptoms that may overlap

but do not reach a threshold for diagnosis it is difficult to assess whether motor timing impairments result from the same underlying risk factor in these two populations.

As described earlier, some of the studies of dyslexia propose that movement difficulties in dyslexia exist due to the presence of ADHD symptoms alongside the diagnosis of dyslexia (Rochelle & Talcott, 2006). Therefore, an alternative theory is that the motor timing deficits are linked to this apparent motor impairment which affects movement and postural control. However, as in dyslexia, the evidence for temporal difficulties in ADHD across duration discrimination, reproduction and anticipation tasks indicates that the impairment is unlikely to be limited to temporal tasks with a motor component (Toplak et al., 2006). The studies which separated out timekeeper and implementation variance using the Wing-Kristofferson model of time series analysis found that both the central timekeeping and motor implementation components may be affected in ADHD. However, it is possible that these two components may be separately linked to different symptoms of ADHD and dyslexia. Such time series analyses have not been used to examine motor timing performance in populations with dyslexia.

As reported above, the studies of ADHD have typically used visual (or bimodal) pacing stimuli for finger tapping tasks whereas studies of motor timing and dyslexia have used auditory pacing stimuli. The reason for this difference is likely because the main diagnostic features of dyslexia are in the phonological and language domains, with links made to the acoustic processing of speech stimuli. In contrast, visual stimuli are likely similar to those used in ADHD assessment paradigm (such as measures of inhibition or executive functioning). One of the studies of ADHD reports using bimodal auditory-visual stimuli to maintain the children's interest in the motor timing paradigm (Ben-Pazi et al., 2003).

In motor timing tasks auditory and visual stimuli are processed differently (Fendrich & Corballis, 2001) and these differences affect the motor synchronisation response (Kolers & Brewster, 1985; Repp & Penel, 2002; and see Chapter 2). As such, visual pacing stimuli typically cause greater response variability than auditory or bimodal stimuli and auditory stimuli dominate in the temporal domain when paired with visual stimuli (Repp & Penel, 2002). Therefore, it may be that by using tasks in different modalities, previous studies of dyslexia and ADHD measured different aspects of the timing mechanism and it is unclear whether the deficits seen in ADHD and dyslexia result from similar underlying problems.

3.5. Research aims and questions

In the preceding chapters I have described how motor timing tasks provide a unique window on temporal processing, being governed by automatic mechanisms of temporal control that are independent of higher cognitive processes such as decision making or memory. In previous studies

of motor timing in dyslexia, increased variability and longer anticipation times have been found but there is insufficient awareness of where in the motor timing control system these difficulties may arise. The aims of this thesis are therefore to advance the understanding of these motor timing deficits using methodologies drawn from the motor timing research field. As described in Chapter 2, the application of time series analysis models can help to discern the components of temporal processing that might be disrupted.

Stochastic time series models (such as the Wing-Kristofferson model) have been successfully applied in other populations of participants who also experience disruption of timing behaviour, such as patients with ADHD, Parkinson's disease, Huntington's disease and damage to the cerebellum (Freeman et al., 1996; Harrington et al., 1998; Ivry & Keele, 1989; O'Boyle et al., 1996; Valera et al., 2010; Zelaznik et al., 2012). Typically these models have been applied to short runs of thirty finger tap responses that are similar to the data collected in the studies of motor timing and dyslexia. Such datasets are preferable when studying children to the larger datasets (of many thousands of responses) required for dynamic systems modelling tools that have rarely been applied to clinical populations. While these dynamic models have the capacity to describe some kinds of long term variability, they may also miss the valuable information that can be gained from data from unpractised participants completing timing tasks for the first time (Madison, 2001b); the very kind of data in which the motor timing deficits in dyslexia are typically recorded. The Wing-Kristofferson model itself allows the typically measured IRI variability to be further decomposed into variance components that reflect putative underlying mechanisms, namely timekeeper and implementation variance (Vorberg & Wing, 1996; Wing & Kristofferson, 1973b). By separating these variance components in patient populations there is evidence for which brain areas contribute to the different components.

The aim of this thesis is therefore to apply the Wing-Kristofferson model of time series analysis to motor timing data from children with varying abilities in reading and attention. The use of the model will help reveal the independent and shared components of timing that are associated with the core symptomatic features of dyslexia and ADHD. If the temporal processing differences found in these developmental disorders have a shared causal mechanism it is expected that the efficiency of the timekeeper or implementation mechanisms will be associated with both literacy and attention variables. In contrast, if the shared difficulties result from different mechanisms these variables should not share common associations with literacy and attention related skills within the same participants.

Given the apparent overlap between motor timing deficits in ADHD and dyslexia, but the inconsistencies in the use of different pacing stimulus modalities, the first study (Chapter 6) addresses the differences between task modalities. As such the study examines the effect of

auditory, visual and bimodal stimuli on timing performance and investigates whether timing performance under each stimulus modality is equally related to symptoms of dyslexia and ADHD. The second experimental study (Chapter 8) examines whether children with dyslexia show differences in both timekeeper and implementation components of timing variability in comparison to control participants. In addition the associations between these different components and symptoms of dyslexia and ADHD are investigated to again examine which components of timing are associated with the symptoms of the disorders. The experiments in Chapter 9 were planned following the completion of the previous experiments and build on the previous results by examining another component of motor timing (namely, error correction).

3.6. Thesis Chapter Structure

The experimental and time series data analysis methods are described in Chapters 4 and 5 respectively. The methods chapters are followed by four experimental chapters examining: the effect of stimulus modality on motor timing behaviour (Chapter 6), the validity of the time series analysis model (Chapter 7), the characteristics of motor timing associated with reading difficulties (Chapter 8), and an assessment of the contribution of error correction to motor timing difficulties (Chapter 9). Chapter 10 provides a discussion of the results in the context of broader theory and suggestions for future research.

4. Methods

4.1. Chapter overview

This chapter provides an overview of the ethical considerations applicable to all the studies in the thesis and the experimental methods implemented in experimental Chapters 6 and 8 of the thesis. The rationale for the studies in Chapters 7 and 9 are described within those Chapters, with Chapter 9 having been developed following the completion of the experiments in Chapters 6, 7 and 8. Chapter 5 provides a separate description of time-series decomposition method applied across the studies.

4.2. Timeline

The studies in Chapters 6, 7 and 8 were planned during year one of the doctorate and data for these studies was collected concurrently during the latter part of year one and during year two. Clinical participants for the study in Chapter 8 were recruited over this entire period, whenever children with reading difficulties within the target age range presented for a clinical assessment. Following the completion of these studies, the rationale and methodology for the final experimental study (Chapter 9) was developed. This final study was planned and completed during the third year of the doctorate.

4.3. Ethical considerations

The procedures in these studies were approved by the Institutional Review Board at Aston University. The main ethical issues considered are described below.

4.3.1. Informed consent and right to withdraw

All participants were given ample opportunity prior to participation to ensure they understood the nature of the study and their rights and responsibilities as a participant. Adult participants were recruited via campus advertisements and an online research participation scheme for first year undergraduates. Adults received verbal and written descriptions of the study and provided their written consent. For the three school samples (in Chapters 6, 8 and 9) head teachers were initially approached for their agreement to participate. Opt-out letters were then sent to parents/guardians to inform them of the purpose of the study; these were returned if they did not want their son/daughter to participate. The nature of the study, details about the confidentiality of data and their right to withdraw were also explained verbally to each child at the start of the first session using age-appropriate language.

The clinical participants in Chapter 8 were recruited from the Dyslexia and Developmental Assessment Unit at Aston University which they were attending for a psychological assessment for

dyslexia with a Psychologist (the assessments typically take 2-3 hours to complete). Prior to attending an assessment at the unit, parents are informed by letter that they may, on occasion, be approached to participate in studies whilst attending for assessments. On the day of an assessment the Chartered Psychologist would ask whether they would be willing to discuss participation in a study with a researcher. If in agreement the author would then discuss the nature of the study with the parent and child using appropriate language, highlighting that: participation in the study did not have any impact on their present or future assessments at the clinic; they could decide at any time to leave the study; and about the confidentiality measures. Participants and parents were informed that, to avoid repeating tests, data would also be collected from their clinical assessment results. Children were given an information booklet about the study, and parents were given all the information and consent documents to take away and read during the course of the morning while their son/daughter was having the assessment. After the clinical assessment and subsequent lunch break the parent and child were again approached to find out whether they would still like to take part. On occasion, the child was too tired to complete further tasks and did not participate. If they were happy to proceed, the consent documents were returned and the child completed the experiment whilst their parent(s) discussed the assessment results with the clinician.

4.3.2. Privacy and confidentiality

Any records (computerised or paper) were anonymised via the use of participant numbers so that participants could not be identified from their data and data was stored securely. Where names were recorded these were stored separately from the data.

4.3.3. Physical and psychological harm

The finger tapping tasks involved small repeated physical movements which could become tiring after some time. Testing sessions involving this task were therefore restricted to short durations, and where necessary trials were completed over more than one session. Participants were also given regular opportunities for breaks during these physical tasks, some of which were enforced through the computer programme to ensure that rest periods were taken. A concern in the study in Chapter 6 examining the influence of stimulus modality was that the rapidly changing visual stimuli could trigger epileptic seizures. Participants were therefore screened for the presence of epilepsy prior to taking part. The other standardised psychological tests that were used to assess the performance of the participants (e.g. reasoning, literacy and attention measures) involved graded stimuli so that after a number of incorrect responses the task was discontinued, preventing participants becoming disheartened with their performance. Participants were all debriefed again regarding the nature of each study following their participation either verbally and/or by letter. For the children this was done in age-appropriate language.

4.3.4. Respect

Some groups were selected on the basis of age or due to their attendance at a clinic due to the presence of learning difficulties. The nature of the study was explained to all participants so that they were aware of the contribution their participation would make to research and the necessity of targeted recruitment of particular groups. Where requested, participants were written to following the studies to update them with an overview of the results which helped to ensure that participants were involved with the research process. Results were also publicised at science related public interest events and conferences.

4.4. Methodology for Chapter 6

4.4.1. Overview of research questions

Previous evidence from timing studies in adults suggests that timing variability is greater in motor tasks when pacing stimuli are presented visually rather than acoustically. Despite this difference, the pacing stimuli have not been consistently applied across investigations of motor timing in developmental disorders, with auditory stimuli being common in studies examining dyslexia and visual or bimodal stimuli being used in studies of ADHD. The aim of the studies in Chapter 6 was to evaluate the effect of these different stimulus modalities on motor timing in adults (Experiment 1) and children (Experiment 2). Performance on the motor timing finger tapping task was measured using simple accuracy and variability measures and the variance in responses was also decomposed using time series analyses (described in Chapters 2 and 5). The relationships between task performance and measures of literacy and attention were also assessed to determine whether timing under different modalities is particularly associated with either of these dimensions. The measures used are described in detail below.

4.4.2. Participant selection

For Experiment 1 an adult sample was recruited from the staff and student populations at the University in order to replicate the earlier evidence that adults show differences in motor timing under auditory, visual or bimodal conditions. In addition, this sample was recruited to allow rapid collection of a sample of time series data in order that the author could learn to apply and understand the time series decomposition model. Evidence suggests that motor timing is relatively stable in adults even into late adulthood (Drewing, Aschersleben, & Li, 2006; McAuley et al., 2006; Vanneste et al., 2001) and so targeting a particular age group was not essential and the final sample included participants from 18-40 years old. Some of the adults taking part in this experiment also took part in the study that forms Chapter 7 of the thesis, with at least two weeks break between their participation in each study.

The effect of the pacing stimulus modality on motor timing performance in children has rarely been studied. With the widespread application of different stimulus modalities across developmental populations it was pertinent to demonstrate whether results of higher variability on visual, compared to auditory, tasks could be extended to children. Within the timeframe of this doctorate it was only feasible to assess these effects in a sample of typically developing school children. This was appropriate given that the characteristics of developmental disorders like dyslexia and ADHD (measured here using literacy and attention tasks) form the lower end of a continuum of ability across the population. It was therefore expected that the results from this study of typically developing children would be applicable to clinical populations.

This study aimed to allow evaluation of previous studies of motor timing in developmental disorders that have implemented varying modalities of pacing stimuli. Previous investigations of developmental disorders where group differences in timing performance have been found have typically assessed children aged 8-14 (Badian & Wolff, 1977; Ben-Pazi et al., 2003; Klipcera et al., 1981; Rubia et al., 2003; Rubia, Taylor, et al., 1999; Thomson & Goswami, 2008; Tiffin-Richards et al., 2004; Waber et al., 2000; Wolff et al., 1984; Zelaznik et al., 2012) with fewer studies examining performance in older adolescents (Toplak & Tannock, 2005b; Wolff, Michel, Ovrut, et al., 1990; Wolff, 2002). Studies which have measured motor timing performance across the lifespan indicate that up until age seven children are unable to produce accurately timed responses across a range of tapping rates (McAuley et al., 2006). Therefore, children aged 8-12 were recruited to balance the need for replication the need for children to be able to produce relatively invariable taps.

The children were recruited from a single primary school in the Derbyshire region. The school was a small rural primary (total pupils approximately 180) with the majority of pupils being from a White British background and few children claiming free school meals. In 2008 the school received an overall rating of 'Good' by OfSTED and the percentage of pupils achieving Level 4 or above in English and Maths in 2009 was 90% (Office for Standards in Education (OfSTED), 2008). The children who participated were from mixed-age classes each comprising of both Year 4 and 5 pupils.

4.4.2.1. Exclusionary criteria

Participants were required to have reasoning ability scores that fell within 1.5 standard deviations of the population mean, no existing diagnosis of psychological or neurological conditions (other than those under study here) and speak English as a first language. Such factors may in themselves contribute to different profiles of performance on the timing, literacy or attention measures (e.g. Holm, Ullén, & Madison, 2011; Madison, Forsman, Blom, Karabanov, & Ullén, 2009; Waber et al., 2000).

4.4.3. Experimental tasks

4.4.3.1. Motor timing task

A synchronisation task was chosen in which participants tap their finger in time with a beat, coupling motor responses to a sensory event with little requirement for additional cognitive processing. It was necessary to use a synchronisation task, rather than a continuation task in which participants continue tapping without the beat, because the modality of the driving stimulus was the variable of interest in this study. Whilst the synchronisation task, by design, allows for online updating of responses relative to the stimulus train, both synchronisation and continuation tasks involve a timekeeping process to generate intervals that match the stimulus intervals to be implemented by the motor system (Vorberg & Wing, 1996). The two tasks have also been found to recruit similar neural networks and result in similar timekeeper variance estimates (Jantzen et al., 2004) particularly at the tapping rate (329ms IOIs; approximating 3Hz) considered in this investigation (Semjen et al., 2000).

To assess the influence of modality, auditory, visual and bimodal stimuli were presented in separate trials. The stimuli were isochronous and participants were required to tap their finger in time with the stimuli. Responses were made via finger taps, replicating the methodology of studies assessing motor timing performance in the general population (e.g. Banerjee, Tognoli, Kelso, & Jirsa, 2012; Jancke, Loose, Lutz, Specht, & Shah, 2000; Madison, Forsman, Blom, Karabanov, & Ullén, 2009; Turgeon, Wing, & Taylor, 2011; Wing & Kristofferson, 1973a; Wing, 1977) and in children with developmental disorders (e.g. Ben-Pazi, Shalev, Gross-Tsur, & Bergman, 2006; Thomson & Goswami, 2008; Waber et al., 2000; Wolff et al., 1984; Wolff, Michel, Ovrut, & Drake, 1990; Wolff, 2002). Participant responses were recorded using a PalPad switch plate (Inclusive Technologies) which had minimal travel when pressed, reducing the duration of the tactile/auditory feedback from the tap, in comparison to a mouse or button press.

For this study, the switch plate was housed within a covered box so that participants were not able to gain visual feedback from their hand movement during any of the trials. Without such controls, participants are able to look at their hands during the auditory task where visual attention to stimuli is not required, providing a greater amount of feedback than in the visual trials where participants are required to look at the screen to follow stimuli. This methodological problem has been highlighted in previous investigations of this type (Loras et al., 2012).

The tapping rate of 3Hz (329ms IOIs) was selected because this rate has been used in studies where visual or bimodal stimuli have been used with developmental populations (Ben-Pazi et al., 2003, 2006; Pitcher, Piek, & Barrett, 2002; see Appendix for conversions between Hertz and IOIs). The present study will therefore allow an evaluation of the validity of results from such studies that have used these stimulus parameters. The auditory stimuli comprised auditory tones and the visual

stimuli, a 2cm diameter red diamond presented on a black background. Auditory tones have typically been used in studies of motor timing in developmental and adult populations (Elliott et al., 2010; Jäncke, Loose, et al., 2000; McAuley et al., 2006; Repp & Penel, 2002; Thomson et al., 2006; Thomson & Goswami, 2008; Tiffin-Richards et al., 2004; Wing & Kristofferson, 1973b). A variety of visual stimuli have been used for visually paced tasks including complex pictures with verbal labels like the aeroplane implemented by Rubia and colleagues (Rubia et al., 2001, 2003) or simple stimuli such as the diamond used by Ben-Pazi et al. with children with ADHD (Ben-Pazi et al., 2003, 2006) and the LEDs used in several studies examining stimulus modality (Aschersleben & Prinz, 1995; Kolers & Brewster, 1985; Patel et al., 2005). The diamond stimulus used here was chosen to replicate the more simple visual stimuli and the image used was red to enable relatively fast conductance through the visual system. All stimuli were presented via E-prime stimulus presentation software (E-Prime 2.0; Schneider, Eschman, & Zuccolotto, 2002) with onsets timed and corrected for the screen refresh-rate to achieve precision of presentation of ±1ms. All stimuli and responses were measured with respect to the onset time.

The number of finger tap responses recorded in each trial was governed by a need to minimise participation time, participant discomfort and to gain sufficient data to allow analysis of the time series. Previous investigations of motor timing in children have demonstrated differences in timing variability amongst groups over series of 20-50 finger taps (Ben-Pazi et al., 2006; Thomson & Goswami, 2008; Waber et al., 2000; Wolff et al., 1984) although some authors did not specify the number of taps per trial (Wolff, 2002). Other groups used as few as 8-15 taps (Overy et al., 2003; Tiffin-Richards et al., 2004) and failed to demonstrate any significant group differences in motor timing performance. Such small datasets are likely to be insufficient for the application of time series analyses required presently.

The original data on which the Wing-Kristofferson model were based was drawn from repeated trials comprising 31 finger taps in order to estimate variance over 30 intervals (Wing & Kristofferson, 1973a, 1973b). Harrington, Haaland and Hermanowicz (1998), who applied the same decomposition method to data from patients with Parkinson's disease, only assessed 20 taps whilst most other studies using this analyses method have measured performance over 31 taps (Carroll et al., 2009; Freeman et al., 1996; Ivry & Keele, 1989; McAuley et al., 2006; Vanneste et al., 2001). Simulation studies indicate that the standard error in the time series model can be reduced by increasing the sample size i.e. the number of finger tap responses collected to 100 or greater (Collier & Ogden, 2004). However, for unpractised participants, particularly children, maintaining a tapping response for such a long period is extremely challenging. To minimise fatigue, the original method of analysing 30 response intervals was implemented. Forty synchronisation stimuli were therefore presented to ensure 30 response intervals were collected for analysis; allowing behaviour to become stabilised

over the first 5 taps and accounting for any participants who stopped tapping before the end of the stimulus train.

4.4.3.2. Simple reaction time

As a control measure, participants also completed a measure of simple reaction time to allow analysis of whether difficulties in finger tapping to isochronous sequences were related to a general slowness in motor speed. Participants made responses to stimuli identical to those used in the three modalities of the finger tapping paradigm (visual, auditory, bimodal). Participants were instructed to respond as quickly as possible with a single finger tap following presentation of a single stimulus. Responses were registered using the same switch plate described above, housed within the container to obstruct viewing. After two practice trials, participants responded to ten reaction time trials within each of the three modalities. Responses occurring less than 150ms or greater than 750ms after the stimulus onset and double tap responses were excluded from the dataset; these typically resulted from participants anticipating the stimulus and responding early. Mean reaction time and SD were calculated for each modality condition.

4.4.3.3. Additional tasks

In addition to the primary focus on stimulus modalities in motor timing, the study also aimed to examine whether timing under these stimulus conditions was related to the psychometric measures that are typically utilised in the assessment of the key indicators of dyslexia and ADHD. The measures included for this purpose are described in Section 4.6 below.

4.5. Methodology for Chapter 8

4.5.1. Overview of research questions

The aim of the study in Chapter 8 was to replicate evidence that children with reading difficulties have motor timing deficits and to apply a time series analysis model to the motor timing data to assess whether differences in timing can be attributed to particular parts of the timing mechanism. The study also examined relationships between the statistically decomposed components of motor timing performance and psychometric measures typically used in the assessment of dyslexia and ADHD.

4.5.2. Participant matching

In this study both reading level (RL) and chronological age (CA) matched controls were used. This type of matched design compares children with reading difficulties to both their same age peers and younger typical readers who are at the same reading level as the children with reading difficulties, but at a lower developmental level. On a particular task, if no difference is found between the CA matched group and the children with reading difficulties then it can be concluded that the task is not

related to reading ability. The inclusion of the RL group can establish whether differences in poor readers are merely due to the reduced level of reading experience in the poor readers (Backman, Mamen, & Ferguson, 1984). Where children with reading difficulties show a difference in performance from both the CA and RL controls it suggests that the reading difficulties are associated with a difference in development or in the rate of development of the variable of interest (Backman et al., 1984; Bryant & Goswami, 1986). Circumstances where no differences are found between the RL matched group and the poor readers are more difficult to interpret. Such a result cannot confirm that the variable of interest is not related to reading because the additional development and reading experience in the older poor reader group may have given rise to superior performance masking any group difference (Goswami & Bryant, 1989).

In light of these different conditions, the design does not necessarily permit conclusions to be drawn about any potential causal routes between motor timing and reading which may be better analysed through longitudinal designs (Bryant & Goswami, 1986; Goswami & Bryant, 1989). However, the combination of RL and CA matched groups is powerful for examining variables associated with reading development and as applied here will help establish the usefulness of the statistical decomposition method in assessing motor timing in children with reading difficulties providing the limitations are recognised (Mamen, Ferguson, & Backman, 1986; Olson, Wise, Conners, & Rack, 1990).

To ensure equivalence between the groups participants were matched on word reading scores and the appropriateness of matching was verified with other literacy measures. Because IQ can have an effect on the development of a range of skills, verbal and non-verbal reasoning were measured to establish group similarity. The full details of the age matching are described in within Chapter 8.

4.5.3. Participant selection

One of the aims of this study was to replicate previous findings of poorer motor timing performance in children with reading difficulties. As described above, previous investigations have most commonly investigated children aged 8-14 (Badian & Wolff, 1977; Ben-Pazi et al., 2003; Klipcera et al., 1981; Rubia et al., 2003; Rubia, Taylor, et al., 1999; Thomson & Goswami, 2008; Tiffin-Richards et al., 2004; Waber et al., 2000; Wolff et al., 1984; Zelaznik et al., 2012), with motor timing performance generally being stable by the age of eight (McAuley et al., 2006). Therefore sampling targeted children aged 8-12. The group with reading difficulties were sampled opportunistically from the University's dyslexia assessment clinic. The clinical sample were recruited either at the time of attending the clinic for a literacy assessment with an Educational Psychologist (as described in Section 4.3.1) or by letter, having previously attended the clinic for an assessment. The children received a small monetary sum for their participation and any additional travel costs were reimbursed.

The control groups comprised different children from those in recruited for Chapter 6 and were recruited from two primary schools from towns in the Derbyshire region. The first had a total of around 270 pupils, with the majority of pupils being from a White British background and few children claiming free school meals. The second school had around 180 pupils in total and a similar distribution of ethnic groups with a minority having a degree of economic disadvantage. In 2008 the schools received overall OfSTED ratings of 'Outstanding' and 'Good', respectively (Office for Standards in Education (OfSTED), 2009) with 89% and 77% of pupils achieving Level 4 or above in English and Maths. The children who participated were sampled from primary years four and six. Exclusionary criteria were the same as those in the previous study (see 4.4.2.1).

4.5.4. Experimental tasks

4.5.4.1. Motor timing task

The time series data collected in this study were to be analysed using the Wing-Kristofferson model of motor timing behaviour. Therefore to replicate the methodology used in the original derivation of this model described by Wing and Kristofferson (1973a, 1973b), a synchronise-and-continue finger tapping task with an auditory pacing stimulus was employed in which participants first synchronise to the beat and then continue tapping once the beat has ceased. Time was provided in the latter phase to ensure at least 30 taps were collected in order to perform the time series analysis. The unpaced continuation phase allows measurement of internal timing in the absence of any external influences from synchronisation stimuli. Participant responses were recorded using the PalPad switch plate already described (Inclusive Technologies). The housing described in Section 4.4.3.1 to obscure viewing was not required in this experiment which only focused on acoustically paced timing.

To assess whether the relationships between motor timing and cognitive predictors of reading and attention were present across a range of tapping speeds, performance was assessed across five different response rates. A pilot study had indicated that children struggled to maintain a tapping response with inter-stimulus intervals smaller than 250ms (4Hz). Studies assessing timing performance in children with ADHD have included faster tapping rates than in the dyslexia research and results indicate that performance variability across participants increases dramatically with stimulus intervals of 200 or 166ms (5 or 6Hz) (Ben-Pazi et al., 2003, 2006). The maximum tapping rate was therefore set at 3.5Hz (285ms intervals) and the slower speeds tested were comparable to those used in some of the recent demonstrations of how timing relates to literacy ability (Thomson et al., 2006; Thomson & Goswami, 2008; Wolff, 2002). The final rates included were equivalent to 1.5, 2, 2.5, 3 and 3.5Hz (a conversion between rates and IOIs is provided in the Appendix). These rates fall within the millisecond timing range, as described in Chapter 2, and have been assessed widely using the Wing-Kristofferson model of time series analysis (e.g. Elliott et al., 2010; Harrington

et al., 1998; Ivry & Keele, 1989; Vanneste et al., 2001; Wing & Beek, 2002; Wing & Kristofferson, 1973a, 1973b).

4.5.4.2. Simple reaction time

Participants also completed a simple reaction time task to control for the effect of general motor speed on motor timing performance. The task was identical to that described in Section 4.4.3.2, except that only auditory trials were included (given that this experiment only assessed acoustically paced motor timing).

4.5.4.3. Additional tasks

To establish the relationships between timing performance and performance on cognitive/behavioural measures that are typically used in the assessment of dyslexia and ADHD participants were tested participants on a range of additional measures described below.

4.6. Cognitive and behavioural measures

In the studies in Chapters 6 (adults and children) and 8 (children only), participants completed a range of measures that assessed performance in areas related to literacy and attention abilities in order to assess the associations of these measures with timing performance.

4.6.1. Reasoning ability

Measures of verbal and non-verbal reasoning were employed to ensure participants were of average ability with no severe learning problems and because reasoning ability has been found to influence motor timing performance (Holm et al., 2011; Madison et al., 2009; McAuley et al., 2006) and mediate the relationship between motor timing and measures of literacy (Thomson & Goswami, 2008; Waber et al., 2000). The Similarities (verbal reasoning) and Matrices (non-verbal reasoning) subscales from the Wechsler Abbreviated Scale of Intelligence (WASI; Wechsler, 1999) were administered to both the adult and child participants. The full scale WASI comprises four subtests, two performance and two verbal reasoning measures and a two-scale IQ score can be calculated from the Vocabulary and Matrices subtests. The Similarities subtest of the WASI was used to measure verbal reasoning rather than the Vocabulary test because the Similarities test takes less time to administer (Axelrod, 2002) yet correlates highly with the Vocabulary measure (0.71 for children and 0.79 for adults; Wechsler, 1999). Both the Similarities and Matrices subscale scores also correlate strongly with the four-scale IQ scores (Saklofske, Caravan, & Schwartz, 2000). It was important to select measures which could be administered in a short amount of time but would yield appropriate estimates of reasoning ability because these measures were to be administered alongside a number of other measures in each of the studies.

The WASI has age-referenced norms for adults and children allowing the same measure to be used across the samples recruited in the present series of studies. The normative sample for the WASI is based on an English speaking population sample from the United States with characteristics matching the 1997 US census data. The WASI measures show substantial correlations with similar measures in other long-form IQ batteries such as the WISC-III and WAIS-III (ranging from 0.66 to 0.76 for the selected subtests) (Wechsler, 1999).

The group of children with reading difficulties described in Chapter 8 were drawn from a dyslexia assessment clinic and had already undergone a full psychological assessment, which included the WISC-IV (Wechsler, 2003), therefore re-administration of reasoning measures was inappropriate. For these cases the Verbal Comprehension and Perceptual Reasoning Indices (VCI and PRI) were used as comparable measures of verbal and non-verbal reasoning. The validity testing conducted in the standardisation of the WISC-IV indicates that these indices are comparable to those in the WASI, with PRI correlating with WASI PIQ (r=0.78) and VCI with WASI VIQ (r=0.85) (Wechsler, 2003).

4.6.2. Literacy

The Wechsler Individual Achievement Test (WIAT-II) Word Reading and Spelling subtests were used to examine literacy skills, providing measures of word recognition (both regular and irregular) and spelling knowledge across both adults and children (Wechsler, 2005). These tests have age-referenced normative data for both adults and children based on large US standardisation samples. The tests show good criterion validity and correlate well with other achievement based tests such as the Wide Range Achievement Tests, as well as showing high test-retest reliability (Lichtenberger & Smith, 2005; Wechsler, 2005).

4.6.3. Literacy component skills

Measures of phonological and orthographic processing skills were employed to assess the different components which contribute to reading ability and may be associated with motor timing performance. Tests of phonological skills assess reading skills reliant on sound based phonological decoding, and orthographic tests assess the ability to map these codes onto the letter based word representations. Most commonly, motor timing performance has been found to be predictive of phonological decoding performance (Thomson et al., 2006; Thomson & Goswami, 2008; Wolff, 2002), however, studies of other forms of auditory temporal processing, such as frequency discrimination are related to measures of both orthographic and phonological sensitivity (Talcott et al., 2002). In comparison, Talcott et al. (2000) found that orthographic sensitivity may be more strongly associated with processing of dynamic stimuli in the visual domain and phonological sensitivity to stimuli in the auditory domain. More recently however, Talcott, Witton and Stein (2013) found that only phonological performance was indicative of atypical development and was uniquely predictive of auditory sensory processing. In that study, orthographic sensitivity was only related to delayed

development. Given that both visual and auditory stimuli of a temporal nature were to be administered in the studies presented in the thesis, measures of both orthographic and phonological processing were included in the test batteries.

The orthographic and phonological tests completed by the adult group in Chapter 6 were computerised forced choice tasks (Olson, Forsberg, Wise, & Rack, 1994). The orthographic measure comprised a two-alternative forced choice (2-AFC) word-pseudoword-homophone discrimination task with eight practice and 80 test trials. Pairs of stimuli, comprising a correctly spelled word (e.g. rain) and an incorrectly spelled foil (e.g. rane), were presented side-by-side on a computer screen and participants were instructed to identify the correctly spelled word on each trial by pressing the appropriate button on a keypad. The phonological choice measure was similar in design with three word choices (3-AFC), and comprised 4 practice trials and 60 test trials. Of the three items in each trial one item was a homophone of a real word (e.g. eer) and two items were foils with pronunciations that did not correspond with any English word (e.g. eem and eep). Participants were instructed to identify the item that sounded like a real word via a key press. For both choice tests, responses and response times were recorded and feedback was provided to participants following each response. Percent correct and average response times were calculated. A principle components analysis was used to combine the total score and response times into a time-error composite measure for each test.

During earlier pilot studies children had found these computerised choice tasks rather onerous and often requested to end the task before all the items were complete. Paper-based orthographic and phonological tasks described by Castles and Coltheart (1993) were instead used. These comprised two printed lists of 30 non-words and 30 irregular-words to be read under timed conditions. The non-word task measures the ability to decode novel words (e.g. torlep) and the irregular word task assesses the ability to read words with spellings that deviate from the typical letter-sound correspondence rules of English (e.g. yacht). Participants were instructed not to sacrifice accuracy for speed while reading the lists. The number of correctly pronounced words and the total reading time for each list was recorded. For each test, a principle components analysis was used to produce a composite score combining the total score and time taken.

Following proposals that speed of processing mediates the relationship between temporal processing and reading ability (Breznitz, 2006), or the overlap between dyslexia and ADHD (McGrath et al., 2011), children were also tested on a rapid naming task. The Rapid Object Naming test from the Comprehensive Test of Phonological Processing (CTOPP) was selected, which has age-referenced norms derived from a large US sample of children aged 5-17 (Torgeson, Wagner, & Rashotte, 1999). In this task participants are required to name aloud a pseudo-randomly arranged matrix of 36

pictures (of 6 different objects). The time taken to complete the two test matrices was summed and converted to an age-referenced standard score.

4.6.4. Attentional measures

4.6.4.1. ADHD symptom ratings

To assess the contribution of ADHD related symptoms to motor timing behaviour a symptom scale was sought. A number of rating scales that assess similar symptom items are available including the Barkley Self-Report (Barkley & Murphy, 1998), the Child Behaviour Checklist (CBCL) and comparable Adult Self Report from the Achenbach System of Empirically Based Assessment (ASEBA; Achenbach & Rescorla, 2001, 2003), and the Conners Rating Scales (Conners, Sitarenios, Parker, & Epstein, 1998). The Barkley Self-Report Forms were chosen because they are relatively short scales, which efficiently record the presence of ADHD symptoms with equivalent forms for adults and children (rated by parents and teachers) that could be used across the studies in the thesis. Each form includes two nine item subscales for respondents to rate symptoms of inattention (ADHD-IA) and hyperactivity-impulsivity (ADHD-HI) occurring over the last six months thus capturing the behavioural dimensions of ADHD included in the Diagnostic and Statistical Manual of Mental Disorders IV (DSM-IV; American Psychiatric Association, 2000). These scales include more symptom items relevant to ADHD than other short-form scales (such as the Connors Rating Scales) and do not incorporate symptom dimensions that were not relevant to the present studies, such as the depression items found on the CBCL and adult ASEBA scales.

4.6.4.2. Attention related processing measures

In addition to the self-report scales, participants completed objective measures of attentional abilities. Adults completed a 112 item colour-word Stroop task and equivalent word-free version (with XXXX's matched in word length to colour names) which assesses behavioural inhibition. The total time taken to complete each version was recorded and the percent increase in time taken for the colour version compared to the matched version was calculated. Adults with ADHD show reduced interference control on these tasks (Lansbergen, Kenemans, & van Engeland, 2007).

For the assessment of attention related skills in children, measures from the Test of Everyday Attention for Children (TEA-Ch) (Manly, Robertson, Anderson, & Nimmo-Smith, 1999) were chosen which have been employed elsewhere to assess attention in children with dyslexia (Snowling, Muter, & Carroll, 2007). The tests from this battery have age-reference norms drawn from a standardisation sample of 293 Australian children.

The Same World/Opposite World test was used to assess attentional control and inhibition. A factor analysis conducted with data from the standardisation sample indicated that this test loads highly on an attentional control/switching factor (Manly et al., 1999). In the Same World task the child follows a printed path in the stimulus booklet naming the digits '1' and '2' along the path. In the Opposite

World task the procedure is repeated, but children must say “two” where they see a ‘1’ and “one” for ‘2’s, inhibiting the automatic verbal response. Each subtest was completed twice and the total time taken to complete each subtest was recorded and converted to a standard score using the published norms.

The Score! measure from the TEA-Ch battery was also used to assess sustained attention. This is a child-friendly sustained attention task similar to the vigilance tasks used to assess sustained attention in adult samples (Manly et al., 1999; Robertson, Manly, Andrade, Baddeley, & Yiend, 1997; Wilkins, Shallice, & McCarthy, 1987). Children are asked to keep score in each of 10 hypothetical “games” played to them via computer speakers. Each game comprises a start and stop signal and intervening “goals” (resembling space noises). The number of correctly scored games was converted to a standard score using the TEA-Ch norms. The factor analysis performed as part of the TEA-Ch standardisation indicated that the Score! task loads as highly on a sustained attention factor as other continuous performance tasks. In addition, a sample of boys with ADHD scored significantly lower than control children drawn from the standardisation sample on both the Score! and Same World/Opposite World measures (Manly et al., 1999).

4.7. Analysis methods

Unless indicated, comparisons between groups and conditions were made using analyses of variance (ANOVA) which are relatively robust to the effects of outliers and non-linearity. Univariate outliers were identified as cases with z-scores of residuals exceeding 1.96 (falling outside the 97.5% confidence intervals). Where appropriate, outliers were corrected by substitution with the value of the next largest or smallest score, plus or minus one. ANOVA solutions were checked in the presence and absence of outlying cases to ensure they did not affect the outcome. Such analyses are not reported in the studies unless a difference in the outcome was found, in which case the amended analysis is also described. Mauchly’s test of sphericity was used to assess whether the variance of the differences between pairs of cases was equal. Where equal variances could not be assumed, the Greenhouse-Geisser correction was implemented which adjusts the degrees of freedom to provide a more accurate significance value. Unless described, the assumption of sphericity was satisfied.

Where associations between variables were examined, Pearson’s correlation and multiple regression analyses were utilized. For these analyses outliers have a greater potential to distort relationships between variables. Outliers were therefore identified and corrected as described above and then the distributions of cases were assessed for skew and kurtosis. Typically the correction of outliers reduced any skew, but where necessary variables were further transformed to improve normality using square root or log transforms as recommended by Tabachnick & Fidell (2001).

The accepted alpha level was 0.05 unless specified and effect sizes (η^2) are reported to give an indication of the variance in the dependent variable accounted for by the independent variable.

5. Time series analysis method

5.1. Chapter overview

Timing performance has been investigated in detail in other clinical populations by applying the time series model described by Wing and Kristofferson (1973a, 1973b). As described in Chapter 3, the studies in this thesis aim to apply this same decomposition model (a) examine the effects of stimulus modality on timing performance (Chapter 6) and (b) compare timing performance in good and poor readers (Chapter 8). Here, the time series analysis method is described in greater detail.

5.2. The Wing-Kristofferson model

The model proposed by Wing and Kristofferson (1973a, 1973b) assumes that variability in motor timing performance is derived from two sub-systems: a timekeeping mechanism responsible for timed outputs and a peripheral implementation system which gives rise to delays as responses are executed through the motor system. When Wing and Kristofferson recorded the inter-response intervals produced when adults made finger movements in time to an isochronous beat they observed that the intervals varied in length around the target interval duration and that there was a negative correlation between adjacent response intervals. These factors were attributed to the action of the two independent systems. The timekeeper (or clock) system represents a relatively invariant mechanism capable of producing regular outputs. In contrast, the implementation (or motor) delays vary with each response due to transmission of motor commands to muscular effectors. This architecture means that every response interval that is produced is the sum of a single timekeeper interval (T) and two implementation delays (P) as shown in Figure 1. and the jth inter-response interval (I_j) can be represented as:

$$I_j = T_j - P_{j-1} + P_j \qquad j = \ldots,-1,0,+1,\ldots \qquad \textit{(Equation 1)}$$

It is assumed that the timekeeper and implementation components are independent, random variables with means (μ_T and μ_P) and variances (σ_T^2 and σ_P^2). As each implementation delay has an effect on two intervals (the one preceding it and the one following it; Figure 5.1), fluctuations in the implementation system can give rise to longer or shorter intervals. Delays that are equal in size but opposite in direction can cancel each other out. This generates a dependency between adjacent intervals (the negative correlation observed by Wing and Kristofferson) yet assumes independence for non-adjacent intervals. Within the model, fluctuations in the timekeeper can also act to reduce variation around the mean.

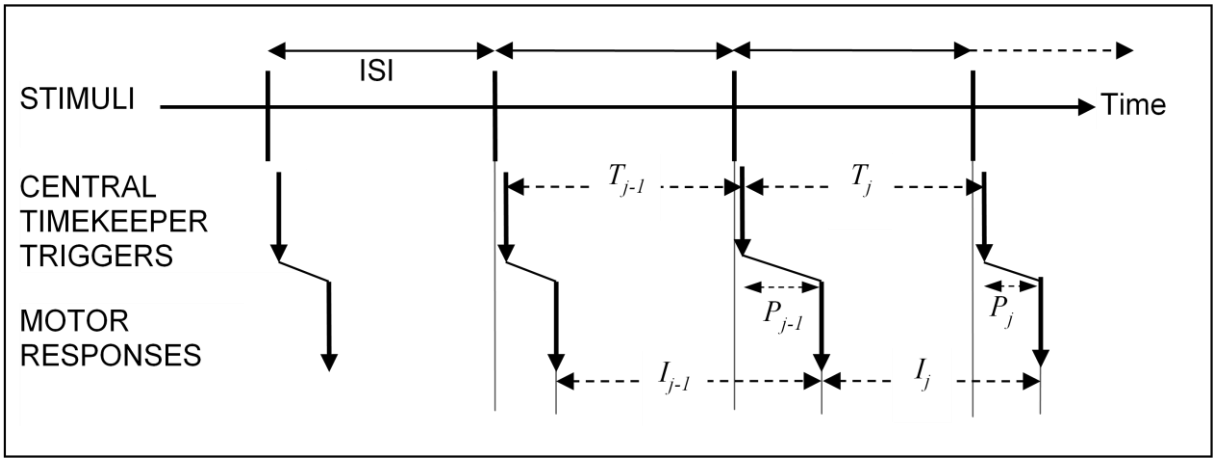

Figure 5.1: Wing-Kristofferson model of timekeeping

A schematic representation of the timekeeping system in which the timekeeper generates triggers with intervals (T_j) that are subject to a different peripheral implementation delay for each response (P_j) and result in the recorded inter-response intervals (I_j).

Whilst T and P are hypothetical components, estimates for timekeeper and implementation variance can be derived from the dependencies between adjacent intervals in observed data. Common covariance functions can be used to demonstrate that implementation variance is equal to lag-one covariance.

For clarity, lag-one covariance ($\gamma_I(1)$) is the extent to which intervals vary with the adjacent (lag-one) interval:

$$\gamma_I(1) = \frac{\sum_{j=2}^{N}(I_j - \mu_I)(I_{j-1} - \mu_I)}{N-1} \qquad \textit{(Equation 2)}$$

Where μ_I is the mean of the inter-response intervals:

$$\mu_I = \frac{\sum_{j=1}^{N} I_j}{N}$$

Substitution into Equation 1 shows that lag-one covariance is equal to negative implementation variance. Because the model assumes that the timekeeper variance is constant the timekeeper terms cancel each other out.

$$\begin{aligned}
&\gamma_I(1) = E\left[(I_j - \mu_I)(I_{j-1} - \mu_I)\right] \\
&= E\left[(T_j - P_{j-1} + P_j - \mu_I)(T_{j-1} - P_{j-2} + P_{j-1} - \mu_I)\right] \\
&= E(P_{j-1} - \mu_I)(P_{j-1} - \mu_I) \\
&= -\sigma_P^2 \qquad \textit{(Equation 3)}
\end{aligned}$$

For clarity, lag-zero covariance ($\gamma_I(0)$) is the total variance of the intervals:

$$\gamma_I(0) = \frac{\sum_{j=1}^{N}\left(I_j - \mu_I\right)^2}{N} \qquad \textit{(Equation 4)}$$

So similarly, with reference to Equation 1, it can be illustrated that variance in performance is comprised of timekeeper variance and twice the variance of the implementation delays:

$$\begin{aligned}\gamma_I(0) &= E\left[\left(I_j - \mu_I\right)\left(I_j - \mu_I\right)\right] \\ &= \sigma_I^2 \\ &= E\left[\left(T_j - P_{j-1} + P_j\right)\left(T_j - P_{j-1} + P_j\right)\right] \\ &= \sigma_T^2 + 2\sigma_P^2 \end{aligned} \qquad \textit{(Equation 5)}$$

Therefore an estimate of timekeeper variance can be calculated from observed covariance by rearrangement:

$$\sigma_T^2 = \gamma_I(0) + 2\gamma_I(1) \qquad \textit{(Equation 6)}$$

This type of time series is governed by the autocorrelation function $\rho_I(k)$ which provides a summary of serial dependence between such variables:

$$\rho_I(k) = \gamma_I(k) \Big/ \gamma_I(0) \qquad k = 1,2,3,... \qquad \textit{(Equation 7)}$$

The lag-one autocorrelation ($\rho_I(1)$) is calculated as lag-one autocovariance divided by lag-zero autocovariance, and when applied to the model it can be seen that:

$$\rho_I(1) = -1 \Big/ 2 + \left(\sigma_T^2 / \sigma_P^2\right) \qquad \textit{(Equation 8)}$$

If observed data fits the model then the lag-one autocorrelation value should fall within the bounds of minus one half and zero. Substituting extreme values into Equation 8 illustrates these bounds. If timekeeper variance is very small compared to implementation variance (or is zero) then the lag-one autocorrelation tends towards -0.5. If timekeeper variance is large compared to implementation variance the autocorrelation will tend to zero (full derivations of these limits are provided by Vorberg & Wing, 1996).

5.2.1. Validity of the model

The lag-one autocovariance model described above assumes that the data arises from a stationary time series in which the variance components (σ_T^2, σ_P^2) are independent random variables. Stationarity means that the covariance function depends only on the position of responses in the sequence (the lag) and not on time (Diggle, 1990). Any drift in the size of response intervals over time, such that the mean IRI is different at the beginning of the time series compared to at the end of the time series, will increase co-variation at all positions in the sequence. Such non-stationarity can inflate the estimates of timekeeper variance, deflate estimates of implementation variance and increase overall variance in the time series. The assumption of independence between components means that implementation variance has no impact upon timekeeper variance, other than at lag-one. As such, the model is considered an open-loop model of timing behaviour, with no additional dependency between responses at lags beyond one. In other words, timing is controlled with reference to the previous response but not with reference to any responses occurring before that (at lags two or greater). Because the assumptions of stationarity and independence are related concepts (O'Boyle et al., 1996) any time series that do not fit within the bounds of the model could suggest either type of violation, or indeed both.

When data fits the model parameters variance estimates should be positive. Equally, lag-one autocovariance should fall within the limits of zero and minus a half (based on Equation 8). For lags greater than 1 the autocovariance should be zero ($\gamma(k) = 0$ for all $k > 1$). Estimates may fall outside these limits if the assumptions of stationarity or independence of variance components are not met. In addition, negative variance estimates may occur by chance due to the statistical variability inherent in variance estimates. Such variability means that the tail of the sampling distribution of variance estimates can fall below zero even in circumstances where the model predicts positive variance, such as in the Wing-Kristofferson model (Collier & Ogden, 2004; Kooistra et al., 1997). As such, negative variance estimates do not necessarily indicate that the model does not fit the data adequately.

In previous studies of motor timing with both clinical and control participants, a non-negligible number of trials have resulted in negative variance estimates (Table 5.1). Typically between 22 and 35 percent of trials result in negative estimates (Bolbecker et al., 2011; Ivry & Keele, 1989; Kooistra et al., 1997; O'Boyle et al., 1996), although two studies found more than 40% of patient estimates were negative (Duchek, Balota, & Ferraro, 1994; Harrington et al., 1998). This was proposed to be due to the severity of disease in these patients. Not all studies have identified the number of violations of the variance assumptions (Carroll et al., 2009; Simmons et al., 2009). Generally, a greater number of violating trials have been found in clinical populations than control groups. As such violations may characterise timing behaviour in populations that show differences in more

general timing measures such as IRI mean or variability and it is necessary to monitor the presence of these features within the experiments in this thesis.

Table 5.1: Percentage of trials showing negative variance estimates in studies using the Wing-Kristofferson Model

Authors	Group Description	% of total trials with negative implementation variance estimates
Kooistra et al. (1997)	Children with thyroid dysgenesis	20
	Controls	32
Duchek et al. (1994)	Patients with Parkinson's disease	20
	Patients with mild/moderate Alzheimer's dementia	46
	Patients with mild Alzheimer's dementia	10
	Healthy controls aged over 80	14
	College students	25
Freeman et al. (1996)	Patients with Huntington's disease	6
	Controls	20[a]
Harrington et al. (1998)	Patients with Parkinson's disease	44
	Controls	17
Harrington et al. (2004)	Patients with cerebellar lesions	10
	Controls	20
Freeman et al., (1996)	Patients with Parkinson's disease	18
	Controls	19
Vanneste et al. (2001)	Adults (aged 20-30 and 60-76 years)	27*
Bolbecker et al. (2011)	Adults with bipolar disorder & controls	26*
Carroll et al. (2009)	Adults with schizophrenia & controls	**
Simmons et al. (2009)	Children with maternal alcohol exposure & controls	**
Collins et al. (1998)	Typically developing adults only (model applied to eye-movement data)	16

**Percentage across both groups; **Number of cases with violations not specified; [a]Estimated based on description in study that controls had four times the number of cases as the patients.*

In previous studies of timing behaviour with clinical populations a variety of strategies have been used to deal with occurrences of violating trials. Where lag-one covariance is found to be positive (giving rise to negative implementation variance estimates), variance estimates are often corrected to zero (Harrington et al., 1998; Ivry & Keele, 1989; Kooistra et al., 1997) under the assumption that

negative implementation variance results from the variance estimates falling in the lower tail of the distribution of possible estimates. Timekeeper variance is then set to the value of the total variance (following from Equation 8). This strategy brings the data in line with the assumptions of the model without excessively inflating the calculated variance estimates and reduces the mean squared error of these estimates (Collier & Ogden, 2004; Ivry & Keele, 1989; Kooistra et al., 1997). Correcting the implementation variance estimates to zero also has the effect of eliminating most instances of negative timekeeper variance.

A more conservative strategy for dealing with violations has been to entirely eliminate trials where the lag-one autocorrelation exceeds the predicted range (of zero to minus one half). This strategy may result in the elimination of large quantities of data, especially where the performance of untrained participants is being assessed over relatively few trials due to time constraints, as in the present series of experiments. Depending on the method of averaging across the remaining trials, this method could result in performance averages being calculated from a different quantity of data for each participant. A similarly conservative method of dealing with violations of the model's assumptions is to only analyse the first trial from each participant in which they achieve a lag-one autocorrelation that falls within the bounds of zero to minus one half (Pastor et al., 1992). This method ensures that estimates from each participant are calculated from an equal sample data (a single trial) but will increase the within subject sampling error (O'Boyle et al., 1996).

Different methods of data elimination were tested by O'Boyle and colleagues in a group of patients with Parkinson's disease who were tested on and off medication (O'Boyle et al., 1996). They compared the unadjusted Wing-Kristofferson data to that adjusted through (i) eliminating all violating trials, (ii) selecting only the first non-violating trial and (iii) trials with negative variance corrected to zero. The effect of the medication for symptoms of Parkinson's was found for these participants regardless of the elimination strategy used.

A final strategy employed to make adjustments for the causes of violations of the model assumptions was tested in a study examining timing performance in patients with congenital hypothyroidism (Kooistra et al., 1997). Kooistra et al. accounted for the factors that lead to violations by (i) correcting negative variance estimates to zero, (ii) calculating exact estimators of variance to account for the limited sample of time series data collected and (iii) adding a drift parameter to account for any linear trends (non-stationarity) in the data series. In a validation study that compared the results of the Wing-Kristofferson estimates to those produced by the Kooistra method using both simulated and experimental data the adjusted model was found to yield equivalent conclusions regarding the data (Kampen & Snijders, 2002). By adequately accounting for negative variance estimates as well as the drift components, this adjusted model therefore deals with factors that are more likely to occur in participants who are unpractised at finger tapping tasks or in clinical populations. Explorations of

these models recommend that such adjustments are sought (Collier & Ogden, 2004). The adjusted model described by Kooistra was therefore implemented for the first experimental study in Chapter 6 and is described in detail below. Following the application of this model in Chapter 6, its suitability for application in time series analyses was reviewed in Chapter 7 prior to the application of such analyses in Chapter 8.

5.2.1.1. Kooistra adjusted model

The initial equation in the Kooistra model is based on the Wing- Kristofferson model Equation 1 but includes term accounting for drift β_j.

$$I_j = T_j - P_{j-1} + P_j + \beta_j \qquad \textit{(Equation 9)}$$

The drift term is calculated as the linear drift over the series of responses with a constant slope. The observed mean ($\bar{I}$), drift coefficient (β), variance ($\gamma_I(0)$) and lag-one auto covariance ($\gamma_I(1)$) are calculated as follows. The terms in these equations account for the fact that estimates are drawn from a limited (biased) sample of populations values (i.e. a sample of only 30 IRIs) such that the estimators are calculated based on the exact sampled data which differ from an infinite sample of possible data.

$$\bar{I} = \sum_{j=1}^{N} I_j$$

$$\hat{\beta} = \frac{1}{S_n} \sum_{j=1}^{N} \tilde{j}\, I_j$$

$$\gamma_I(0) = \frac{\sum_{j=1}^{N} \left(I_j - \bar{I} - \hat{\beta}\, \tilde{j} \right)^2}{n-2}$$

$$\gamma_I(1) = \frac{\sum_{j=1}^{N-1} \left(I_j - \overline{I_{(1)}} - \hat{\beta}\, \check{j} \right) \left(I_{j+1} - \overline{I_{(2)}} - \hat{\beta}\, \check{j} \right)}{n-1}$$

Where:

$$\tilde{j} = j - \frac{n+1}{2}$$

$$S_n = \sum_{j=1}^{N} \left(\tilde{j} \right)^2 = \frac{n\left(n^2-1\right)}{12}$$

$$\overline{I_{(1)}} = \frac{\sum_{j=1}^{N-1} I_j}{n-1}$$

$$\overline{I_{(2)}} = \frac{\sum_{j=2}^{N} I_j}{n-1}$$

$$\overset{\vee}{j} = j - \frac{n}{2}$$

For large sample sizes $\gamma_I(0)$ and $\gamma_I(1)$ can be shown to be equal to their theoretical quantities of ($\sigma_T^2 + 2\sigma_P^2$) and ($-\sigma_P^2$) such that the expected values of variance ($\gamma_I(0)$) and lag-one auto covariance ($\gamma_I(1)$) are given by:

$$E\left\{\bar{I}\right\} = \mu + \beta\frac{n+1}{2}$$

$$E\left\{\overset{\wedge}{\beta}\right\} = \beta$$

$$E\left\{\gamma_I(0)\right\} = \sigma_T^2 + \sigma_P^2\left(2 + \frac{3n+5}{n(n+1)}\right)$$

$$E\left\{\gamma_I(1)\right\} = \frac{1}{n-1}\left(-2 + \frac{7n-5}{n^2-1}\right)\sigma_T^2 - \left(1 - \frac{3(n+3)}{n(n-1)^2(n+1)}\right)\sigma_P^2$$

However, for smaller sample sizes the differences from these theoretical values are not negligible. Unbiased estimators for the parameters can be defined as follows, taking into account the limited sample of data (n).

$$\overset{\wedge}{\sigma}_P^2 = \frac{1}{a_{\gamma_I(1)n}b_{\gamma_I(0)n} - b_{\gamma_I(1)n}}\left(-b_{\gamma_I(1)n}\gamma_I(0) + b_{\gamma_I(0)n}\gamma_I(1)\right) \qquad \textit{(Equation 10)}$$

$$\overset{\wedge}{\sigma}_T^2 = \frac{1}{a_{\gamma_I(1)n}b_{\gamma_I(0)n} - b_{\gamma_I(1)n}}\left(a_{\gamma_I(1)n}\gamma_I(0) - \gamma_I(1)\right) \qquad \textit{(Equation 11)}$$

Where:

$$b_{\gamma_I(0)n} = 2 + \frac{3n+5}{n(n+1)}$$

$$a_{\gamma_I(1)n} = \frac{\left(-2 + \frac{7n-5}{n^2-1}\right)}{n-1}$$

$$b_{\gamma_I(1)n} = -1 + \frac{3(n+3)}{n(n-1)^2(n+1)}$$

6. Effects of stimulus modality on motor timing performance and relationships with reading and attention variables.

6.1. Chapter overview

This chapter examines whether the motor timing task parameters used in the separate studies of motor timing in dyslexia and ADHD are comparable to establish which parameters are most appropriate for use in studies of motor timing in children.

Data from the second experiment in this chapter (Section 6.4) has already been published (Birkett, E. E. & Talcott, J. B. (2012). Interval Timing in Children: Effects of Auditory and Visual Pacing Stimuli and Relationships with Reading and Attention Variables*. PLoS ONE,* 7, 8, e42820). This paper is included in the Appendix.

6.2. Introduction

As described in the introductory chapters, motor timing tasks have been used in studies of timing in both dyslexia and ADHD to help describe the temporal deficits in these developmental disorders and the extent to which they are predictive of the key symptoms associated with each of these phenotypes (i.e. reading and attention difficulties). In each case, such research is warranted in the context of contemporary theories which highlight the potential contribution of temporal processing to the aetiology or manifestation of behavioural phenotypes (Farmer & Klein, 1995; Goswami, 2011; Rubia et al., 2009; Toplak et al., 2006). Given the extent to which these two disorders co-occur and the multifactorial nature of these disorders (as described in Chapters 1 and 3), it is important to establish whether any overlap in cognitive risk factors can be linked to diagnostic overlap in the disorders. At a neurophysiological level, a risk factor related to timing functions might explain some of the cognitive and behavioural symptoms that are seen within and across disorder phenotypes in dyslexia and ADHD. Impairments in temporal processing have been identified both for ADHD and dyslexia and similarly, processing speed has recently been put forward by the Pennington group as accounting for overlap between dyslexia and ADHD (McGrath et al., 2011).

Tasks of motor timing assess the ability to synchronise movements (typically finger movements) with external pacing stimuli and are well-suited for use with children because they do not require complex subjective judgements about the nature of the stimuli presented. Participants with a history of developmental dyslexia show greater response variability on such tasks (Badian & Wolff, 1977; Klipcera et al., 1981; Thomson et al., 2006; Thomson & Goswami, 2008; Wolff et al., 1984; Wolff, Michel, Ovrut, et al., 1990; Wolff, 2002). These performance measures are sensitive to individual variation in the symptom dimensions relevant to dyslexia diagnosis, such as reading accuracy and working memory, for both clinical and control samples (Thomson et al., 2006; Thomson & Goswami, 2008; Waber et al., 2000). Similar performance decrements are reported in paediatric samples with

ADHD, with greater response variability at the individual level (Ben-Pazi et al., 2006; Rubia et al., 2001, 2003; Toplak & Tannock, 2005b) and errors in selecting the appropriate response rate compared to controls (Ben-Pazi et al., 2003, 2006). As in the case of dyslexia these motor timing differences are found to correlate with continuous measures of ADHD symptoms (Ben-Pazi et al., 2006; Pitcher et al., 2002).

Given such evidence of temporal processing deficits in ADHD and dyslexia (Farmer & Klein, 1995; Goswami, 2011; Toplak et al., 2006), it is tempting to speculate that such a generic functional property of the nervous system may help to explain the high co-morbidity of dyslexia and ADHD. However, motor timing studies of groups with ADHD and dyslexia have differed in respect to the sensory modality through which pacing stimuli are delivered: auditory stimuli have been typically employed in investigations of dyslexia (Badian & Wolff, 1977; Klipcera et al., 1981; Overy et al., 2003; Thomson et al., 2006; Thomson & Goswami, 2008; Wolff et al., 1984; Wolff, Michel, Ovrut, et al., 1990; Wolff, 2002) and visual or combined auditory-visual stimuli have been predominantly used in studies of ADHD (Ben-Pazi et al., 2003; Rubia et al., 2001, 2003; Toplak & Tannock, 2005b). As described in Chapter 2, investigations of timing performance paced by different stimulus modalities show that the high temporal acuity of the auditory system facilitates precise synchronisation of motor behaviour with acoustically-presented pacing stimuli (Fendrich & Corballis, 2001; McAuley & Henry, 2010; Patel et al., 2005). In contrast, motor synchronisation to visual stimuli typically results in greater response variability (Jäncke, Loose, et al., 2000; Kolers & Brewster, 1985; McAuley & Henry, 2010). This effect has been interpreted as evidence that limited information is available to timekeeping systems in visually paced tasks, preventing effective monitoring and updating of associated output responses (Kolers & Brewster, 1985). Recent evidence from behavioural (Loras et al., 2012) and neuroimaging studies (Jäncke, Loose, et al., 2000) further highlights the importance of stimulus mode as a critical variable in understanding intra- and inter-subject differences in motor synchronisation tasks.

The aim of this study was therefore to evaluate the potential clinical relevance of motor synchronisation tasks in the context of these important methodological considerations. The behavioural effects of altering task parameters on timing performance were examined in adults (Experiment 1) and children (Experiment 2). It was expected that children, like adults would have more accurate timing performance with auditory pacing stimuli compared to visually paced tasks. Furthermore, performance with bimodal (simultaneous visual and auditory) stimuli should closely replicate auditory performance because the auditory stimuli tend to dominate over visual stimuli when presented bimodally, driving behaviour under such conditions (Repp & Penel, 2002). In addition to recording behaviour with standard measures of performance (e.g. IRI and IRI variability), performance was also analysed using the variance decomposition model described in Chapters 2 and 5 (Wing & Kristofferson, 1973a, 1973b). As described in Chapter 5, a method of adjusting the Wing-

Kristofferson model was applied to take account of the limited sample of time series data collected, non-stationarity in the data and any negative variance estimates by correcting to zero, as described by Kooistra, Snijders, Schellekens, Kalverboer and Geuze (1997).

As well as comparing between the tasks, statistical relationships were assessed between timing variables and measures of literacy and attention which tap the key cognitive dimensions that form the core deficits in developmental dyslexia and ADHD respectively. Timing performance has been found to be associated with intelligence, literacy and attention measures across both typically developing populations and those with specific disorders and so correlations were expected between timekeeper variance and these measures. Both of these analyses allow the validity of the task parameters to be assessed, and are an important prerequisite to the application of these methods to clinical samples, including children with developmental disorders (e.g. Chapter 8).

6.3. Experiment 1: Effect of stimulus modality on motor timing in adults

6.3.1. Methods

Further details of methods, measures and analyses were presented in Chapters 4 and 5.

6.3.1.1. Participants

The sample comprised a group of thirty-five adults drawn from University staff and student populations. Eight participants met the exclusionary criteria: three for whom English was their second language; one due to the presence of a chronic neurological disorder; three due to a failure to record sufficient tapping responses in the motor timing task and one due to a Verbal IQ score more than 1.5 SD below the population mean. The remaining 27 participants included 14 males and 13 females (age range 18-40 years, two left-handed). On a preliminary questionnaire 63% of the group reported previous experience with musical training (mean = 2.8 years, range = 1-16 years) and all participants reported no recent use of medication which could affect their performance on the tasks employed in this battery.

6.3.1.2. Psychometric measures

The following psychometric measures were employed to assess cognitive dimensions associated with the core diagnostic symptoms of dyslexia and ADHD.

The Similarities and Matrices subscales from the Wechsler Abbreviated Scale of Intelligence (WASI, Wechsler, 1999) were administered to measure verbal and non-verbal reasoning ability. Literacy skills were assessed using the Word Reading and Spelling subtests from the Wechsler Individual Achievement Test-II UK (WIAT-II UK, Wechsler, 2005). Age-referenced standard scores were derived for these tests using the published norms. Reading component skills were further assessed using computerised measures of orthographic and phonological processing skills comprising forced choice

measures of orthographic and phonological sensitivity (Olson & Forsberg, 1994). Total score and mean reaction time were converted into a time-error composite measure for each test. The Self Report Form from the ADHD Current Symptoms Scale (Barkley & Murphy, 1998) was used to measure symptoms of ADHD, yielding Inattention (ADHD-IA) and Hyperactivity-Impulsivity (ADHD-HI) subscale scores. In addition, a Stroop task was administered as a measure of attentional control. This comprised a colour-word form and a parallel word-free version (with coloured 'X' symbols matched in length to the colour words). The total time taken to complete each Stroop task was recorded and the percentage increase in time taken to complete the colour-word task compared to the symbol task was calculated.

6.3.1.3. Motor timing measures

6.3.1.3.a. Simple reaction time

Participants completed a measure of simple reaction time to allow analysis of whether difficulties in finger tapping to isochronous sequences were related to a general slowness in motor speed. This task was completed in each of the three stimulus modalities used in the finger tapping paradigm described below (visual, auditory, bimodal). Participants completed 10 reaction time trials within each of the three modalities. Mean reaction time and SD were calculated for each condition. Responses were made on a flat switch plate housed within a covered box so that participants were not able to gain visual feedback (from their hand movements) during any of the trials.

6.3.1.3.b. Synchronised finger tapping

The primary experimental measure was a synchronisation task in which participants were instructed to tap the index finger of their dominant hand 'in time' with the repeated onsets of externally delivered pacing stimuli. The trials were presented in separate blocks, distinguished only by the different modes of stimulus presentation (auditory, visual or bimodal). Within each block, participants completed three separate trial sequences, each consisting of 40 isochronous pacing stimuli with onsets timed to achieve an inter-onset interval (IOI) of 329ms (3Hz). Three further blocks of filler sequences were interspersed between the trial blocks to reduce the potential effects of entrainment to the stimulus presentation rate which can occur even across modalities (McAuley & Henry, 2010). These auditory, visual and bimodal distracter blocks each comprised three synchronisation trials, each with 20 pacing stimuli presented at an IOI of 505ms (2Hz). The order of the six blocks (2 speeds x 3 modalities) was randomised across participants. Responses were registered with the same switch plate housed in the covered box as described above.

In the auditory condition the stimuli were 50ms auditory tones presented through computer speakers. The visual stimuli comprised a 2 cm diameter red diamond presented in the centre of a CRT computer monitor for 50ms. In the bimodal condition the same auditory and visual stimuli were presented with simultaneous onsets.

6.3.1.4. Data analysis

For the synchronised finger tapping task, the first five finger tap responses from each trial run were removed from the analyses to allow stabilisation of responses. An inter-response interval (IRI) was calculated for each of the 30 remaining responses. IRIs that that fell outside the range of 50% of the target interval (i.e., greater than 495ms or less than 165ms for the 329ms target interval) were removed from the analysis as invalid responses on the basis that they likely resulted from response errors (for e.g., doubled responses). This practice is common in studies of this kind (e.g. Freeman et al., 1996; Jäncke et al., 2000; Kooistra et al., 1997; O'Boyle, Freeman, & Cody, 1996; Pastor, Jahanshahi, Artieda, & Obeso, 1992) increasing the validity of the time series data and reducing the probability that the autocorrelation of successive intervals will exceed the bounds (of -0.5 to 0) set out in the Wing-Kristofferson model (Kooistra et al., 1997).

Data were not analysed for a given trial if more than 10 responses were considered invalid (presently, 7% of trials, including nine trials from the auditory blocks, four trials from the visual blocks and four from the bimodal blocks). Five of the missing trials were from a single participant who had average intelligence and literacy skills; their remaining data was included in the analyses below. Mean and standard deviation (SD) of IRIs were calculated for each trial. Mean absolute interval difference was also obtained, defined as the difference between the target stimulus interval and the IRI achieved by the participant, averaged within a trial.

From the raw data collected for each trial, estimates of the different components of timing variance were calculated using a method consistent with the Wing-Kristofferson model (Kooistra et al., 1997) such that each interval results from the timekeeper and implementation variance and the drift parameter (Equation 9: $I_j = T_j - P_{j-1} + P_j + \beta_j$). As described in Chapter 5, the observed variance and lag-one covariance in the time series were used to generate estimates of the contribution of the timekeeper and peripheral components (Equations 10 and 11). Upon screening these estimates one trial from one participant was removed given that the variance estimates were so large it was unlikely that the participant had completed the task appropriately. Despite the adjustments in the Kooistra method accounting for sample size and non-stationarity, negative variance estimates may still be present (Kooistra et al., 1997) because statistical variability in the estimates means that they can, by chance, fall in the tail of the sampling distribution of estimates (Collier & Ogden, 2004; Kooistra et al., 1997). In previous investigations such negative variance estimates are commonly observed in 10-30% of trials and have been corrected to zero (Collier & Ogden, 2004; Harrington et al., 1998; Ivry & Keele, 1989; Kooistra et al., 1997). Here the number of estimates with negative variance that were corrected to zero was comparable to previous studies, comprising 12.5% of trials (Table 6.1). The drift component (β_j) is effectively a measure of the slope of a linear regression between the inter-response intervals and the tap number in the sequence. When examined across

trials, the slope coefficient was not significantly different from zero, except for in the third auditory trial ($t=2.22$, $df=21$, $p<0.05$).

Table 6.1: Number of trials where negative variance estimates were corrected to zero

	Violations of the lag-one autocorrelation bounds where:	
	$\rho_I(1) < -0.5$	$\rho_I(1) > 0$
Auditory	7	13
Visual	13	6
Bimodal	15	7
Total Trials with negative variance	14.4 %	10.7 %
Total of all trials with negative variance		12.5%

The Wing-Kristofferson model predicts that the lag-one autocorrelation falls within the bounds of zero to minus one half. Violations of the lower limit result from negative timekeeper variance and violations of the upper limit result from negative implementation variance. Total n trials = 243.

The model fit was evaluated by calculating the average autocorrelation of successive intervals across trials within each modality condition. The autocorrelation values were within the accepted limits of minus one half and zero for dependence between intervals (based on Equation 8: Chapter 5). The autocorrelation function (ACF) was also calculated as a measure of the ratio between the two variance components (timekeeper and implementation) in the absence of individual differences in variance magnitude. Higher autocorrelation values indicate that there is a greater proportion of timekeeper variance relative to implementation variance. The ACF was calculated on variance values in which any negative estimates had been corrected to zero. Where presented, the square root of variance estimates are used to provide values as standard deviations (ms), a common transformation in the literature to reduce skew in the data (Collier & Ogden, 2004; O'Boyle et al., 1996; Pastor et al., 1992).

6.3.2. Results

6.3.2.1. Psychometric measures

Descriptive statistics for the psychometric measures are shown in Table 6.2. As a group, the reasoning abilities and reading and spelling standard scores were within the average range. On the tests of phonological and orthographic skill participants performed close to ceiling level indicating that these measures did not sufficiently discriminate amongst these skills in this sample. These measures were therefore excluded from the later assessment of the relationships between literacy and motor timing variables. The group mean scores on the ADHD-IA and ADHD-HI rating scales were within one point of the normative averages of the standardisation sample and did not meet the clinical threshold for symptom ratings (of 1.5 SD above the mean) (Barkley & Murphy, 1998).

Table 6.2: Descriptive statistics for the psychometric measures

	Mean	Standard deviation
Age (years)	21.3	(4.5)
Music experience (years)	2.8	(3.7)
Verbal Reasoning	109.3	(9.3)
Non-verbal Reasoning	103.4	(13.2)
Word Reading	108.4	(7.4)
Spelling	108.7	(11.1)
Orthographic Choice (% correct)	96.3	(6.0)
Phonological Choice (% correct)	84.4	(11.9)
Orthographic Time (average RT, ms)	1085	(410.0)
Phonological Time (average RT, ms)	3173	(750.2)
ADHD-IA (subscale score)	6.9	(3.3)
ADHD-HI (subscale score)	5.2	(2.7)
Stroop (% time increase)	45.2	(15.2)

Values are standard scores (mean 100, SD 15) unless otherwise indicated.

6.3.2.2. Motor timing measures

The effect of stimulus modality on timing performance was evaluated using a series of Analyses of Variance (ANOVA).

6.3.2.2.a. Stimulus modality and simple reaction time

The mean reaction times (Table 6.3) were typically faster with bimodal stimuli than with visual or auditory stimuli. A within-subjects analysis of variance (ANOVA) confirmed that there was a significant difference in reaction times across the modalities ($F_{(2,52)}$=9.44, p<0.01, η^2=0.27) and post-hoc t-tests confirmed that this was attributable to significantly faster response times in the bimodal condition than in the auditory ($t_{(26)}$=4.09, p<0.01, η^2=0.25) or visual conditions ($t_{(26)}$=3.56, p<0.01, η^2=0.20) which did not significantly differ from one another ($t_{(26)}$=0.87, p=0.39, η^2=0.01).

Table 6.3: Average reaction times across stimulus modalities

	Mean (ms)	Standard deviation
Auditory	291	(45.8)
Visual	285	(34.5)
Bimodal	262	(36.2)

6.3.2.2.b. Stimulus modality in synchronised finger tapping

Two-way within subjects ANOVAs were conducted to assess the effect of stimulus modality (three levels: auditory, visual and bimodal) and trial number (three levels) on timing performance. Trial number was included as a factor to assess the influence of practice effects that can arise in motor timing tasks (Kolers & Brewster, 1985; McAuley & Henry, 2010). Descriptive statistics are provided in Table 6.4 and Figure 6.1.

Table 6.4: Synchronised finger tapping performance across stimulus modalities

Trial		IRI		IRI SD		Mean Absolute Interval Difference	
		Mean (ms)	(SD)	Mean (ms)	(SD)	Mean (ms)	(SD)
Auditory	1	326	(3.9)	24	(5.5)	18	(4.6)
	2	327	(4.5)	26	(5.9)	20	(5.0)
	3	326	(4.4)	26	(7.2)	20	(5.5)
Visual	1	320	(40.2)	26	(9.1)	36	(22.1)
	2	323	(41.2)	22	(5.8)	34	(23.3)
	3	312	(44.2)	28	(12.3)	41	(26.7)
Bimodal	1	325	(9.7)	26	(6.9)	20	(6.0)
	2	324	(6.0)	25	(5.4)	20	(4.9)
	3	325	(7.7)	25	(6.4)	20	(5.6)

Variables include means (ms) and SD for: within trial IRI, within trial SD of IRIs and absolute interval difference, calculated as the average of differences between the target stimulus interval of 329ms and the IRI achieved by the participant.

A two-way ANOVA showed that neither trial nor modality had a significant effect on mean IRI (Greenhouse Geisser corrected df: trial - $F_{(1.1,15.3)}$=1.94, p=0.16, η^2=0.12 and modality - $F_{(1.8,24.7)}$=0.46, p=0.64, η^2=0.03), despite the indications from Figure 6.1 that mean IRIs were smaller in the visual condition and that there was more variability in Mean IRI across the group in this condition compared to the auditory or bimodal trials.

The mean data (Table 6.2) indicated similar levels of IRI variability across all three stimulus conditions and a two-way ANOVA confirmed that differences between the three modalities were not significant ($F_{(2,28)}$=1.41, p=0.26, η^2=0.09) and that there was no effect of trial number ($F_{(2,28)}$=2.58, p=0.09, η^2=1.56).

Examination of the average data for absolute interval difference (Table 6.4) suggests that the asynchrony between IRIs and the target IOI was greater in the visual modality than in the other stimulus conditions. Variability across the group was also higher in the visual condition. The ANOVA showed a significant effect of modality for mean absolute interval difference ($F_{(1.11,15.5)}$=9.89, p<0.01,

η^2=0.41), but no effect of trial number ($F_{(1.6,22.5)}$=2.35, p=0.11, η^2=0.14). Collapsing across the three trials, post-hoc t-tests confirmed that the values were significantly higher in the visual condition than in either the bimodal ($t_{(73)}$=6.23, p<0.01, η^2=0.21) or auditory conditions ($t_{(66)}$=6.39, p<0.01, η^2=0.23) which also did not differ significantly from one another ($t_{(68)}$=-1.74, p=0.09, η^2=0.02).

6.3.2.2.c. Decomposed timing variance

As there was no effect of trial number in the above analyses of timing performance, it was deemed appropriate to collapse across the three trials for analyses of the effect of stimulus modality on the decomposed timing variables (descriptive data provided in Table 6.5 and Figure 6.2). The main effect of stimulus modality on timekeeper variance approached significance (Greenhouse-Geisser corrected df: $F_{(1.4,36.7)}$=2.89, p=0.08, η^2=0.10), however this effect was due to an outlying case and was not significant once the outlier had been removed from the solution. For implementation variance, a significant main effect of stimulus modality was found ($F_{(2,50)}$=7.09, p<0.01, η^2=0.22). Post-hoc t-tests indicated that implementation variance in the auditory condition was significantly higher than in the visual ($t_{(25)}$=-3.28, p<0.01, η^2=0.17) and bimodal conditions ($t_{(25)}$=2.56, p<0.05, η^2=0.11) which did not differ significantly from one another ($t_{(25)}$=-0.91, p=0.37, η^2=0.02). This result is in contrast to the results described above for the other performance measures.

An ANOVA showed a significant main effect of stimulus modality on ACF values ($F_{(2,50)}$=3.88, p<0.05, η^2= 0.13). Post-hoc tests showed that this was the case, with higher values in the auditory condition compared to both the visual ($t_{(25)}$=2.39, p<0.05, η^2=0.11) and bimodal conditions ($t_{(25)}$=2.42, p<0.05, η^2=0.11) which did not differ from one another ($t_{(26)}$=-0.22, p=0.83, η^2<0.01).

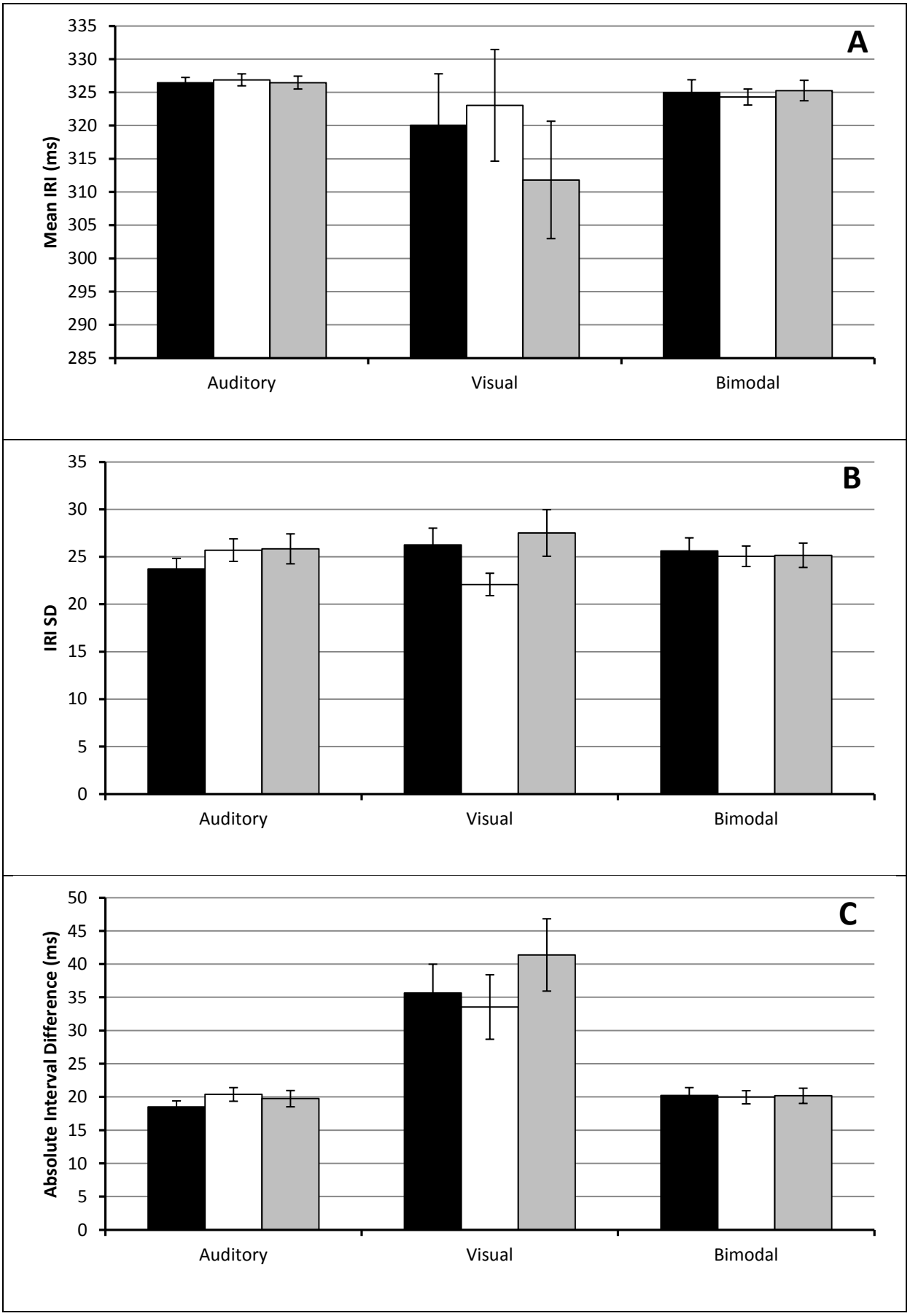

Figure 6.1: Synchronised finger tapping performance

Showing performance variables Mean IRI (A), IRI SD (B) and Absolute Interval Difference (C) across stimulus modalities and trials (trial 1-black bars; trial 2-white bars and trial 3-grey bars).

Table 6.5: Decomposed timing variance across stimulus modalities

Trial	Mean Timekeeper Variance		Mean Implementation Variance		ACF	
	Mean (ms)	(SD)	Mean (ms)	(SD)	Mean	(SD)
Auditory	323	(222.3)	199	(140.1)	-0.3	(0.1)
Visual	583	(648.4)	103	(57.8)	-0.2	(0.1)
Bimodal	477	(356.7)	124	(111.4)	-0.2	(0.2)

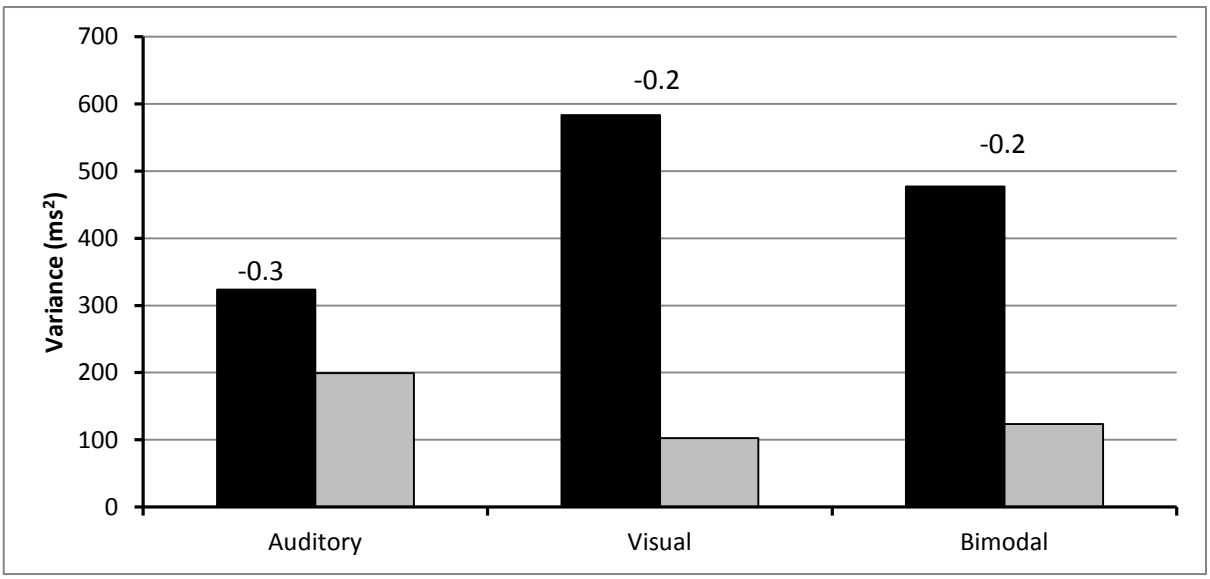

Figure 6.2: Decomposed timing variance across stimulus modalities
Decomposed variables of timekeeper variance (black bars) and implementation variance (grey bars) are shown with the autocorrelation function values (above the bars) which represents the ratio of timekeeper to implementation variance (the ACF).

6.3.2.3. Correlational analyses

To evaluate the consistency of timing across modalities, Pearson's product moment correlations were calculated. Outliers had the potential to distort the solutions in these analyses and were therefore identified and dealt with through procedures described in Chapter 4. In the absence of any effects of trial number and to reduce the risk of Type 1 errors that would result from a large number of comparisons the decomposed timing variables (timekeeper variance, implementation variance and ACF) were averaged across the three trials.

6.3.2.3.a. Relationships between motor timing performance and reaction time

The correlations between motor timing performance variables and reaction time were assessed to examine the contribution of perceptual reaction time to performance. Reaction times across the conditions were not associated with any of the timing measures (Table 6.6).

Table 6.6: Correlations between motor timing performance measures and reaction time

Trial	Auditory TK	Auditory IMP	Auditory ACF	Visual TK	Visual IMP	Visual ACF	Bimodal TK	Bimodal IMP	Bimodal ACF	Auditory RT	Visual RT
Auditory RT	-0.22	0.08	-0.23	0.13	0.08	-0.08	-0.12	0.15	-0.13	-	-
Visual RT	-0.09	-0.02	-0.01	-0.03	0.22	-0.23	-0.01	-0.09	0.13	0.53**	-
Bimodal RT	0.14	-0.04	0.14	0.0	-0.06	-0.07	-0.01	-0.07	0.05	0.59**	0.59**

*Variables include timekeeper variance (TK), implementation variance (IMP), autocorrelation function (ACF) and simple reaction time (RT); *p<0.05, **p<0.01.*

6.3.2.3.b. Relationships between motor timing performance and cognitive / behavioural predictors

To examine the ability of motor timing performance to predict of symptoms of dyslexia and ADHD, correlations were also calculated between the same component measures of timing (timekeeper, implementation and ACF) and measures of literacy and attention. Previous investigations (e.g. Holm, Ullén, & Madison, 2011; Mcauley, Jones, Holub, Johnston, & Miller, 2006) have found that reasoning ability relates to timing performance so reasoning ability was controlled in the partial correlations with timing performance and are presented alongside the zero-order correlations in Table 6.7.

Auditory and bimodal timing performance measures were not associated with the literacy or attention variables. Implementation variance under visual conditions was strongly associated with non-verbal reasoning performance ($r=-0.49$, $p<0.05$) and with the Stroop measure once reasoning ability was controlled ($r=-0.47$, $p<0.05$), with higher interference scores on this task being associated with lower implementation variance.

Table 6.7: Correlations between motor timing performance and measures of literacy and attention

	Auditory TK	Auditory IMP	Auditory ACF	Visual TK	Visual IMP	Visual ACF	Bimodal TK	Bimodal IMP	Bimodal ACF	Reading	Spelling	ADHD-IA	ADHD-HI	Stroop % Increase	Non-verbal Reasoning	Verbal Reasoning
Auditory TK	-	-0.41*	0.85**	0.08	0.05	0.05	0.28	-0.21	0.22	-0.15	-0.16	0.05	0.21	0.26	-0.16	-0.30
Auditory IMP	-0.48*	-	-0.71**	0.25	0.02	0.25	0.03	0.37	-0.25	-0.08	-0.13	-0.28	-0.19	0.18	-0.17	-0.15
Auditory ACF	0.86**	-0.75**	-	0.09	-0.09	0.08	0.29	-0.38[A]	0.38[A]	-0.10	-0.12	0.05	0.15	0.10	-0.08	-0.11
Visual TK	0.03	0.24	0.07	-	-0.38*	0.87**	0.51**	0.05	0.15	-0.27	-0.37	-0.10	0.08	0.30	-0.06	-0.19
Visual IMP	-0.04	-0.07	-0.15	-0.48*	-	-0.64**	-0.32	0.08	-0.16	-0.04	0.16	0.08	0.22	-0.38[A]	-0.49*	-0.29
Visual ACF	0.04	0.26	0.08	0.89**	-0.67**	-	0.58**	-0.08	0.27	-0.08	-0.31	-0.12	-0.02	0.32	0.08	-0.06
Bimodal TK	0.28	0.02	0.29	0.52**	-0.39	0.61**	-	-0.63**	0.77**	-0.01	-0.14	-0.02	0.09	0.00	0.01	-0.05
Bimodal IMP	-0.25	0.39	-0.40[A]	0.01	0.20	-0.18	-0.66**	-	-0.92**	-0.19	-0.07	-0.13	0.03	0.30	0.09	-0.10
Bimodal ACF	0.27	-0.25	0.40[A]	0.21	-0.27	0.37	0.80**	-0.91**	-	0.15	-0.01	0.13	-0.01	-0.22	-0.03	0.12
Reading	-0.08	-0.02	-0.07	-0.25	0.17	-0.10	-0.01	-0.23	0.17	-	0.80**	-0.08	-0.19	-0.05	0.35	0.24
Spelling	-0.11	-0.08	-0.09	-0.37	0.39[A]	-0.36	-0.15	-0.10	-0.00	0.77**	-	0.06	-0.07	-0.17	0.33	0.21
ADHD-IA	0.01	-0.31	0.03	-0.12	0.04	-0.13	-0.03	-0.14	0.15	-0.05	0.10	-	0.66**	-0.04	-0.07	-0.11
ADHD-HI	0.20	-0.22	0.14	0.08	0.18	-0.00	0.10	0.05	-0.02	-0.16	-0.03	0.66**	-	0.04	-0.15	-0.06
Stroop % Increase	0.19	0.17	0.08	0.25	-0.47*	0.30	-0.02	0.26	-0.18	-0.04	-0.18	-0.08	0.06	-	0.02	-0.30
Non-verbal Reasoning															-	0.52**

*Pearson's product moment correlations (top right) between motor timing performance and psychometric variables of interest, with partial correlations controlling for verbal and non-verbal reasoning (bottom left). Variables include timekeeper variance (TK), Implementation variance (IMP) and Autocorrelation Function (ACF); *p<0.05, **p<0.01, [A]p=0.05.*

6.3.3. Discussion

6.3.3.1. Modality dependent effects

The results of this experiment support evidence that the modality of the pacing stimuli in synchronisation paradigms strongly modulates timing performance (Fendrich & Corballis, 2001; Jäncke, Loose, et al., 2000; Kolers & Brewster, 1985; McAuley & Henry, 2010; Patel et al., 2005). Adults were able to maintain regular IRIs in the visual condition but responses were further from the stimulus onset in this condition than with either auditory or bimodal stimuli. This is indicative of a difficulty in judging the synchrony of responses with visually presented temporal stimuli, a finding that concurs with previous studies that have found a difference in asynchrony between tap and tone onsets in the visual modality (Jäncke, Loose, et al., 2000; Kolers & Brewster, 1985; Loras et al., 2012; Pollok et al., 2009; Repp & Penel, 2002).

Use of the Wing-Kristofferson model allowed the variability in performance to be further analysed and revealed differences in the modus of temporal control across the different conditions. There were no substantial differences between the stimulus conditions for timekeeper variance, but implementation variance was significantly higher when finger tapping to auditory pacing stimuli compared to tapping with bimodal or visual stimuli. This result is somewhat unexpected given that the auditory task should be relatively undemanding. However, when the variance components were analysed together the ratio of timekeeper variance to implementation variance was much smaller (higher ACF) in the auditory condition than in the other two conditions. This suggests that under auditory guidance very little of the variance in responses was attributable to timekeeping process leading to minimal performance variability across the course of the 30 taps. Due to the nature of the two component Wing-Kristofferson model any additional variance was therefore attributed to the motor system.

This response profile under auditory pacing conditions is in contrast to that for the visual and bimodal trials where there was a higher ratio of timekeeper variance to implementation variance, with much higher timekeeper variance relative to implementation variance. The analyses indicate that under these conditions the timekeeping system was not entrained to the stimulus. Rather than becoming entrained to a response rate, it appears that these participants were attempting to follow the task instructions and keep in time with the stimuli. Because visual stimuli are generally found to be unreliable perceptual indicators of temporal events, such a behavioural strategy would lead to the low ACF values and the large asynchrony between stimulus and response onsets that were observed. This supports studies which have found that visual timing is not lead by generation of an internal rhythm but instead stems from a focus on the on-going perception of stimulus characteristics in order to maintain synchrony (Jäncke, Loose, et al., 2000; Jantzen et al., 2004; Kolers & Brewster, 1985; Pollok et al., 2006). Although differences between the stimulus conditions were not evident

from the simple measure of variability (SD of IRIs), the use of the Wing-Kristofferson model allowed the discrepancies between modalities to be elucidated. It is unlikely that the differences between the conditions resulted from a perceptual effect because simple reaction times were faster with bimodal stimuli than with either auditory or visual stimuli, replicating previous results (Alpert, Hein, Tsai, Naumer, & Knight, 2008).

Both the bimodal and visual conditions were found to result in autocorrelation values (ACF) that were similar to each other but significantly different from those in the auditory condition. This is expected given that visual stimuli are present in both the visual and bimodal trials; stimuli which generally give rise to poor temporal resolution. The two conditions were not wholly similar, however. The higher timekeeper variance in the visual modality was accompanied by higher absolute interval difference values, such that responses were more discrepant from the target stimulus rate. In comparison, in the bimodal condition, large asynchronies were not present despite the presence of relatively higher timekeeper variance. The timekeeper variance variable likely measures the output from multiple processing stages within the temporal processing system as well as any pulse generation by the timekeeper (Elliott et al., 2010). Therefore the timekeeper estimates may incorporate perceptual compensation processes that make use of the auditory stimuli present in the bimodal condition, allowing synchrony to be maintained in spite of the presence of the visual stimuli.

Previous investigations of the differences between performance under different stimulus modalities have typically found higher variability in visual tasks (Jäncke, Loose, et al., 2000; Kolers & Brewster, 1985; Repp & Penel, 2002). Here, the variability (IRI SD) did not differ across trials. It is possible that the participants may have disregarded the perceived visual stimuli that give rise to unreliable temporal information in favour the rate remembered from the auditory or bimodal trials. The dominance of the auditory stimuli over visual stimuli may contribute to such a strategy (Elliott et al., 2010; Fendrich & Corballis, 2001; Recanzone, 2003; Repp & Penel, 2002). The correlation found between timekeeper variance in the visual and bimodal trials does suggest there were similarities between performance strategies in these two modalities. Prior experience of the tapping rate has been shown to allow participants to learn the correct response rate without needing to monitor the stimuli within a given trial (Kolers & Brewster, 1985; McAuley & Henry, 2010). Here, participants completed the trials in a single experimental session, and so entrainment to the stimulus rate was possible. However, the extent to which prior learning of the rate on auditory or bimodal trials could affect performance should have been minimised by the randomisation of the three target modality blocks and inclusion of distracter trials at different response rates.

6.3.3.2. Relationships between motor timing performance and cognitive / behavioural predictors

In this sample of adults no significant associations were found between timing variables and the measures of literacy ability. In previous studies, samples where relationships between timing variables and literacy have been found have included participants with specific diagnoses of dyslexia, providing a larger degree of variance in literacy scores and improving the chances of finding such relationships (Thomson et al., 2006). For the attention measures, greater behavioural interference on the measure of attentional control was associated with reduced visual implementation variance. The participants who were more disrupted in naming colour stimuli on the Stroop task performed the visual task in a manner more similar to the auditory or bimodal tasks, perhaps ignoring the stimuli more in favour of a remembered rhythm. Participants with better performance on the Stroop task may have, in contrast, attempted to follow the task instructions to tap in time to the stimuli, a strategy which increased the amount of implementation variance. The Stroop task was not correlated with the self-reports of inattention and may not have been a valid measure of attentional control despite this measure being found to be a predictor of ADHD group membership (Lansbergen et al., 2007). The Stroop measure was not however associated with performance on the auditory or bimodal tasks, and these discrepancies between the conditions supports the evidence for a difference between these timing tasks.

6.4. Experiment 2: Effect of stimulus modality on motor timing in children

This study replicates Experiment 1 with a sample of children in order to assess whether altering the stimulus parameters has the same effects on children. Although visual, bimodal and auditory stimuli have all been used in previous experiments of motor timing with children with developmental disorders, the differences between the modalities has not been directly studied in this manner.

6.4.1. Methods

Additional details of the methodology were provided in Chapters 4 and 5.

6.4.1.1. Participants

A group of 25 children were recruited from primary years 4 and 5. Four were excluded prior to analysis: one child with an existing diagnosis of an emotional-behavioural disorder; one for whom English was a second language and two who failed to complete the experimental protocol. The remaining 21 children included 10 boys and 11 girls (age range 98-127 months; three left-handed). All 21 had, by chance, received musical instruction through either home- or school-based music lessons and all had received weekly classroom-based Samba drumming lessons throughout the previous academic year.

6.4.1.2. Psychometric measures

As in Experiment 1 (Section 6.3.1.2), psychometric measures were administered to the children to assess the cognitive dimensions associated with the core diagnostic symptoms of dyslexia and ADHD and their relationships with motor timing performance. The same measures as in Experiment 1 were employed to assess verbal and non-verbal reasoning ability (WASI; Wechsler, 1999) and Word Reading and Spelling ability (WIAT-II UK; Wechsler, 2005) and age-referenced standard scores derived using the published norms. Orthographic and phonological decoding skills were assessed using paper based measures of non-word (e.g. torlep) and irregular word (e.g. yacht) reading accuracy (Castles & Coltheart, 1993). The number of correctly read words for each word list (out of a total of 30) and total reading time were converted to a composite accuracy-speed score using a principal components analysis.

Teacher ratings of ADHD symptoms were obtained using the ADHD Behaviour Rating Scale Teacher Form (Barkley & Murphy, 1998) yielding ADHD-IA and ADHD-HI subscales. Attentional control was assessed using the Same World-Opposite World task from the Test of Everyday Attention for Children (TEA-Ch) (Manly et al., 1999). The time taken to complete the Same World and Opposite World trials was recorded and the percentage increase in time between the two tests was calculated. The Score! subscale from the TEA-Ch battery was used to measure sustained attention (Manly et al., 1999). The number of correctly scored trials was converted to an age-referenced standard score using the TEA-Ch norms.

6.4.1.3. Motor timing measures

The children were all administered the same Simple Reaction Time and Synchronised Finger Tapping tasks as the adults in Experiment 1. Responses were again recorded using the switch plate that was housed within a box to minimise visual feedback (see Section 6.3.1.3). The motor timing tasks were administered before the psychometric measures and the tasks were divided across two or three testing sessions, each of which lasted approximately 20 minutes.

6.4.1.4. Data analysis

Analysis of data from the synchronised finger tapping task followed the same procedures as in Experiment 1 (see Section 6.3). Trials with more than 10 invalid responses comprised 9% of trials, with six auditory trials, four visual trials and seven bimodal trials. The greatest number of trials missing for any one participant was three and these were spread across the stimulus modalities. Mean IRI, SD of IRIs and mean absolute interval difference were calculated for each trial and estimates of timekeeper and implementation variance were calculated using the Kooistra et al. (1997) adaption of the Wing-Kristofferson model (see Section 6.3.1.4 and Chapter 5). The degree of drift in the data was quantified using the drift parameter (β_j) which measures the linear trend in the time series from each trial. Across the trials in Experiment 2, the slope coefficients were not

significantly different from zero. Despite the adjustments in the model a number of the variance estimates were found to be negative (see Table 6.8) and were adjusted to zero. The percentage of trials with negative variance estimates was similar to that found in Experiment 1 and other studies of this kind (Collier & Ogden, 2004; Harrington et al., 1998; Ivry & Keele, 1989; Kooistra et al., 1997). The model fit was also evaluated by calculating the average auto correlation of successive intervals across trials within each modality condition and this fell within the acceptable limits of 0 to -0.5.

Table 6.8: Number of trials where negative variance estimates were corrected to zero

	Violations of the lag-one autocorrelation bounds where:	
	$\rho_I(1) < -0.5$	$\rho_I(1) < -0.5$
Auditory	6	6
Visual	3	15
Bimodal	8	7
Total Trials with negative variance	9.0 %	14.8 %
Total of all trials with negative variance		11.0 %

The Wing-Kristofferson model predicts that the lag-one autocorrelation falls within the bounds of zero to minus one half. Violations of the lower limit result from negative timekeeper variance and violations of the upper limit result from negative implementation variance. Total n trials = 189.

6.4.2. Results

6.4.2.1. Psychometric measures

Descriptive statistics for the psychometric measures are presented in Table 6.9. The children, as a group, had reading, spelling and non-verbal reasoning performance in the average range. The group mean for verbal reasoning was somewhat higher than the typical population mean of 100. On the non-word reading test, participants were performing close to ceiling level (Mean = 82.7, SD = 15.15). There was more variance in the scores on the test of irregular word reading, and variability was evident in the completion times for both measures. Children scored in the average range on the measures of attentional control and sustained attention. The group mean scores on the ADHD-IA and ADHD_HI rating scales were close to the normative averages of the standardisation sample and were below the clinical threshold for symptom ratings (of 1.5 SD above the mean) (Barkley & Murphy, 1998).

6.4.2.2. Motor timing measures

The effect of stimulus modality on timing performance was tested using a series of ANOVA. Solutions were checked in the presence and absence of outliers and outliers remain in the analyses below unless they had a significant effect on the statistical outcome, in which case the amended analysis is also reported.

Table 6.9: Descriptive statistics for the psychometric measures

	Mean	Standard deviation
Age (years)	115.3	(9.2)
Music experience (years)	1.4	(0.7)
Verbal Reasoning	119.4	(9.4)
Non-verbal Reasoning	104.9	(10.5)
Word Reading	106.9	(10.2)
Spelling	104.9	(12.3)
Non-word Reading (% correct)	82.7	(15.2)
Irregular-word Reading (% correct)	60.2	(8.9)
Non-word Reading (completion time)	46.4	(20.2)
Irregular-word Reading (completion time)	36.2	(16.5)
ADHD-IA (subscale score)	4.8	(5.4)
ADHD-HI (subscale score)	2.9	(4.0)
Opposite World	102.6	(11.6)
Opposite World-Same World Time Increase	32.4	(18.5)
Score!	96.9	(15.5)

Values are standard scores (mean 100, SD 15) unless otherwise indicated.

6.4.2.2.a. Stimulus modality and simple reaction time

The mean reaction time data are presented in Table 6.10. A within subjects ANOVA confirmed a main effect of stimulus type ($F_{(2,40)}$=5.04, p<0.01, η^2=0.20). Post-hoc t-tests showed that the visual and auditory reaction times were not significantly different from each other ($t_{(20)}$=1.12, p=0.27, η^2=0.03) and reaction times were faster in the bimodal condition than either the auditory ($t_{(20)}$=3.01, p<0.01, η^2=0.19) or visual conditions ($t_{(20)}$=3.06, p<0.01, η^2=0.19).

Table 6.10: Average reaction times across stimulus modalities

	Mean (ms)	Standard deviation
Auditory	335	(81.7)
Visual	320	(44.7)
Bimodal	301	(48.0)

6.4.2.2.b. Stimulus modality in synchronised finger tapping

Two-way within subjects ANOVA were conducted to assess motor timing performance across the three stimulus modalities (auditory, visual and bimodal) and across the three trials performed within each modality. Trial number was included as a factor to assess the influence of practice effects that can arise in motor timing tasks (Kolers & Brewster, 1985; McAuley & Henry, 2010). Descriptive statistics are presented in Table 6.11 and Figure 6.3.

The data in Table 6.11 illustrate that the mean IRIs were close to the target IOI in the bimodal and auditory conditions and that tapping was faster than the target rate in the visual condition. In addition, the between-participant variability (IRI SD) was higher in the visual condition than in the other two stimulus modalities. A two-way ANOVA showed no effect of stimulus modality on mean IRI ($F_{(2,18)}$=0.95, p=0.41, η^2=0.1), but a significant main effect of trial number ($F_{(2,18)}$=4.08, p<0.05, η^2=0.25). Post-hoc t-tests showed that this resulted from smaller mean IRIs on the third trial than in the first ($t_{(51)}$=2.40, p<0.05, η^2=0.05) or second trials ($t_{(18)}$=2.31, p<0.05, η^2=0.05), which did not differ from each other ($t_{(55)}$=0.13, p=0.90, η^2<0.01). The interaction effect was not significant ($F_{(4,36)}$=2.04, p=0.11, η^2=0.19). Four outlying cases were identified, spread across the modalities (two in the auditory condition and one in the bimodal condition with extremely small mean IRIs and one in the visual condition with a very large mean IRI. When the ANOVA was repeated, omitting these outliers, the main effect of trial number was not significant ($F_{(2,12)}$=2.12, p<0.16, η^2=0.19).

The means presented in Table 6.11 suggest that the within participant variability (IRI SD) was similar across all trials. The ANOVA confirmed that the main effects of trial and modality were not significant ($F_{(2,18)}$=0.62, p=0.55, η^2=0.06 and $F_{(2,18)}$=0.19, p=0.83, η^2=0.02 respectively) and this result was consistent irrespective of the presence of outliers in the analysis. An ANOVA also confirmed a significant main effect of stimulus modality on mean absolute interval differences (Greenhouse-Geisser corrected: $F_{(1.08, 9.72)}$=13.80, p<0.01, η^2=0.60) but no effect of trial number ($F_{(2,18)}$=0.04, p=0.96, η^2<0.01). Post-hoc t-tests showed that the modality effect was due to absolute interval differences being larger in the visual mode than the auditory ($t_{(52)}$=7.10, p<0.01, η^2= 0.32) or bimodal conditions ($t_{(52)}$=7.24, p<0.01, η^2=0.34) (Table 6.11) which did not differ significantly from each other ($t_{(49)}$=-0.36, p=0.72, η^2<0.01). These results remained even in the absence of outliers.

6.4.2.2.c. Decomposed timing variance

As trial number did not affect performance, it was appropriate to collapse across trials for the analyses of the effect of stimulus modality on the decomposed timing variables. Inspection of the means (Table 6.12 and Figure 6.4) suggests there was higher timekeeper variance and lower implementation variance in the visual condition than in the other two modalities as well as more between participant variability (SD of timekeeper variance). The main effect of stimulus modality on timekeeper variance was significant ($F_{(2,40)}$=3.54, p<0.05, η^2=0.15). Post hoc t-tests showed that

timekeeper variance was significantly higher with visual pacing stimuli than with auditory stimuli ($t_{(20)}$=2.30, p<0.05, η^2=0.12). The difference between timekeeper variance in the visual and bimodal conditions did not reach significance ($t_{(20)}$=1.98, p=0.06, η^2=0.09) and there was no difference between timekeeper variance in the auditory and bimodal conditions ($t_{(20)}$=-0.29, p=0.77, η^2<0.01).

A main effect of stimulus modality was also found with implementation variance as the dependent variable (Greenhouse-Geisser corrected: $F_{(1.5,29.49)}$=6.77, p<0.01, η^2=0.25). Post-hoc t-tests showed that implementation variance was lower in the visual modality than in either the auditory ($t_{(20)}$=2.43, p<0.05, η^2=0.13) or bimodal trials ($t_{(20)}$=3.32, p<0.01, η^2=0.22), which did not differ from one another ($t_{(20)}$=-0.54, p=0.60, η^2<0.01). A main effect of modality on ACF was confirmed with an ANOVA ($F_{(2,40)}$=5.35, p<0.01, η^2=0.21) and post-hoc t-tests confirmed that this was due to higher ACF values in both the auditory and bimodal conditions than in the visual condition ($t_{(20)}$=2.84, p<0.01, η^2=0.18 and $t_{(20)}$=2.56, p<0.05, η^2=0.15, respectively). The ACF values in the auditory and bimodal conditions were not significantly different from one another ($t_{(20)}$=-0.34, p=0.74, η^2<0.01).

Table 6.11: Synchronised finger tapping performance across stimulus modalities

Trial		IRI		IRI SD		Mean Absolute Interval Difference	
		Mean (ms)	(SD)	Mean (ms)	(SD)	Mean (ms)	(SD)
Auditory	1	326	(4.5)	34	(10.8)	28	(8.6)
	2	326	(4.3)	33	(8.9)	26	(7.2)
	3	324	(6.8)	35	(10.3)	28	(8.8)
Visual	1	309	(46.2)	31	(6.3)	48	(23.8)
	2	305	(45.4)	34	(9.9)	50	(22.6)
	3	298	(35.0)	34	(11.3)	47	(23.8)
Bimodal	1	326	(7.0)	34	(7.3)	27	(6.3)
	2	327	(4.9)	35	(11.4)	27	(8.0)
	3	325	(8.4)	35	(8.9)	27	(8.3)

Variables include means (ms) and SD for: within trial IRI, within trial SD of IRIs and absolute interval difference, calculated as the average of differences between the target stimulus interval of 329ms and the IRI achieved by the participant.

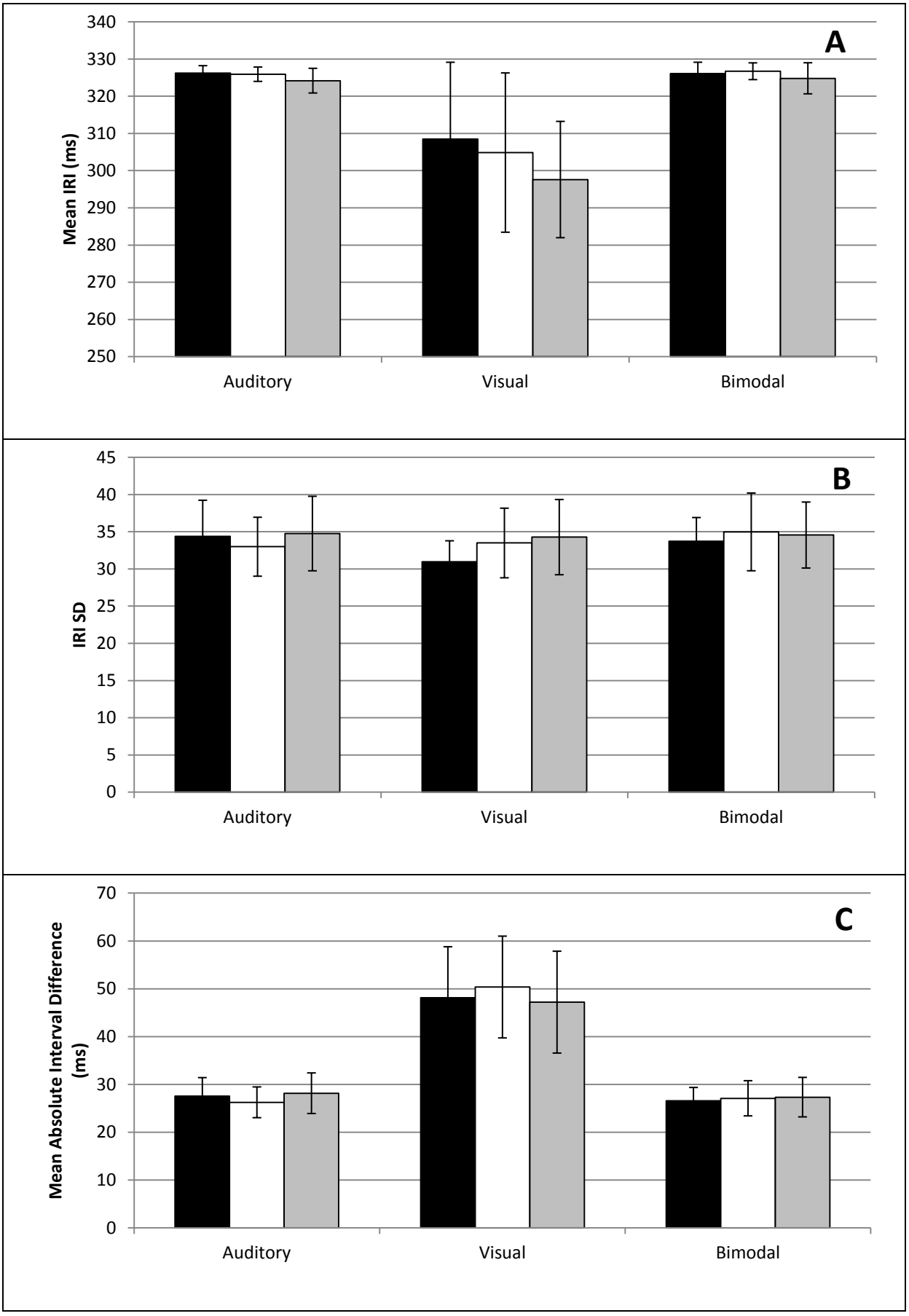

Figure 6.3: Synchronised finger tapping performance

Showing performance variables Mean IRI (A), IRI SD (B) and Absolute Interval Difference (C) across stimulus modalities and trials (trial 1-black bars; trial 2-white bars and trial 3-grey bars).

Table 6.12: Decomposed timing variance across stimulus modalities

Trial	Mean Timekeeper Variance		Mean Implementation Variance		Lag-one ACF	
	Mean (ms)	(SD)	Mean (ms)	(SD)	Mean (ms)	(SD)
Auditory	578	(389.2)	342	(280.3)	-0.3	(0.1)
Visual	897	(608.0)	171	(122.7)	-0.2	(0.1)
Bimodal	610	(414.1)	363	(217.0)	-0.3	(0.1)

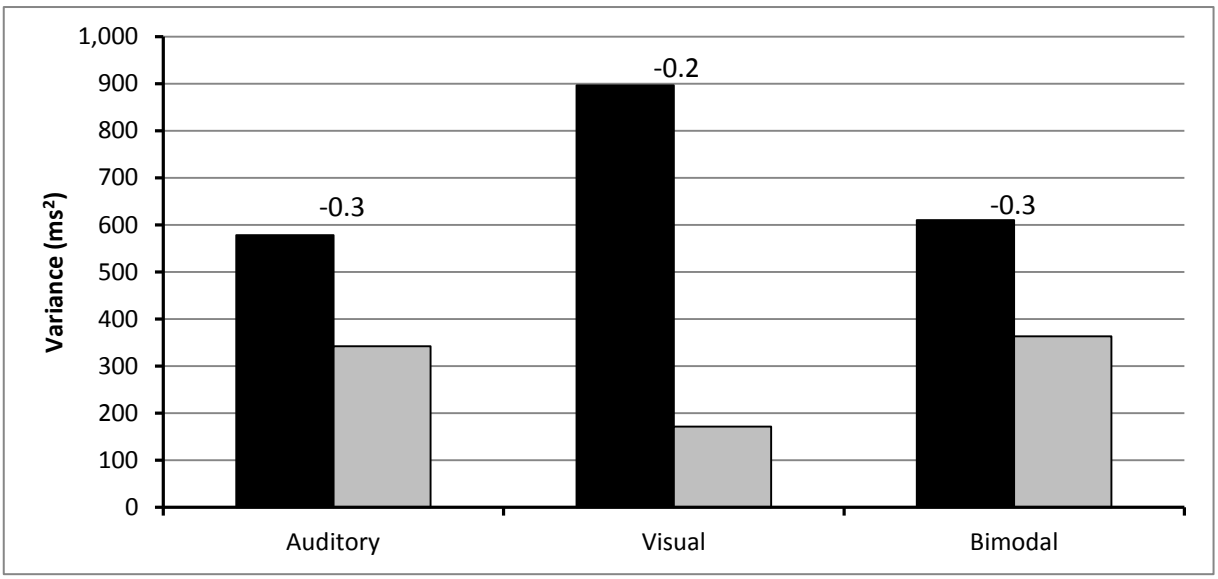

Figure 6.4: Decomposed timing variance across stimulus modalities
Decomposed variables of timekeeper variance (black bars) and implementation variance (grey bars) are shown with the autocorrelation function values (above the bars) which represents the ratio of timekeeper to implementation variance.

6.4.2.3. Correlational analyses

Differences in performance across the stimulus modalities were again examined with Pearson's product moment correlations. Outliers were identified and dealt with through procedures described in Chapter 4. In the absence of any effects of trial number and to reduce the risk of Type 1 errors that would result from a large number of comparisons the decomposed timing variables (timekeeper variance, implementation variance and ACF) were averaged across the three trials.

6.4.2.3.a. Relationships between motor timing performance and reaction time

Correlations were performed to assess the contribution of perceptual reaction time to timing performance. Bimodal timekeeper variance was found to be associated with auditory reaction time, but no other significant associations were present.

Table 6.13: Correlations between motor timing performance measures and reaction time

Trial	Auditory TK	Auditory IMP	Auditory ACF	Visual TK	Visual IMP	Visual ACF	Bimodal TK	Bimodal IMP	Bimodal ACF	Auditory RT	Visual RT
Auditory RT	-0.06	-0.13	0.13	-0.11	0.34	-0.38	-0.45*	-0.02	-0.18	-	-
Visual RT	0.03	0.04	0.22	-0.13	0.18	-0.36	-0.38	0.19	-0.33	0.68**	-
Bimodal RT	-0.06	0.10	0.20	-0.08	0.14	-0.23	-0.30	0.18	-0.22	0.79**	0.84**

*Variables include timekeeper variance (TK), Implementation variance (IMP), autocorrelation function (ACF) and simple reaction time (RT); *p<0.05, **p<0.01.*

6.4.2.3.b. Relationships between motor timing performance and cognitive/behavioural predictors

Relationships were examined between motor timing performance and predictors of symptoms of dyslexia and ADHD. Previous investigations (e.g. Holm, Ullén, & Madison, 2011; Mcauley, Jones, Holub, Johnston, & Miller, 2006) have found that reasoning ability is associated with timing performance and so reasoning ability was controlled for in partial correlations with timing performance. These are presented alongside the zero-order correlations in Table 6.14.

A large positive correlation was present between implementation variance under auditory stimulus and the measure of sustained attention (Score! subtest), with lower scores on the measure of attention being associated with decreased implementation variance. This relationship remained significant even after controlling for reasoning ability. Under auditory paced conditions neither timekeeper variance nor ACF values were associated with the psychometric variables of interest.

Reading scores were positively correlated with timekeeper variance and negatively correlated with implementation variance in the visual condition. Only the association between reading and implementation variance remained significant after controlling for reasoning ability. A moderate positive association was found between non-word reading performance and visual implementation variance, the association between non-word reading and ACF values did not reach significance (p=0.07). Neither of these associations was present after controlling for reasoning ability. Sustained attention performance was negatively correlated with visual implementation variance, and positively with the ACF variable. As the ACF provides an estimate of the ratio of implementation to timekeeper variance these results indicates that poorer sustained attention was associated with increased implementation variance overall, as well as relative to timekeeper variance. Both of these relationships survived in the partial correlations.

A significant positive correlation was found between non-verbal reasoning and bimodal timekeeper variance. An association between symptoms of hyperactivity-impulsivity and bimodal

implementation variance was present in the zero order correlations, but did not survive when reasoning ability was controlled for in the partial correlations.

6.4.2.3.c. Regression analyses

To evaluate the importance of these different timing variables as predictors of the psychometric variables of interest, a series of linear multiple regressions were performed. Outliers were accounted for following the procedures described in Chapter 4. Multivariate outliers were assessed using mahalanobis distances and these are described if present. In each of the regressions, the contribution of reasoning ability was first controlled by entering the non-verbal and verbal reasoning at the first step in each equation.

The first regression analysis examined the effect of the decomposed timing variables in the visual stimulus modality on reading ability. Reading ability was entered as the dependent variable with reasoning ability, visual implementation and timekeeper variance entered in a fixed order, three-step model. The reasoning measures did not predict reading scores at step one (r^2=0.10, $F_{(2,47)}$=2.60, p=0.09), but after controlling for reasoning ability, visual implementation variance entered at step two was a significant predictor (Δr^2=0.15, β=-0.40, $t_{(46)}$=-3.04, p<0.01) and this model accounted for 20% of the variance in reading accuracy scores (r^2=0.25, $F_{(3,46)}$=5.13, p<0.01). In contrast, timekeeper variance was not a significant predictor when entered after reasoning ability at step two (β=0.04, $t_{(46)}$=0.27, p=0.79) or in the presence of implementation variance at step three (β=-0.15, $t_{(45)}$=-1.09, p=0.28). With both variables included at step three, the equation remained significant but did not explain more variance in reading performance beyond that contributed by visual implementation variance alone (Δr^2=0.02, $F_{(4,45)}$=4.16, p<0.01).

The second multiple regression analysis evaluated the proportion of variance in sustained attention that was predicted by visual ACF, auditory implementation variance and visual implementation variance. The ACF value is however calculated from the ration of implementation timekeeper variances and so visual ACF and visual implementation variance were, as expected, highly correlated with each other. The contribution of timekeeper variance to the ACF value will act to alter its relationship with the dependent variable. Thus, the two measures of visual performance were entered into separate regression analyses as independent variables along with auditory implementation variance. In each analysis reasoning ability scores were first entered at step one and then the two predictors were entered step-wise into the equation in order of strength of association with the dependent variable.

Table 6.14: Correlations between motor timing performance and measures of literacy and attention

	Auditory TK	Auditory IMP	Auditory ACF	Visual TK	Visual IMP	Visual ACF	Bimodal TK	Bimodal IMP	Bimodal ACF	Reading	Spelling	Non-word Reading	Irregular-word Reading	ADHD-IA	ADHD-HI	Opposite World Increase	Score!	Verbal Reasoning	Non-verbal Reasoning
Auditory TK	-	0.03	0.63**	0.24	0.04	0.13	0.28	0.38	0.00	-0.02	-0.07	0.29	0.28	-0.06	0.16	0.15	0.00	0.02	0.23
Auditory IMP	-0.01	-	-0.53*	0.18	-0.19	0.29	0.07	0.74**	-0.41	-0.01	-0.08	0.04	-0.04	0.20	0.41	0.09	0.51*	0.03	0.20
Auditory ACF	0.67**	-0.57*	-	0.18	0.04	0.04	0.36	-0.19	0.42	0.32	0.32	-0.13	0.01	-0.23	-0.05	0.06	-0.21	0.28	0.10
Visual TK	0.24	0.17	0.09	-	-0.62**	0.76**	0.25	0.25	0.07	0.44[A]	0.25	-0.25	-0.37	-0.01	0.22	0.34	0.36	0.34	0.15
Visual IMP	0.03	-0.20	0.12	-0.59**	-	-0.79**	-0.20	-0.18	-0.08	-0.53*	-0.35	0.43[A]	0.27	-0.18	-0.14	0.06	-0.49*	-0.26	-0.02
Visual ACF	0.18	0.33	-0.05	0.74**	-0.77**	-	0.26	0.29	0.13	0.39	0.25	-0.40	-0.37	0.06	0.25	0.11	0.65**	0.31	-0.03
Bimodal TK	0.21	-0.04	0.32	0.14	-0.18	0.27	-	-0.13	0.68**	0.27	0.09	-0.19	-0.24	0.01	0.29	0.05	0.25	0.36	0.55*
Bimodal IMP	0.36	0.73**	-0.20	0.26	-0.20	0.34	-0.24	-	-0.70**	-0.09	-0.18	0.29	0.31	0.27	0.43*	-0.01	0.39	-0.01	0.14
Bimodal ACF	-0.05	-0.47*	0.39	0.00	-0.05	0.10	0.67**	-0.75**	-	0.32	0.32	-0.37	-0.41	-0.33	-0.18	-0.04	0.05	0.20	0.23
Reading	-0.08	-0.06	0.19	0.30	-0.51*	0.27	-0.02	-0.13	0.24	-	0.81**	-0.71**	-0.53*	-0.18	-0.10	-0.04	0.30	0.68**	0.31
Spelling	-0.14	-0.16	0.21	0.08	-0.27	0.13	-0.25	-0.23	0.24	0.72**	-	-0.66**	-0.61**	-0.33	-0.18	0.14	0.00	0.53*	0.32
Non-word Reading	0.31	0.04	-0.02	-0.14	0.37	-0.31	-0.11	0.30	-0.35	-0.67**	-0.60**	-	0.77**	0.27	0.12	0.00	-0.37	-0.40	-0.05
Irregular-word Reading	0.37	0.01	0.09	-0.31	0.24	-0.37	-0.07	0.36	-0.36	-0.50*	-0.56*	0.79**	-	0.45*	0.25	-0.23	-0.16	-0.25	-0.28
ADHD-IA	-0.05	0.21	-0.26	-0.03	-0.17	0.04	-0.01	0.27	-0.35	-0.29	-0.42	0.33	0.49*	-	0.79**	-0.07	0.05	0.06	0.00
ADHD-HI	0.13	0.39	-0.14	0.15	-0.09	0.22	0.15	0.44	-0.29	-0.43	-0.43	0.25	0.39	0.82**	-	0.13	0.21	0.26	0.25
Opposite World Increase	0.18	0.11	0.00	0.30	0.13	0.03	0.02	0.01	-0.08	-0.26	0.05	0.11	-0.21	-0.09	0.10	-	-0.41	0.21	-0.02
Score!	0.01	0.53*	-0.27	0.33	-0.47*	0.65**	0.27	0.41	0.03	0.27	-0.09	-0.33	-0.14	0.04	0.19	-0.46*	-	0.16	0.01
Verbal Reasoning																			0.33

*Pearson's product moment correlations (top right) between motor timing performance and psychometric variables of interest, with partial correlations controlling for verbal and non-verbal reasoning (bottom left). Variables include timekeeper variance (TK), Implementation variance (IMP) and Autocorrelation Function (ACF); *p<0.05, **p<0.01, [A]p=0.05.*

The reasoning measures did not predict sustained attention scores at step one (r^2=0.03, $F_{(2,18)}$=0.25, p=0.79), but after controlling for reasoning ability, the visual ACF variable entered at step two was a significant unique predictor of sustained attention (Δr^2=0.41, β=0.68, $t_{(17)}$=3.48, p<0.01), accounting for 33% of variance in sustained attention (r^2=0.43, $F_{(3,17)}$=4.31, p<0.05). The model remained significant with the inclusion of auditory implementation variance as a predictor at step three ($F_{(4,16)}$=4.77, p<0.01) and accounted for a further 10% of the variance in sustained attention, although auditory implementation variance was not a significant unique predictor (Δr^2=0.11, β=0.36, $t_{(16)}$=1.98, p=0.07). When auditory implementation variance was entered first at step two, the model was not significant (Δr^2=0.28, $F_{(3,17)}$=2.49, p=0.10). When the analysis was repeated with visual implementation variance entered instead of visual ACF at step two, the model was not significant (Δr^2=0.18, $F_{(3,17)}$=1.85, p=0.17). The inclusion of auditory implementation variance at step three produced a significant model (Δr^2=0.20, $F_{(4,16)}$=3.15, p<0.05). For this second analysis, auditory implementation variance was the only unique predictor at step three, predicting 30% of the variance in sustained attention (r^2=0.44, β=0.46, $t_{(16)}$=2.36, p<0.05).

These analyses indicate that reading ability is related to visual implementation variance, whereas sustained attention is related more to the relative proportions of implementation variance and timekeeper variance under visual timing conditions.

6.4.3. Discussion

6.4.3.1. Modality effects

For this sample of typically developing children the modality of the pacing stimulus had no effect on mean IRI or IRI variability during a motor synchronisation timing task. An effect of modality was found for the mean absolute interval difference variable due to the higher IRI-IOI asynchrony found in the visual mode compared to the auditory mode. Thus, like the adults tested in Experiment 1, the children were able to maintain a regular response interval across the three conditions but tapped further from the beat with visual pacing stimuli, suggesting a difference in synchronisation behaviour in this condition. The decomposition of variance into timekeeper and implementation variance demonstrated that under auditory pacing, timekeeper variance was lower than under visual pacing, with timekeeper variance in the bimodal conditions intermediate to the two (but not significantly different from the visual trials). This result is in contrast to the adults in Experiment 1 for whom timekeeper variance did not vary across the different modalities. In addition, like the adults, the children showed lower implementation variance in the visual condition. Together these data suggest that these children used a rather different method of timing control than the adults across the different pacing conditions. It is unlikely that the differences found across modalities resulted from a basic difference in perception of the stimuli because the reaction time results did not follow the same pattern across modalities, with no significant difference found between the auditory and visual

conditions, and faster responses with the bimodal stimuli. For the timing measures the bimodal condition was intermediate to the other two conditions.

Previous research has suggested that under visually paced timing conditions participants select and implement a regular response output based on a perceived impression of the stimulus IOI, irrespective of the information available to them that might improve response accuracy such as the asynchronies between stimuli and responses that provide feedback on temporal performance (Jäncke, Loose, et al., 2000; Kolers & Brewster, 1985). This response strategy has been called a stereotypical motor response (Kolers & Brewster, 1985) and the hypothesis is supported by converging evidence from behavioural and brain imaging experiments. For example, errors in timing performance are typically neither noticed nor corrected under visually paced conditions, but are corrected effectively when performance is paced by auditory stimuli (Kato & Konishi, 2006; Kolers & Brewster, 1985). Furthermore, neural areas engaged in internal rhythm guidance, updating motor responses and recalibrating sensory-motor coupling (such as the ventral pre-motor cortex, left inferior parietal lobule, right inferior cerebellum, left thalamus and supplementary motor area) are particularly active in auditory timing tasks but less so when timing is visually paced (Jäncke, Loose, et al., 2000; Jantzen et al., 2005). Under visual timing conditions areas associated with imagination (such as the right superior cerebellum, vermis and right inferior parietal lobule) are more active.

Adopting a stereotyped motor response would be expected to reduce implementation variance relative to timekeeper variance. The decomposed variance estimates for the adults and children in the visual condition were consistent with this explanation. Implementation variance was lower and timekeeper variance was higher when pacing stimuli were presented visually compared to the other conditions. This pattern of results means that the validity of using visually paced timing tasks in studies assessing timing behaviour is questionable because it cannot be assumed that responses are being generated by the timekeeping system in response to the perceptual information being received. Instead responses in visual timing tasks reflect the stereotyped responses reliant on mechanisms other than the entrainment of the timekeeping system. Visually paced tasks may fail to adequately assess the internal timekeeping capacities that are of most interest when assessing the putative differences in temporal processing in the clinical populations of interest.

In contrast to the results obtained with visual stimulation, participant responses in the auditory conditions were characterised by lower estimates of timekeeper variance, coupled with higher estimates of implementation variance. This confirms that children, like adults (Fendrich & Corballis, 2001; Kolers & Brewster, 1985; McAuley & Henry, 2010), have relatively invariant output from timekeeper mechanisms when synchronising motor responses with auditory stimuli. This result reinforces evidence for the higher temporal precision of the auditory system compared to the visual system (Fendrich & Corballis, 2001; McAuley & Henry, 2010) and demonstrates the importance of

considering task parameters in research of this kind. Studies that have employed visual or bimodal auditory-visual stimuli (Ben-Pazi et al., 2003; Rubia et al., 2003; Toplak & Tannock, 2005b) may have underestimated the true capacities of central timing processes. When considering temporal processing in developmental populations, these results suggest that more confidence can be placed in results from auditory stimulated motor timing tasks (e.g. Badian & Wolff, 1977; Klipcera et al., 1981; Thomson et al., 2006; Thomson & Goswami, 2008; Waber et al., 2001; Wolff, Melngailis, Obregon, & Bedrosian, 1995; Wolff, 2002) than similar visual paradigms.

The absence of an effect of modality on IRI variability (SD of IRIs) may indicate that the stereotypical response behaviour was more successful in maintaining a regular output than the strategy employed by the adults. An effect of modality on IRI variability was expected given previous findings in a number of studies with samples of adults (Grahn, 2012; Jäncke, Loose, et al., 2000; Jantzen et al., 2005; Kolers & Brewster, 1985; Loras et al., 2012; Patel et al., 2005; Semjen & Ivry, 2001; Stauffer et al., 2012). An alternative explanation is that in the visual trials, the participants remembered the response rate from the auditory or bimodal trials, although the randomisation of the different blocks of trials and inclusion of distracter trials should have prevented this from occurring. However, studies where visual tasks have been used in isolation to assess timing in children with developmental disorders, with no other cues to the stimulus rate (Rubia, Taylor, et al., 1999; Rubia et al., 2001, 2003) may have been measuring the ability of participants to gauge the stimulus rate from the visual stimulus and then to establish a stereotyped response output, ignoring subsequent visual stimulus to maintain that rate. Thus, the recorded performance may be an indicator of participant's perceptual ability to determine the stimulus rate from the unreliable visual stimuli in addition to the functioning of the internal timekeeping system. There is some evidence that the visual task may reflect timekeeper performance, with similar between group differences found between children with and without ADHD for both self-paced finger tapping (in the absence of a stimulus) and a visual synchronisation task (Rubia et al., 2003).

When the correlations between the decomposed variables in the different modalities were considered the children showed an association between the bimodal and auditory implementation variance estimates. As proposed earlier, the adults appeared to be attempting to use the visual stimuli to guide responses, despite this being an ineffective strategy. The correlation found for the adults between bimodal and visual implementation variance estimates may therefore be due to their attempts to make use of the visual stimuli in both the visual and bimodal conditions at a cost to implementation variance. The behaviour of the children in the bimodal trials was, in contrast, more like that in the auditory condition as if the visual stimuli were not used in favour of the acoustic tones in both conditions. Studies conducted previously using bimodal stimuli to investigate timing performance in children with ADHD (Ben-Pazi et al., 2003, 2006) may have therefore measured

timing performance which resembles that on auditory-alone tasks. However the additional processing demands of bimodal tasks described by numerous authors still indicate that bimodal timing performance is not equivalent to the output resulting from acoustically paced tasks (Droit-Volet et al., 2004; Elliott et al., 2010; Fendrich & Corballis, 2001; Patel et al., 2005; Penney et al., 2000; Recanzone, 2003; Roach et al., 2006, 2011; Wada et al., 2003). Such tasks are therefore not recommended for use in assessing the basic underlying capabilities of the timing system in children with disorders such as dyslexia or ADHD.

6.4.3.2. Relationships between motor timing performance and cognitive/behavioural predictors

Measures of timing performance have previously been found to explain unique variance in measures of underlying neuro-cognitive impairments in developmental disorders (Rubia et al., 2009; Thomson & Goswami, 2008; Wolff, 2002; Zelaznik et al., 2012). In this study, the relationships were therefore assessed between timing performance and measures of reading and attention that tap the core behavioural symptoms upon which developmental dyslexia and ADHD are diagnosed. These two disorder phenotypes have been repeatedly studied with measures of interval timing, with visual tasks often used in investigations of ADHD (Ben-Pazi et al., 2003; Rubia et al., 2003; Rubia, Overmeyer, et al., 1999; Toplak & Tannock, 2005b) and auditory tasks most frequent in studies of dyslexia (Thomson et al., 2006; Wolff et al., 1984; Wolff, Michel, Ovrut, et al., 1990). Here, sustained attention and reading ability were statistically associated with the decomposed variance estimates in the visual modality (visual ACF and visual implementation variance).

Participants with low scores on the cognitive dimensions of reading and attention had relatively larger estimates of implementation variance on the visually paced task. More specifically, the regression analyses indicated that reading was associated with implementation variance (the degree to which the stereotypical response pattern was used) and that sustained attention was more closely related to the autocorrelation function (the ratio between timekeeper and implementation variance or the balance between entrainment and use of the stereotypical response set). Under visually paced conditions, timing performance may lack precision, effects that are hypothesised to result from a combination of the inefficiency of the visual control mechanisms for generating internal rhythms (Grahn, 2012; Jäncke, Loose, et al., 2000) and the poorer functional coupling of these mechanisms with the motor system (Jäncke, Loose, et al., 2000; Jantzen et al., 2005). As described above, a stereotyped, motor-focused strategy has been suggested to be the most efficient approach to such tasks (Jäncke, Loose, et al., 2000; Kolers & Brewster, 1985) and would be predicted to result in reduced implementation variance. Our data suggest that children with lower scores on reading and attention measures do not consistently implement such a strategy on a visually paced task. It is possible that these children failed to take account of the inadequacy of these timing stimuli and instead show higher implementation variance.

In order to generate an isochronous stereotyped response set, feed-forward prediction of temporal events is required (Arnal & Giraud, 2012; Fraisse, 1984; Macar & Vidal, 2009). This entrainment is especially important under conditions such as the visual task where timing behaviour is being challenged. The children with lower scores did not appear to ignore the more challenging visual pacing stimuli in favour of a stereotyped response set, and did not entrain to the stimulus rate, resulting in higher implementation variance in order to achieve the same level of IRI variability as other children. This result is similar to the findings of Smits-Engelsman and colleagues who found that children with learning difficulties did not alter their response strategy when task demands changed during a movement task (Smits-Engelsman, Wilson, Westenberg, & Duysens, 2003). Comparatively, the control children in this study were able to use feed-forward prediction and this was thought to be due to the presence of an appropriate internal representation of features of movements. In the present study, the children with lower scores on the measures of attention and reading may have lacked the ability to build an appropriate anticipatory model of the timed movement demands and use this for prediction and entrainment of responses under the challenging conditions of the visual task. The specific demands of the visual timing task may elicit reallocation of cognitive resources to facilitate task completion (Droit-Volet et al., 2004; Heron, Roach, Whitaker, & Hanson, 2010). The correlations between timing performance and reading and sustained attention variables may therefore be similar to those that might be predicted for a measure of processing speed (McGrath et al., 2011).

In both experiments described, the children and adults who showed an atypical performance strategy on the visual task (compared to their peers) were those with poor performance on the sustained attention or Stroop tasks. For adults, this was those participants who ignored stimuli in favour of a steady response output and for the children this was those who attempted to use the stimuli to guide responses. These measures of attention are not necessarily equivalent measures of resource allocation but do indicate that the visual task likely interacts with attention abilities. In Experiment 2 the children with low scores on the sustained attention measure also showed lower implementation variance on the auditory trials, a timing strategy that differed from other children on a task where internal rhythm generation and reduced timekeeper variance are expected, rather than a motor-focused response (Jäncke, Loose, et al., 2000). The dissociations between the visual and auditory tasks question the suitability of the visual task for studies assessing timing mechanisms.

Correlations between bimodal performance and the cognitive or behavioural predictors were not apparent in this study. Earlier studies have described how bimodal tasks require the resolution of auditory-visual stimuli in order to achieve synchronous behaviour (Roach et al., 2006), as described in Chapter 2. Consequently, previous evidence of correlations between bimodal timing performance and attention (Ben-Pazi et al., 2003; Rubia et al., 2003) may only reflect a participant's ability to

resolve auditory and visual stimuli rather than their timing ability per se. The need for dynamic attention in assimilating these multi-source stimuli (Droit-Volet et al., 2004; Elliott et al., 2010; Fendrich & Corballis, 2001; Heron et al., 2010; Mates, 1994; Nozaradan et al., 2012) may have contributed to these associations. Future research may be useful to interrogate the extent to which children with ADHD are affected by the additional processing demands of bimodal stimuli by testing uni-modal auditory and visual stimuli alongside the bimodal stimuli and by manipulating the delay between the auditory and visual pacing stimuli under bimodal conditions (for example following the methodology of Elliot et al., 2010).

Although reading was associated with visual performance measures, correlations were expected between auditory timing and measures of literacy ability, given the evidence for motor timing deficits in children who are poor readers described in Chapter 3. In this sample of typically developing children there may have been insufficient variation on the literacy measures to give rise to such correlations. In addition, the tapping rate used here was faster than that used in experiments where reading or spelling performance has been found to be associated with performance at rates of 1.5-2.5Hz (Thomson et al., 2006; Thomson & Goswami, 2008).

6.5. General Discussion

Motor timing tasks provide clinically useful measures of temporal processing, which may help researchers to understand the mechanisms contributing to the aetiology of developmental disorders such as ADHD and dyslexia for which deficits in implicit and explicit timing functions are a common feature (Farmer & Klein, 1995; Goswami, 2011; Rubia et al., 2009; Toplak et al., 2006). The increased recognition that risk factors for developmental disorders are expressed continuously in the population (Pennington, 2006) underpins the need to establish the extent of dimensional overlap between disorder phenotypes on traits such as temporal processing. Previous studies of motor timing have, however, presented stimuli via different stimulus modalities, with interval timing in ADHD assessed primarily using visual pacing stimuli and in developmental dyslexia with auditory stimuli. Here a comprehensive assessment of such stimulus modality effects on paced motor timing performance in adults and children has been provided, within the context of relationships with measures of literacy and attention variables. The addition of a variance decomposition method enabled evaluation of the sources of variability in finger tapping performance in children and to assess the contribution of these different underlying mechanisms to literacy ability and attention skills. An adaptation of the Wing-Kristofferson model (Kooistra et al., 1997) enabled separation of components that reflect the variability of both the on-going timekeeping system and the motor implementation of timing signals.

For both the adults and children in Experiments 1 and 2 the data confirmed the hypothesis that finger tapping performance is strongly modulated by the modality of the pacing stimuli. Decomposition of variance components using the Wing-Kristofferson methodology confirmed differences in the strategies used to complete the timing task under the different pacing conditions. The statistical associations between timing performance and indices of cognition and behaviour provide further support for the importance of the differences between timing assessed with visual and auditory pacing stimuli. In visual conditions, synchronisation of outputs with stimuli may be more difficult due to the lack of accurate temporal information available to a timekeeping mechanism (Repp & Penel, 2002), resulting in poorer precision of the clocking output for implementation (Kolers & Brewster, 1985). In order to overcome such inaccuracies, beat-entrained response strategies may be used instead of using feedback from the on-going stimuli. In bimodal trials, there are additional processing steps required to resolve the auditory and visual stimuli into one percept for use in timing motor responses.

These results pose some problems for previous studies which have used visual or bimodal tasks to assess motor timing performance in children with ADHD (Ben-Pazi et al., 2003, 2006; Rubia et al., 2003) and indicate that auditory tasks are most appropriate for assessing timing performance. The interactions between timing performance and sustained attention, on both the auditory and visual tasks, suggest that measures of attention should be included in future studies to confirm the extent to which attention and literacy are separately associated with timing performance. Children with attention difficulties have sometimes (Thomson et al., 2006; Thomson & Goswami, 2008; Wolff, 2002) but not always (Badian & Wolff, 1977; Klipcera et al., 1981; Wolff, Michel, Ovrut, et al., 1990) been excluded from studies of timing in dyslexia. The data presented here indicate, however, that non-clinical symptoms of ADHD may still affect performance and so exclusions may not be a sufficient substitute for measuring attention related abilities.

The results above provide some evidence that the bimodal task may give similar results to the auditory task, however, given the additional processing required to resolve such stimuli, auditory stimuli should be used where possible. If it is necessary to include visual stimuli to engage children in the task then stimuli should contain spatial or motion cues which can improve beat-based encoding (Grahn, 2012). In the experience of the author, most children are however, easily engaged by auditory motor timing tasks. Further to this, timing abilities could be measured on continuation tasks which assess timing once the stimulus train has ceased. This task allows measurement of timing capabilities in the absence of any modality effects and studies where synchronisation and continuation phases have been used indicate that the modality of stimuli in the synchronisation phase has no effect on the performance in the continuation phase (Y. Chen, Repp, & Patel, 2002; Semjen & Ivry, 2001).

The results here demonstrated main effects of modality on a timing task performed at 329ms intervals. There have been suggestions that a 3Hz tapping speed may be too fast for participants to produce time series suitable for analyses (Kurgansky & Shupikova, 2011; Repp, 2003). We implemented this particular speed of finger tapping as it was previously used in a study of finger tapping in children with ADHD (Ben-Pazi et al., 2003, 2006). Repp (2003) deemed that trials that were unsuitable for analysis were those with asynchrony standard deviations exceeding 16.7% of the IOI. Here, trials were, on average, within these strict criteria, suggesting that children were able to maintain the stimulus rate appropriately.

6.5.1. Limitations

It has been noted that associations were found between behavioural measures of attentional control (Stroop and Score! performance) and measures of timing performance for both the children and adults. However, these relationships were found amongst multiple pairwise comparisons which give rise to a high chance of Type I errors being made (where a true null hypothesis is incorrectly rejected). For example with an alpha value of 0.05, 5% of comparisons would be expected to be significant by chance even if all the null hypotheses were true. As such, the small number of significant correlations found in the present experiments may fall into this category. It is therefore wise to exercise caution in interpreting these findings and to acknowledge the limitations of this statistical analysis method.

One method of adjusting for the use of multiple comparisons is to apply Bonferroni correction to the accepted p-value: dividing the p-value by the number of tests completed to give an adjusted value against which significance values can be evaluated. Applying this strategy to the present data would result in no correlations being significant at the corrected level, given the large number of comparisons in the correlation matrices. This method of correction may, however, be overly conservative for the present investigation where some of the measures are mutually correlated due to the overlap in the skills they assess (e.g. reading, spelling, non-word reading and irregular word reading scores). Some correlations may therefore come from the same family of comparisons (McDonald, 2009) and applying the Bonferroni correction may increase the risk of Type II errors (i.e. false negatives).

The statistically significant associations that were found (between attentional control and measures of the proportion of implementation variance on the visual task only) were present for both samples of adults and children. This suggests that the findings may have statistical relevance, indicating that attentional control is an important contributor of variance to visual timing performance. Nevertheless, the reliability of these findings remains to be tested via further experiments in which only seek to test this proposed association between visual timing and attentional control; in particular the use of different timing strategies by those with attentional failures. In the present

study, where hypotheses were being generated regarding the putative relationships between aspects of timing and cognitive performance, a less cautious approach to the issue of multiple comparisons was justified (Perneger, 1998).

6.5.2. Validity of the Decomposition Model Applied

As applied in this study, the introduction of time series analysis to data obtained from motor timing tasks has helped to provide a richer account of the potential links between individual variability in timing performance and in the cognitive dimensions that underlie highly prevalent developmental disorders. The Wing-Kristofferson model (Wing & Kristofferson, 1973a) has been applied previously to data obtained from other clinical populations with varying results (Duchek et al., 1994; O'Boyle et al., 1996; Pastor et al., 1992). These studies have demonstrated the limitations of this approach for the analysis of data with very large IRIs, linear trends or negative variance estimates. Such parameters would not be unexpected in data derived from clinical groups, compared to that obtained from highly practiced individuals. In the present study, the proportion of trials where the resulting data did not satisfy the assumptions of the model (see Chapter 5) in both groups of participants was modest in our sample compared to that reported in clinical groups (O'Boyle et al., 1996; Pastor et al., 1992). However, this proportion may be expected to be higher in studies of children with developmental disorders and so further examination of these limitations is required and is provided in the next chapter (Chapter 7).

Whilst such difficulties are present, they do not pose insurmountable problems for interpretation and instead, data sets from atypical populations can provide additional information that is useful for understanding the nature and extent of timing deficits in clinical or developmental populations (Madison, 2001a; Pastor et al., 1992; Zelaznik et al., 2012). Here the adjusted model developed by Kooistra et al. (1997) and validated by Kampen and Snijders (2002) was used to attempt to address some of these potential causes of violations.

Linear drift was present in the time series recorded, but the coefficients did not differ significantly from zero. The Kooistra model however, only accounts for the presence of linear drift in the time series and so the drift parameter may have been insufficient to identify other forms of drift which could influence variance estimates (Collier & Ogden, 2004; Jucaite, Forssberg, Dahlstrom, & Madison, n.d.; Madison et al., 2009; Madison, 2001b). It is also possible that the assumption (of the Wing-Kristofferson model) of statistical dependence in the time series at lags beyond one did not hold true, such that the participants may not have been controlling timing with an open-loop mechanism, but instead with reference to previous taps at lags greater than one (as found in a recent study of children with ADHD; Zelaznik et al., 2012).

Within the model, negative variance estimates were also corrected to zero; a common, conservative, strategy in experiments of this kind (Collier & Ogden, 2004; Harrington et al., 1998; Ivry & Keele, 1989; Kooistra et al., 1997; O'Boyle et al., 1996). However, the potential presence in the data of drift or dependence at lags beyond one may cause the underestimation of implementation variance or overestimation of timekeeper variance (Collier & Ogden, 2004; O'Boyle et al., 1996). An analysis conducted by O'Boyle et al. (1996) showed that when different strategies for dealing with negative violations were compared (setting implementation variance to zero versus the selection of only non-violating trials), the effect sizes for the experimental comparisons between groups were not substantially different. Therefore the present results would likely hold even if a more conservative approach to violations of the model assumptions had been taken.

6.6. Conclusion

The results described above highlight the differences in the way auditory and visual tasks are processed behaviourally as well as differences in the way in which performance on temporal processing tasks correlates with cognitive constructs associated with highly prevalent disability phenotypes. While the use of visual timing tasks may ultimately be useful for demonstrating the type of processing difficulties experienced by children with attention deficits or reading difficulties, such measures may not adequately assess the timekeeping capability of central neural mechanisms. The evidence suggests that central timekeeping mechanisms may be more accurately assessed with auditory paced tasks. The results highlight the methodological importance of assessing the construct of attention in temporal processing tasks (Heron et al., 2010; Zelaznik et al., 2012), particularly in clinical populations where attention difficulties often co-occur with the primary diagnostic symptoms (Pauc, 2005; Willcutt & Pennington, 2000).

7. Assessing the Validity of the Wing-Kristofferson Model

7.1. Chapter overview and introduction

In Chapter 6 the Wing-Kristofferson model was found to be useful for separating out the components of variance which contribute to differences in timed responses across different experimental conditions. The adjusted Wing-Kristofferson model provided by Kooistra et al. (1997) was used in order to attempt to account for some of the potential causes of violations of the assumptions for an autocovariance model of this kind. However, as described in Chapters 5 and 6 and in several previous studies (Ivry & Keele, 1989; O'Boyle et al., 1996; Pastor et al., 1992), the Wing-Kristofferson model can over- or underestimate variance components in the presence of drift in the time series or dependencies between variance components. In the next chapter the Wing-Kristofferson model will be applied to data from a population of children with reading difficulties (Chapter 8) and therefore further assessment of the validity of the model is warranted. Presently, the causes of violations of the model assumptions are examined together with methods of dealing with these potential problems. The analysis is conducted using time series data collected from an additional sample of adult participants.

As described in Chapter 5, the Wing-Kristofferson model posits that the lag-one autocorrelation should fall between the bounds of zero and minus one-half (Equation 8: $\rho_I(1) = -1/2 + \left(\sigma_T^2 / \sigma_P^2\right)$). Some violations of this assumption will inevitably occur by chance because the tails of the distribution of variance estimates can fall below zero resulting in (statistically impossible but theoretically possible) negative variance estimates (Collier & Ogden, 2004; Kooistra et al., 1997). In addition, these bounds may be violated if: (a) the variance components are not independent at lags beyond one or (b) the time series are not stationary. The contribution of these factors was assessed using the methods described in Section 7.1.1.

7.2. Sources of model violations

7.2.1. Autocovariance for lags beyond one

If data conforms to the open-loop method of timing described by Wing & Kristofferson (1973) the observed estimates of autocovariance at lags beyond one should be equal to zero ($\gamma(k) = 0$ for all k>1). Open-loop timing means that responses are updated only with respect to neighbouring responses at lag one, but further reference or feedback from responses at greater lags is not used to improve the accuracy of performance (closed-loop timing). To test for the presence of open-loop timing, averaged autocovariance values at lags two to five can be calculated and compared to predicted unbiased estimates of autocovariance at each lag (Collier & Ogden, 2004; Diggle, 1990; Vorberg & Wing, 1996). The model is taken to be violated if the expected unbiased values of

autocovariance do not fall within the 99% confidence intervals of the observed (biased) mean autocovariance. The observed values are considered biased because they are drawn from a limited set of observations. They are therefore compared to a value corrected for the number of responses collected (i.e. 30 finger taps), rather than comparing them to zero (given that $\gamma(k) = 0$ for all $k > 1$). This adjustment for sample size was described by Vorberg and Wing (1996, eqn. 13, pp. 195):

$$E[\hat{\gamma}(j)] = -\left(1/N\right)\gamma(0) - \left(2/N\right)\left\{1 - j/[N(N-1)]\right\}\gamma(1) \qquad j > 1 \qquad \textit{(Equation 12)}$$

where j is the lag (typically lags 2 through to 5 are examined), $E[\hat{\gamma}(j)]$ is the expected value of autocovariance at that lag, N is the number of responses in the trial and $\gamma(j)$ is the observed autocovariance at that lag. The assumption that $\gamma(k) = 0$ for all k>1 is taken to be violated if the expected value of autocovariance does not fall within the 99% confidence intervals of the observed mean autocovariance.

7.2.2. Stationarity

The Wing-Kristofferson model is also based on the assumption that time series are stationary. The covariance function should therefore depend only on the lag, not on time (Diggle, 1990). As observed in the previous chapter (Chapter 6), linear drift in the time series can be measured by plotting a linear regression line for inter-response intervals against tap number, yielding a slope value and a measure of the proportion of the total variance accounted for by the regression line (r^2). In Chapter 6 the slope of this regression line was incorporated into the calculation of the timekeeper and implementation components (following Kooistra et al., 1997). By assuming that that any drift in the time series is linear this approach may fail to quantify non-linear forms of drift (Collier & Ogden, 2004; Jucaite et al., n.d.; Madison et al., 2009; Madison, 2001b) and so a method was sought which would allow analysis of non-linear forms of drift.

Madison (2001a, 2001b) devised a method of assessing the development of drift over time using a non-parametric estimator which makes no assumptions about the form of drift. This method assesses the absolute differences between values in the time series as a function of their lag in the series. Consider that a single response (I_j) has neighbouring responses at different lags (I_{j+k}), such that the lag-one neighbour is represented as (I_{j+1}). The absolute difference between a response interval and its neighbour at any lag (k) is:

$$\Delta I_j = \left| I_j - I_{j+k} \right| \qquad \textit{(Equation 13)}$$

So for lag-one neighbours $\Delta I_j = \left| I_j - I_{j+1} \right|$. These differences can be calculated across the time series (e.g. the lag-one neighbour for response three is response four). To provide a measure of stationarity, these differences are also calculated at all other lags, up to lag 15 (half the series length; ΔI_j for $j = 1, 2, \ldots N/2$). Thus for lag 5, the differences for each response are calculated as $\Delta I_j = \left| I_j - I_{j+5} \right|$ (the lag 5 differences for I_1, I_2, I_{15}, I_{26} are shown Figure 7.). The difference values (ΔI_j) are calculated as absolute values to avoid positive and negative differences between responses being cancelled out, allowing non-linear trends to be observed.

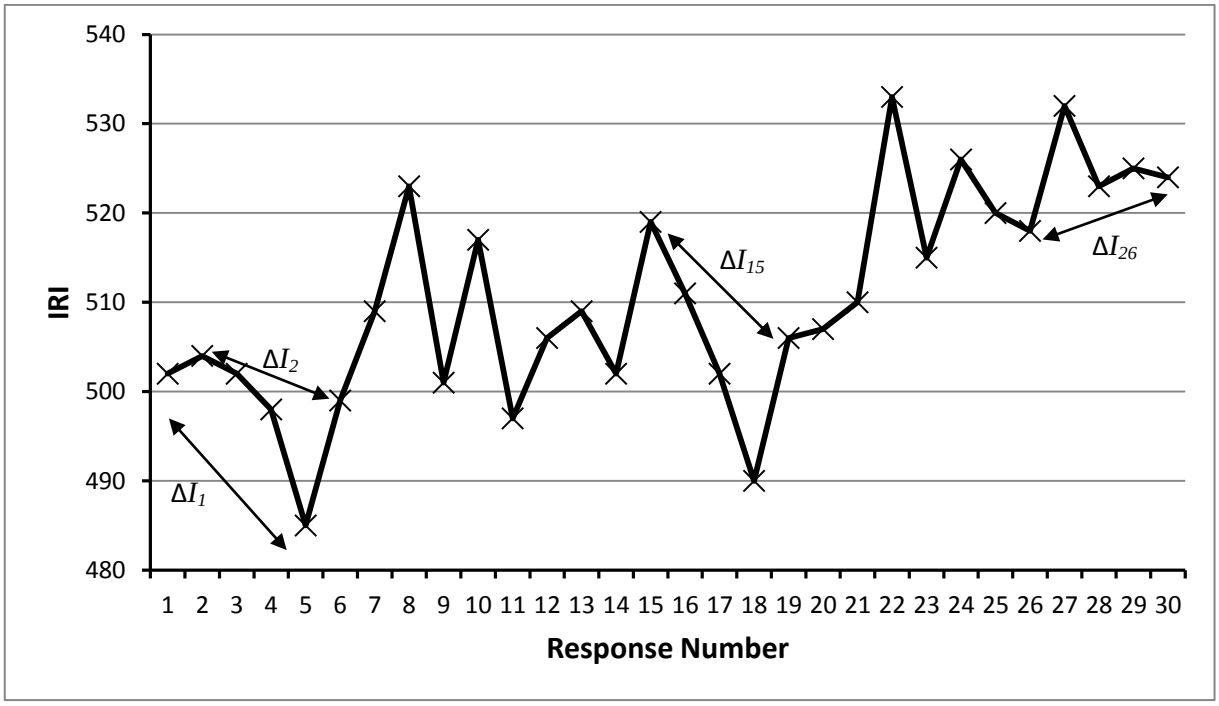

Figure 7.1: Diagram showing how differences were calculated according to Equation 13, here showing differences for lag 5 (k=5).

From: Madison, G. Variability in isochronous tapping: Higher order dependencies as a function of intertap interval, Journal of Experimental Psychology: Human Perception and Performance 27, pp.416, 2001, APA, adapted with permission.

The median of the differences at each lag is then calculated giving a vector of the median differences across lags ($\Delta_j = med\left[\Delta I_1, \Delta I_2, \ldots \Delta I_{N-j}\right]$). So for lag 5 this is calculated as $median\,(I_1 - I_{1+5,}\, I_2 - I_{2+5}, \ldots, I_{15} - I_{15+5})$. Finally these median differences (Δ_j) are plotted as a function of lag number ($f'(k)$) for lags between 4 and 15 (as shown in Figure 7.2) and a least squares regression line fitted to this graph provides a value of $f'(k)$. The limits (4 to 15) are included to avoid the small lags where variability is expected and avoids over-representation of the lags in the centre of the series which would occur if longer distances ($k > N/2$) were included (Madison, 2001b). If the data is stationary the median differences (Δ_j) should not increase as the

lag increases and so $f'(k)$ should be equal to zero. If a trend is present then a slope will be present in the function.

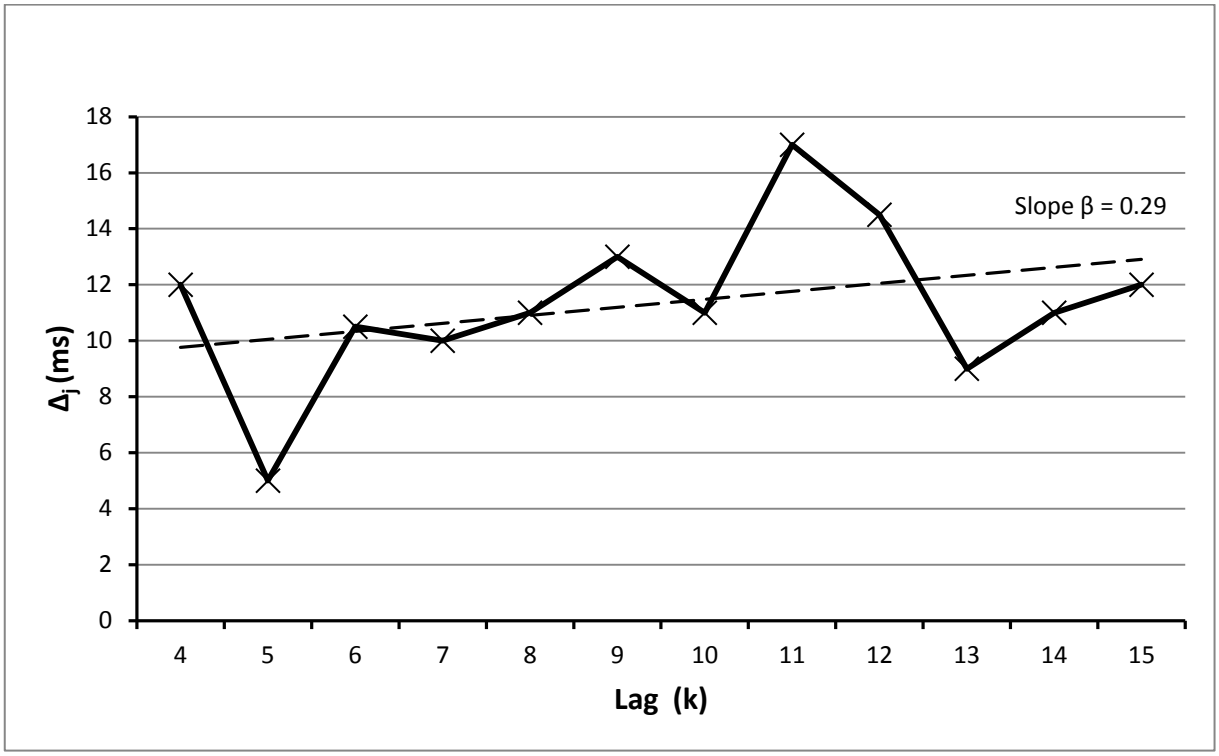

Figure 7.2: Median differences at each lag, with a least squares regression line giving $f'(k)$

7.3. Methods of dealing with violations

In previous studies that have used the Wing-Kristofferson method a number of different strategies have been used to account for potential violations of the original model. Here the effect of these different methods of dealing with violations on the resulting estimates of timekeeper and implementation variance was assessed alongside an analysis of the presence of non-stationarity and closed-loop timing. A similar assessment of such methods was undertaken by O'Boyle et al. (1996), although they did not evaluate the adjusted model derived more recently by Kooistra et al. (1997) (used in Chapter 6). Here, four methods of adjusting variance estimates in order to deal with violations were tested:

Method 1) Where $\rho_I(1)$ does not satisfy $-0.5 < \rho_I(1) < 0$, set implementation variance to zero and timekeeper variance to the value of total variance.

Method 2) Eliminate all trials where $\rho_I(1)$ is not in the range $-0.5 < \rho_I(1) < 0$.

Method 3) Select the first valid trial that satisfies $-0.5 < \rho_I(1) < 0$.

Method 4) Implementing the Kooistra et al. (1997) adjustments for negative variance, sample size and drift.

7.3.1. Method 1

The first method, used by several authors (Bolbecker et al., 2011; Ivry & Keele, 1989; Kooistra et al., 1997), is to set any negative values of implementation variance to zero. These instances result from negative lag-one covariance, and the correction can be seen to result in timekeeper variance being set to the value of total variance (see Equations 3 and 6, Chapter 5/Appendix). Studies simulating tapping data over multiple runs have found that correcting negative variance estimates to zero does not excessively inflate the estimates of timekeeper and implementation variance and may also reduce the mean squared error of the estimates (Collier & Ogden, 2004; Kooistra et al., 1997). This correction also has the effect of eliminating most of the occurrences of positive lag-one autocorrelations (given that $-0.5 < \rho_I(1) < 0$).

7.3.2. Method 2

This more conservative strategy for dealing with violations entirely eliminates any trials where the lag-one autocorrelation exceeds the predicted range of zero to minus one-half (e.g. Duchek, Balota, & Ferraro, 1994; Freeman et al., 1996; O'Boyle et al., 1996; Pastor et al., 1992; Vanneste, Pouthas, & Wearden, 2001). This method can, however, result in deletion of large quantities of data, particularly in studies examining timing in untrained participants where relatively few trials are completed due to time/fatigue constraints (e.g. when assessing children). The strategy allows values to be averaged over any remaining trials but averages for each different participant may be derived from a different numbers of data points.

7.3.3. Method 3

A third method of dealing with violations is to restrict analyses to the first trial from each participant which satisfies the assumption of a lag-one autocorrelation between minus one-half and zero, a method explored by O'Boyle et al. (1996) and Pastor et al. (1992). Like Method 2, this is a conservative approach that increases the within subject sampling error but in contrast to Method 2 ensures an equivalent number of trials are included for each participant.

7.3.4. Method 4

The final strategy for dealing with violations was described by Kooistra et al. (1997) and used in Chapter 6. This method not only corrects negative variance estimates to zero but also (a) incorporates a measure of linear drift directly into the analysis and (b) accounts for the fact that the estimates result from a limited sample of data from the total population of possible values. An alternative to the Kooistra method of adjusting for drift in the time series would be to incorporate a

drift factor which does not assume that the drift is linear. Such an approach was described by Ogden & Collier (2002), but their simulations indicated that this strategy made only small improvements to the estimates in trials with a small number of responses, like the series of 30 finger tap responses considered here. They conclude that such detrending can add noise to variance estimates, and given that violations can result from different sources, the contribution of drift is difficult to determine (Collier & Ogden, 2004; Madison, 2001b).

In summary, the analysis below examines the relative contribution to variance estimates of drift and dependence at lags greater than one using time series data collected from an adult sample. In addition, the four different methods of dealing with violations are assessed. In order to allow comparisons between these data and those from the original Wing-Kristofferson model (Wing & Kristofferson, 1973a, 1973b) as well as with the time series in the next study (Chapter 8), finger tapping data was collected across five finger tapping rates. If the original Wing-Kristofferson model holds, timekeeper variance is expected to increase at slow tapping rates compared to fast rates (Vorberg & Wing, 1996; Wing & Kristofferson, 1973b). In contrast, implementation variance should not vary across the different rates of tapping.

7.4. Methods

7.4.1. Participants

The sample comprised 40 adults from University staff and student populations with recruitment following procedures described in Chapter 4. Five participants met the exclusionary criteria, one with a prior neurological condition, two scoring more than 2 SD below the mean on tests of verbal or non-verbal reasoning from the WASI (Wechsler, 1999), and two who failed to complete the motor timing task. Of the remaining thirty-five, 13 were male and 3 were left handed with a mean age of 20.9 years (SD 4.3). Fifteen of these participants had also taken part in the experiment examining stimulus modalities (Chapter 6) with at least a 2 week gap between participation in the first study; allowing participants to complete both studies maximised recruitment. This strategy was thought appropriate given that the data collected for this second study was to act as a control sample of time series data on which to test the model, regardless of the success or failure of participant performance. On measures of verbal and non-verbal reasoning from the WASI (Wechsler, 1999) mean standard scores were 112.0 and 104.1 respectively (SD 7.1 and 8.4). Twenty-two of the participants had some prior musical experience (average musical experience 2.5 years, SD 3 years, maximum experience 10 years).

7.4.2. Motor timing measures

Participants completed a synchronise-and-continue motor timing task, synchronising with auditory pacing stimuli for 10 taps and then continuing to tap when the stimuli had stopped, with sufficient time provided for up to 40 further continuation responses to be recorded in each trial. Participants were provided with a practice trial with pacing stimuli occurring with an inter-onset interval of 550ms (2Hz). Participants then completed five blocks of three trials. In each block a different target inter-onset interval (IOI) was used with five different IOIs presented (670, 505, 400, 329, and 282ms; equivalent to 1.5, 2, 2.5, 3 and 3.5Hz). The order of the blocks was randomised. The pacing stimulus comprised an auditory tone of duration 49ms presented via computer speakers at a constant rate through E-prime stimulus presentation software (E-Prime 2.0; Schneider, Eschman, & Zuccolotto, 2002) with onsets timed and corrected for the screen refresh-rate to achieve precision of presentation of ±1ms. The time of occurrence of finger tap responses was recorded using a flat switch plate which had minimal vertical travel when pressed.

7.4.3. Data pre-processing and analysis

The first five responses in the continuation phase were discarded to allow responses to stabilise and the subsequent 30 continuation responses were analysed. Trials where more than 10 inter-response intervals (IRIs) fell outside the range of 50% of the target interval (e.g. with a target IOI of 670ms, responses outside the range of 335-10005ms) were removed from the analysis as responses that likely resulted from response errors (e.g. doubled responses). In all, 7.8% of trials were excluded, with the most failures occurring at the 3Hz tapping speed (3% of all trials). Following the above exclusions two participants had only 50% of their trials remaining and these remaining trials showed excessively high CV values (greater than 15%). These two participants were therefore excluded from further analyses. The remaining valid trials comprised 88% of the original data (464 out of 525 total trials). IRIs were calculated for 30 continuation responses and mean IRI, SD of IRIs and coefficient of variation (CV) calculated for each trial. Averages of these variables were computed collapsing across the three trials completed at each tapping speed.

The first aim of this experiment was to compare the trials which did and did not satisfy the assumptions of the Wing-Kristofferson model. The IRI data were analysed using the original Wing-Kristofferson model described in Chapter 5, equations 1 to 8 to provide estimates of timekeeper and implementation variance. Where presented, the square root of variance estimates are used to provide values as standard deviations (ms), a common transformation in the literature to reduce skew in the data (Collier & Ogden, 2004; O'Boyle et al., 1996; Pastor et al., 1992). Where the effect of tapping rate was examined, one-way ANOVA were used with five levels of the independent variable. Outliers were identified using the methods described in Chapter 4.

The autocovariance values for lags two to five were calculated and averaged across the three trials and across all participants for each tapping speed. The average observed (biased) autocovariance values were then plotted with 99% confidence intervals and were compared to the expected biased autocovariance values calculated using Equation 12. Drift in the time series was assessed using a least squares linear regression (linear drift) and the $f'(k)$ non-parametric estimator developed by Madison (2001a). The data were also adjusted using the four methods set out in the introduction (see 7.3) to examine the impact of the methods on variance estimates.

7.5. Results

7.5.1. Motor timing performance

The mean IRIs achieved for the five response rates are shown in Table 7.1. The rate of tapping had a significant effect on within-participant response variability (SD of IRIs) ($F_{(4,124)}$=46.00, p<0.01, η^2=0.60) with less variability at the faster tapping rates. Even when normalised by mean IRI using the CV measure, the effect of tapping rate on variability remained significant ($F_{(4,116)}$=4.92, p<0.01, η^2=0.15).

Table 7.1: Mean performance at different tapping speeds

IOI (ms)	Mean IRI (ms)		SD of IRIs (ms)		CV	
	Mean	(SD)	Mean	(SD)	Mean	(SD)
670	644	(30.0)	37	(9.9)	0.06	(0.02)
505	485	(24.7)	31	(9.5)	0.07	(0.02)
400	385	(14.2)	25	(5.6)	0.07	(0.02)
329	319	(17.9)	23	(4.9)	0.07	(0.02)
282	265	(17.7)	19	(3.4)	0.07	(0.01)

Summary of violations

Table 7.2 shows the number of trials (as a percentage of all trials) in which the lag-one autocorrelation fell outside the bounds of zero to minus one-half. Violations occurred most frequently with 670ms IOIs; a result anticipated given that the variability in adjacent intervals is greatest at slower speeds in a clock-counter model in which response intervals are thought to be timed by counting the appropriate number of timekeeper intervals (Wing & Kristofferson, 1973b). Negative estimates of timekeeper variance are possible (given $\sigma_T^2 = \gamma_1(0) + 2\sigma_P^2$) where the value of implementation variance is greater than half the value of timekeeper variance. There were a greater number of such cases at the fastest tapping rates. Again, this was expected in view of the fact that the timekeeper mechanisms should be less variable at high speeds relative to the implementation system. The number of cases which showed negative estimates of timekeeper or

implementation variance is comparable to that found in previous studies (see Chapter 2). For example, Collier and Ogden (2004) found 38% of trials had negative implementation variance and 12% of trials had negative timekeeper variance across a range of tapping speeds (from 333ms to 811ms).

Table 7.2: Violations of the assumption of positive variance

IOI (ms)	N trials	% trials with violations of the lag-one autocorrelation bounds where: $\rho_I(1) > 0$	$\rho_I(1) < -0.5$	Total % trials with violations of $-0.5 < \rho_I(1) < 0$
670	94	35.8	5.3	40.4
505	94	22.3	5.3	27.7
400	92	13.0	10.9	23.9
329	88	4.6	19.3	23.9
282	95	10.5	22.1	32.6

7.5.2. Analysing the source of model violations

7.5.2.1. Autocovariance at lags beyond one

The trials which violated the assumption $-0.5 < \rho_I(1) < 0$ were analysed separately from those which satisfied these bounds. The autocovariance functions for violating and valid trials are presented in Figure 7.3 and Figure 7.4. For the valid trials which satisfied the autocorrelation assumption, the expected values of autocovariance all fell within the confidence intervals of the observed values, except on the trials with 400ms IOIs where the expected value exceeded the confidence intervals at lag-four (Figure 7.3, C). This is indicative of dependence at lag 4 i.e. closed-loop timing based on responses at lag 4 being used to predict subsequent responses. For the trials which did not satisfy the lag-one autocorrelation bounds, the expected values of autocovariance fell outside the confidence intervals of the observed values across all five tapping rates at various lags from two to five (Figure 7.4). This means that in the violating trials, participants were frequently using feedback from lags beyond one to update the time of their responses. In such trials, participants were therefore consistently using a closed-loop form of timing control rather than the open-loop timing control predicted by the model.

7.5.2.2. Stationarity

The median linear slope and r^2 values are presented in Table 7.3 and Figure 7.5. The results indicate that linear trends were present in many trials, although the average r^2 values across trials were low. The largest linear trend was found on violating trials at the slowest tapping speeds (670 and 505ms IOIs). In contrast, trials which satisfied the model did not show such pronounced linear trends, even at the slowest tapping speeds. To assess non-linear drift, $f'(k)$ was calculated and the summary

data is provided in Table 7.4 and Figure 7.6. Again, the average r^2 values for non-linear drift were small. The largest amount of drift was present on violating trials at the slowest tapping speed (670ms IOIs).

7.5.3. Methods of dealing with violating cases: effects on variance estimates

The Wing-Kristofferson variance estimates were calculated using the four adjustment methods described in the introduction and are presented in Figure 7.7 and Figure 7.8. In the original Wing-Kristofferson model of timing control, timekeeper variance is predicted to decrease as tapping rate increases whilst implementation variance should be unaffected by tapping rate. The effects of tapping rate (5 levels) and adjustment method (4 levels) on the variance estimates were tested using repeated measures ANOVA.

For timekeeper variance, the predicted effect of tapping rate on timekeeper variance was confirmed with significantly lower variance at fast performance speeds ($F_{(4,88)}$=27.71, p<0.01, η^2=0.55). The main effect of adjustment method was not significant ($F_{(3,66)}$=7.49, p=0.74, η^2=0.02), but the interaction between rate and method was significant ($F_{(12,264)}$=2.26, p<0.01, η^2=0.09). On examination of Figure 7.7, the greatest difference in timekeeper variance between the methods is evident on trials with 670 and 505ms IOIs. Pair wise comparisons demonstrated that Method 1 produced estimates that were significantly different from Methods 2 and 3 at the slowest tapping rate ($t_{(29)}$=2.14, p<0.05 and $t_{(29)}$=2.46, p<0.05 respectively). Method 4 was not significantly different from the other methods on the 670ms trials. On the 505ms IOI trials there were no significant differences between the timekeeper variance estimates across the methods (p>0.14).

For implementation variance, the main effect of tapping rate was significant ($F_{(4,88)}$=3.88, p<0.01, η^2=0.15), diverging from the predictions of the Wing-Kristofferson model. The main effect of method did not reach significance (($F_{(3,66)}$=2.50, p=0.07, η^2=0.10) but the interaction was significant ($F_{(12,264)}$=4.36, p<0.01, η^2=0.17). Examination of Figure 7.8 suggests that the largest difference in implementation variance between the Methods was at the two slowest tapping speeds. Pair wise comparisons at these speeds showed that Methods 1 and 4 were both significantly different from Methods 2 and 3 on the 670ms IOI trials (p<0.05). For the 505ms IOI trials Method 1 and 4 were significantly different from Method 2 (p<0.01) but not from Method 3 (p>0.60). Given these results, the effect of rate was separately assessed for each method. There was no effect of rate on implementation variance for Methods 1 and 4 respectively ($F_{(4,124)}$=1.74, p=0.15, η^2=0.05 and $F_{(4,124)}$=0.45, p=0.8, η^2=0.01 respectively), but a significant effect of rate for Methods 2 and 3 ($F_{(4,88)}$=8.33, p<0.01, η^2=0.26 and $F_{(4,88)}$=3.86, p<0.01, η^2=0.15).

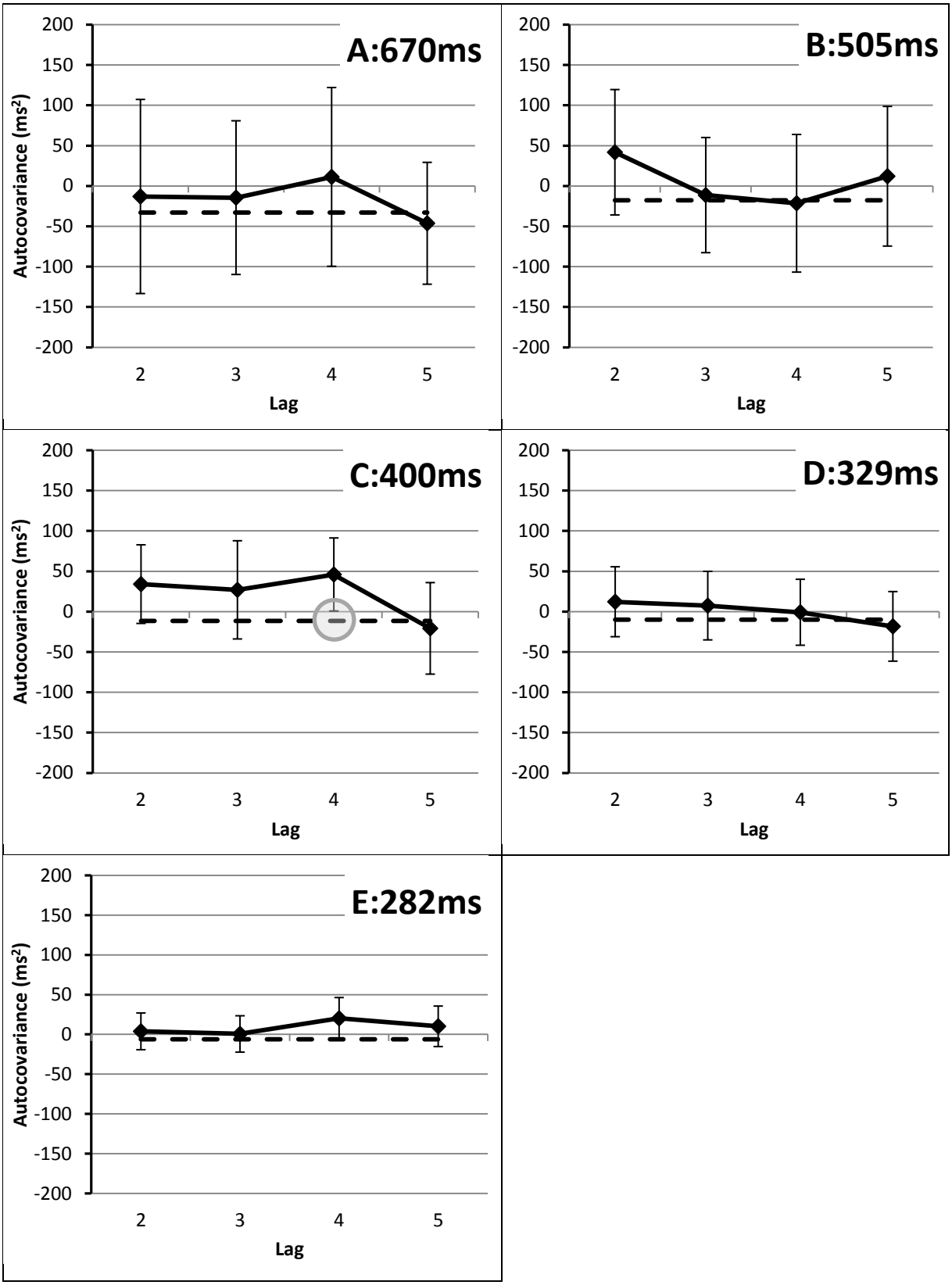

Figure 7.3: Autocovariance at lags beyond one for valid trials

Solid lines represent observed mean autocovariance at lags 2-5 with 99% confidence intervals. Dotted lines show the expected value of autocovariance. Grey circles indicate instances of closed-loop timing, where the expected value is outside the confidence intervals for the observed data.

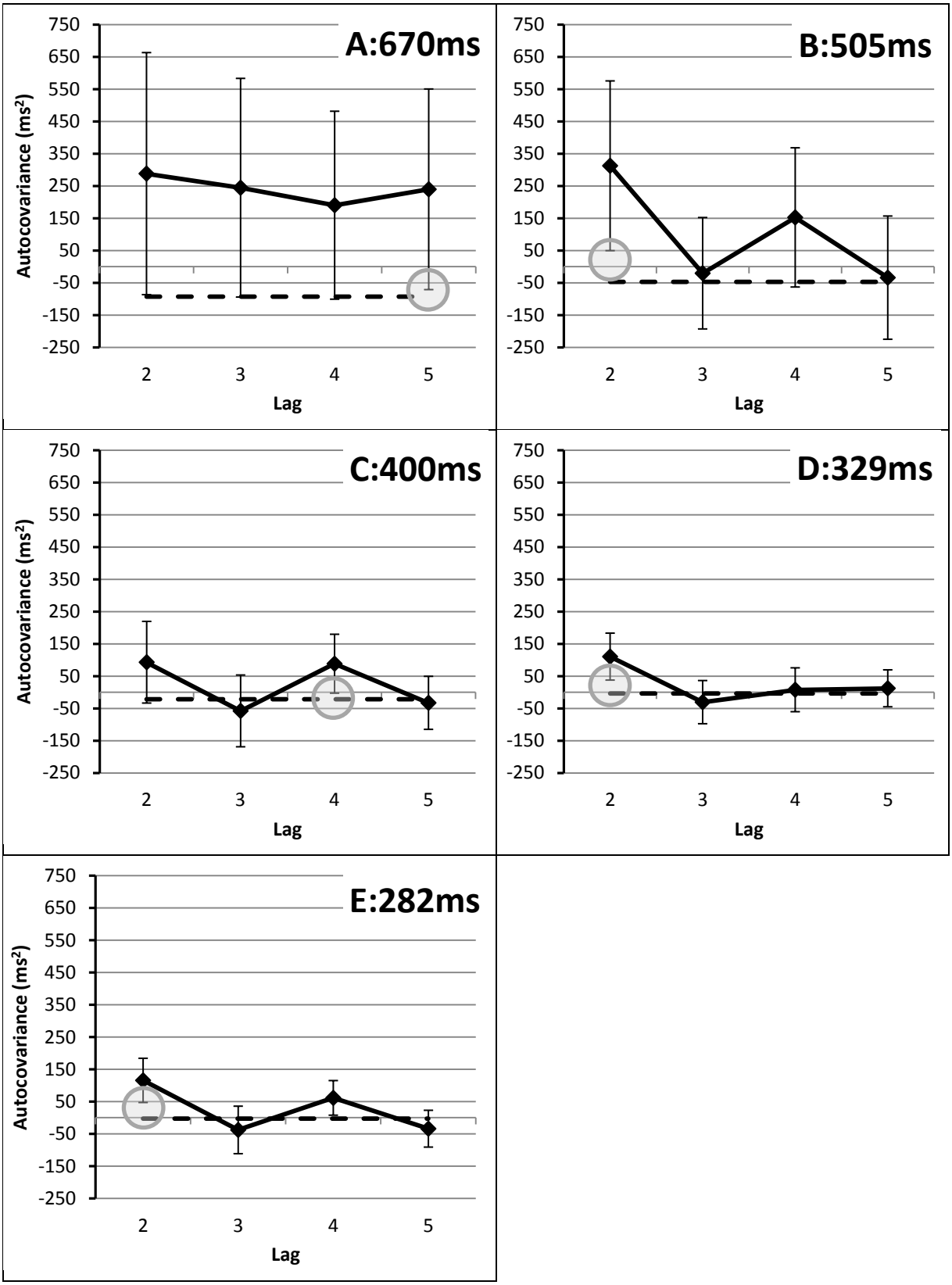

Figure 7.4: Autocovariance at lags beyond one for violating trials

Solid lines represent observed mean autocovariance at lags 2-5 with 99% confidence intervals. Dotted lines show the expected value of autocovariance. Grey circles indicate instances of closed-loop timing, where the expected value is outside the confidence intervals for the observed data.

Table 7.3: Median Slope of linear regression for time series which did and did not satisfy the autocorrelation assumption

IOI (ms)	Valid trials				Violating trials			
	N	Slope	Slope (disregarding sign)	r^2	N	Slope	Slope (disregarding sign)	r^2
282	31	-0.19	0.36	0.03	64	-0.18	0.23	0.01
329	21	-0.23	0.34	0.02	67	-0.05	0.17	0.01
400	22	-0.21	0.44	0.03	70	-0.21	0.37	0.01
505	26	-0.34	0.45	0.02	68	-0.65	0.76	0.05
670	38	-0.19	0.73	0.03	56	-1.02	1.38	0.12

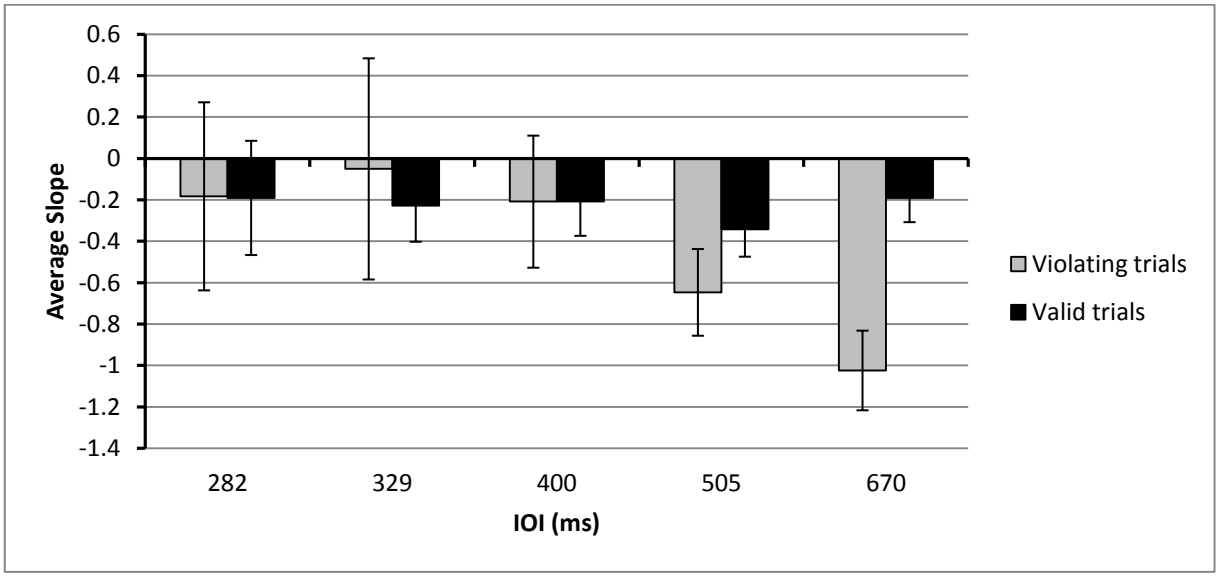

Figure 7.5: Group median linear slope across tapping rates for trials which did and did not satisfy the autocorrelation assumption
Error bars represent 2 SEM.

Table 7.4: Median $f'(k)$, for trials which did and did not satisfy the autocorrelation assumption

IOI (ms)	Valid trials				Violating trials			
	N	$f'(k)$	$f'(k)$ (disregarding sign)	r^2	N	$f'(k)$	$f'(k)$ (disregarding sign)	r^2
282	64	0.00	0.32	0.04	31	0.38	0.45	0.03
329	67	0.06	0.48	0.07	21	-0.03	0.36	0.07
400	70	0.03	0.46	0.05	22	0.24	0.34	0.06
505	68	0.03	0.27	0.04	26	0.20	0.48	0.15
670	56	0.01	0.48	0.04	38	0.09	0.67	0.09

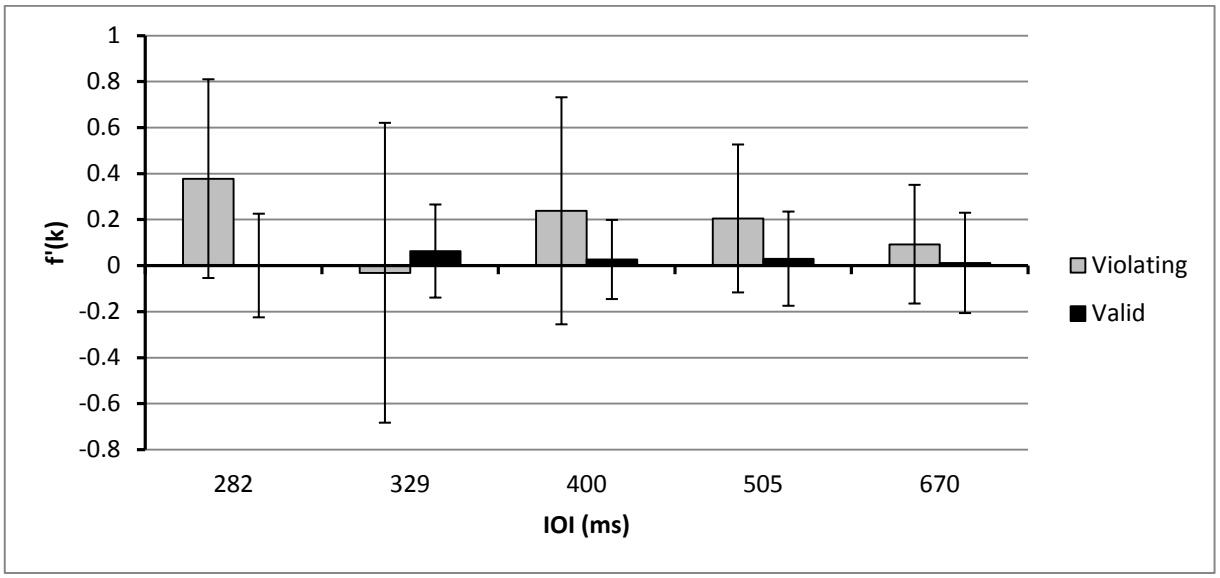

Figure 7.6: Group median slope of $f'(k)$ across tapping rates for trials which did and did not satisfy the autocorrelation assumption

Error bars represent 2 SEM.

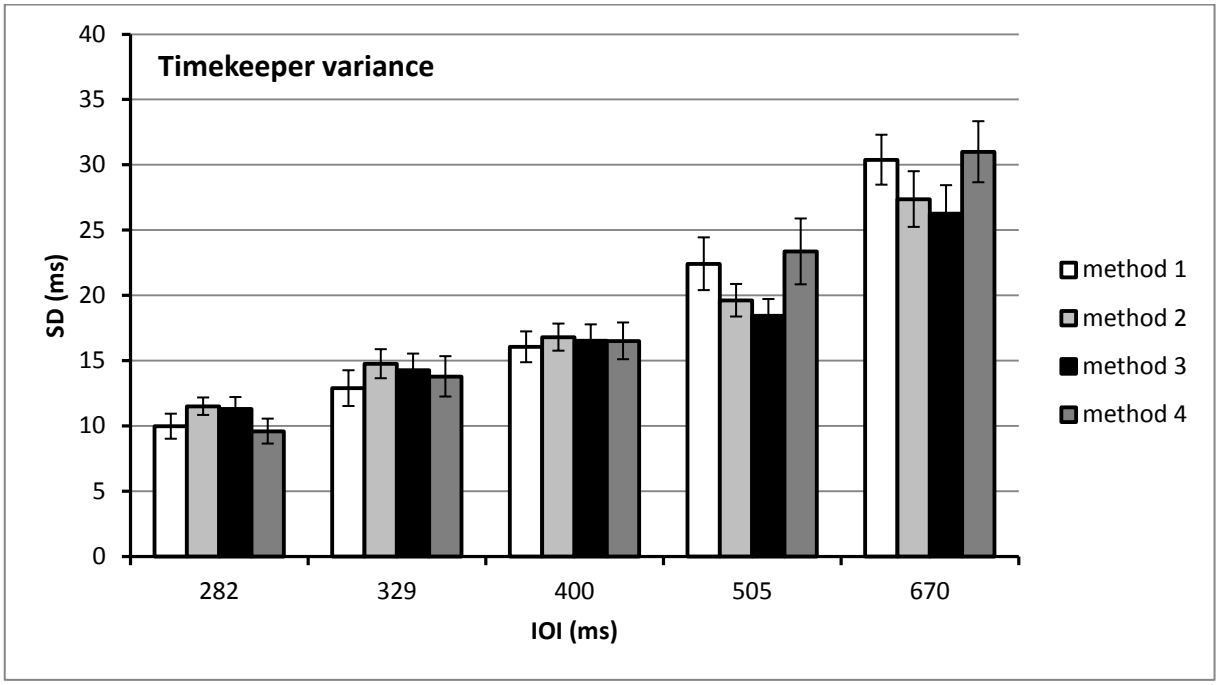

Figure 7.7: Timekeeper variance estimates calculated via different methods of adjustment
Variance presented as standard deviations

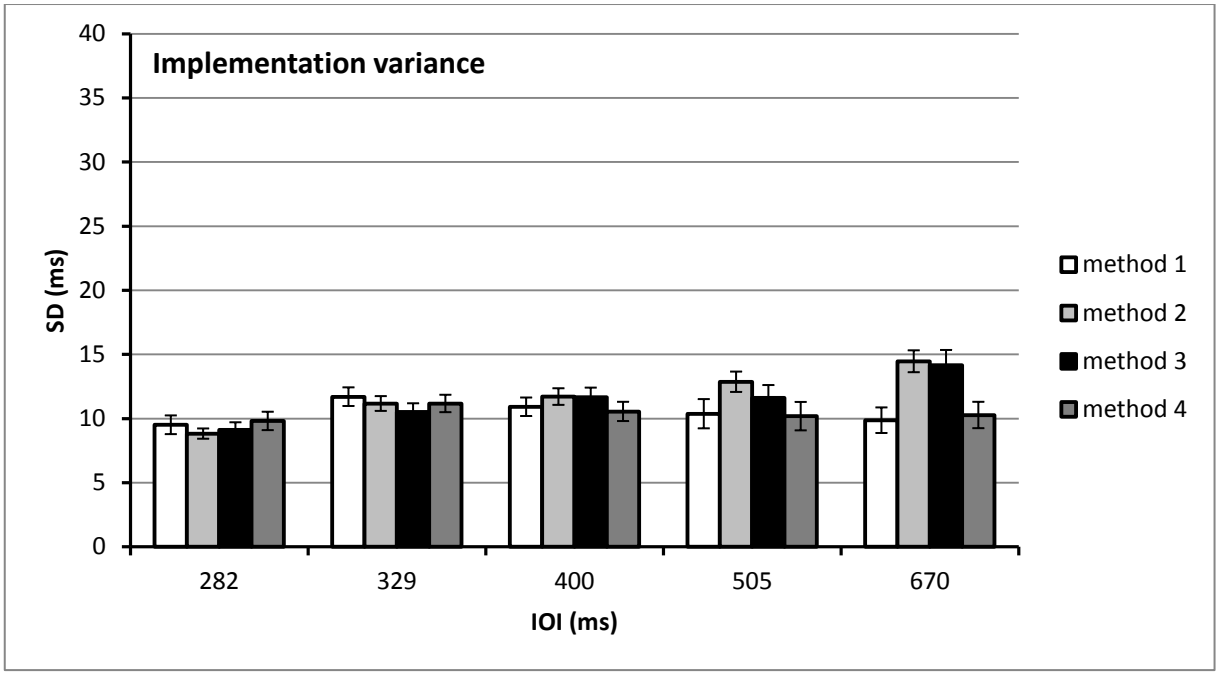

Figure 7.8: Implementation variance estimates calculated via different methods of adjustment
Variance presented as standard deviations

7.6. Discussion

Based on the violations of the Wing-Kristofferson model observed in the experiments in Chapter 6, the aim of this experiment was to explore the factors which contribute to violations of the model and to examine the different methods that can be used to reduce the effects of such factors. This was

completed to determine the appropriate method of data analysis for use in the subsequent experiments in the thesis.

In general, the performance of the participants was comparable to that in previous investigations of timing in adults (Freeman et al., 1996; Ivry & Keele, 1989; Vanneste et al., 2001; Wing & Kristofferson, 1973a; Wing, 1980). The participants were able to match the target intervals with reasonable accuracy at a level comparable to adult control samples in other studies which found similar IOI-IRI differences ranging from 5-47ms (Harrington et al., 1998; Thomson et al., 2006). The group showed a reduction in within-participant variability at the fastest tapping rates compared to the slow rates, an effect which supports a pacemaker model of timing in which fewer pulses of a timekeeper are required to be summed for each interval, resulting in less variability in the timing of motor responses generated (Wing & Beek, 2002).

In an autocorrelation model, violations that influence the estimation of variance components can result from the presence of drift or dependences at lags beyond one (Collier & Ogden, 2004; Vorberg & Wing, 1996). Wing-Kristofferson estimates for timekeeper and implementation variance were calculated and trials were divided into those which satisfied the autocorrelation function (i.e. that the lag-one autocorrelation falls between zero and minus one-half) and those that did not. The number of trials which violated the model assumptions ranged from 23-40% of trials across the five speeds assessed. This is comparable to the number of trials violating this function in other control samples of adults (Bolbecker et al., 2011; Collier & Ogden, 2004; Harrington et al., 1998; O'Boyle et al., 1996; Pastor et al., 1992).

There was only one instance in the valid trials where the average expected autocovariance values at lags beyond one fell outside the bounds calculated based on the observed data, indicating relatively few dependencies in the data. In contrast, the trials that did not satisfy the model showed dependencies at lags beyond one at all tapping speeds. These trials therefore contained more instances of closed-loop timing control. Closed-loop timing is more likely to occur when tapping in synchrony with a pacing stimulus than when continuing to tap in the absence of a tone, because the presence of the stimulus allows adjustment of responses (Repp, 2005). When timing is self-paced in the absence of a pacing stimulus, such updating should not be required under optimal conditions. Closed loop timing may occur during unpaced timing if a participant is disturbed by a distraction in the environment that disrupts timing control or movement (Wing, 1977b). The presence of closed loop timing in this experiment may therefore result from these participants being unpractised at the tapping task and/or becoming fatigued or distracted from providing accurately timed responses.

The valid trials also showed less drift than the violating trials, when drift was calculated using both a linear regression and a non-parametric estimator ($f(k)$). The amount of linear drift was greatest at

the slowest tapping speeds (670 and 505ms IOIs; 1.5 and 2Hz). Trials at these slow speeds were also those which showed the largest number of violations of the autocorrelation bounds (of zero to minus one-half). This association is expected given that drift will increase the total variance in responses and that the Wing-Kristofferson model is based on the properties of a stationary time series. However, r-squared values for both the linear and non-parametric estimators of slope were relatively small, indicating that the non-stationarity present did not account for substantial variance in the data.

Previous investigations have found that non-stationarity in time series can artificially inflate estimates of timekeeper variance and deflate implementation variance estimates (Collier & Ogden, 2004; Jucaite et al., n.d.; Madison & Delignières, 2009; Madison, 2001b). When the different methods of adjusting the Wing-Kristofferson method were applied, the presence of violations (including the non-stationarity) did appear to lead to higher estimates of timekeeper variance and lower estimates of implementation variance. The two methods in which only the data which satisfied the autocorrelation model was used (Method 2 and 3) were found to be less affected by the presence of drift at the slowest speeds, with no changes in timekeeper or implementation variance that were present with Methods 1 (Figure 7.7 and Figure 7.8). In Method 1, violating cases were not eliminated but instead corrected and the influence of non-stationarity on variance estimates could be observed at the slowest speeds. Method 4, the adjusted model described by Kooistra et al. (1997), attempted to reduce the effect of drift by incorporating a linear slope value into the variance model. However, the presence of other problems (such as closed loop timing) meant that this method showed the same deflation of implementation variance as Method 1 in the original Wing-Kristofferson model. In contrast, for timekeeper variance, Method 4 performed like Methods 2 and 3. Thus, the controls introduced by Kooistra et al. modulated the effect of violations on timekeeper variance, but the dependencies in the data or non-linear drift still had an influence on the estimates of implementation variance.

In the presence of violating trials in Methods 2 and 3, a linear effect of rate was observed, with increasing implementation variance as tapping rate decreased. Such a trend is not predicted by the Wing-Kristofferson model because the implementation of motor responses should be independent of external influences such as rate (Wing & Kristofferson, 1973b; Wing, 1980). However, the effect size for this linear relationship between implementation variance and tapping rate was rather small (η^2=0.15) in contrast to that for the expected main effect of rate on timekeeper variance (η^2=0.55).

The effects of the Kooistra et al. adjustment (Method 4) were minimal such that the data resembled those from the original Wing-Kristofferson model. The method only accounted for linear but not other forms of drift or dependencies in the data resulting from closed loop timing control. Other studies have also reported that linear detrending had minimal effects on the variance estimates

(Helmuth & Ivry, 1996; Ivry et al., 1988; Ivry & Keele, 1989; Kolers & Brewster, 1985). An alternative to this strategy would be to incorporate a drift factor which does not make assumptions about the nature of the drift. Such an approach has been described by Ogden and Collier. However, statistical simulations revealed that this strategy produced only minor improvements to estimates in trials which only comprised a small number of finger tap responses, such as those considered here (Collier & Ogden, 2004; Ogden & Collier, 2002). An alternative strategy is to quantify any non-stationarity and dependencies in the data in order to estimate their influence over timing performance. For studies of individual differences in timing behaviour, individual measures of drift also allows comparison of whether participants differ in the degree to which their time series are non-stationary, which may give further insight into the reasons for any differences in behaviour. A similar analysis of such factors was recently used by Zelaznik et al. (2012) in an examination of motor timing in children with ADHD. As applied here, the non parametric estimator $f(k)$ described by Madison (2001a, 2001b) allows quantification of drift without making assumptions about the form of drift. Similar analyses will therefore be applied in the subsequent experiments on motor timing.

7.6.1. A comment on Chapter 6

Given that the Kooistra et al. adjustment (Method 4) was used in Chapter 6, it is pertinent to consider the impact of the present findings on the findings from that chapter. The data here indicates that the presence of drift will be minimal for fast tapping rates such as the 333ms IOIs (3Hz) used in Chapter 6. Therefore it is likely that the effects of non-stationarity (linear or otherwise) would have been minimal. Similarly, the presence or absence of violating cases had the least impact on timekeeper or variance estimates at the faster tapping rates and so the correction of negative variance estimates to zero in the Kooistra adjustment would be unlikely to significantly affect the results reported in Chapter 6.

7.7. Conclusion

This experiment shows that the measurement of drift and dependency at lags beyond one can be useful in order to identify differences in timing control which may invalidate the use of a lag-one autocorrelation timing models. Under optimal conditions, unpractised participants are able to produce time series that do satisfy the bounds of the Wing-Kristofferson model in order to allow assessment of the relative contributions of timekeeper and implementation variance. The measurement of the presence of closed-loop timing can demonstrate whether participants are using an alternative method of control (as demonstrated by Zelaznik et al., 2012), and the presence of drift can reveal an inability to appropriately monitor and maintain control over timing. The inclusion of these analyses alongside the traditional application of the Wing-Kristofferson model, will supplement understanding of timing control in these developmental populations.

The two adjustment methods that performed most satisfactorily were those that only analysed successful trials (Methods 2 and 3). In comparing these adjustment strategies, the difficulty with Method 2, in which all valid trials are analysed, is that averages for different participants will be based on different quantities of data, whereas Method 3 results in equal samples of data per participant. Therefore, in the next experiment (Chapter 8), Method 3 (selecting the first valid trial) will be used for the analyses. Although this does increase the within-participant sampling error, the analysis of successful trials eliminates those affected by momentary distractions providing an assessment of basic timing control abilities under baseline conditions (Zelaznik, Spencer, & Ivry, 2008). This strategy has been previously implemented by Pastor et al. (1992) and Zelaznik et al. (2012).

8. Assessing the nature of motor timing in children with reading difficulties

8.1. Introduction

As described in Chapter 3, children with dyslexia are often found to be impaired at producing accurate and reliable responses on motor timing tasks, yet little is known about which aspects of temporal processing contribute to these performance decrements. In Chapter 6, the Wing-Kristofferson statistical method of decomposing motor timing performance (Wing & Kristofferson, 1973a, 1973b) was shown to be useful in determining the differences between experimental conditions in motor timing tasks. The decomposed variance components quantified by the Wing-Kristofferson model have not previously been examined in relation to the motor timing variability exhibited by children with dyslexia. Therefore this study assesses the different components of timing that contribute to motor timing performance in children with reading difficulties in comparison to age and reading level matched controls. In addition, the relationships between the components of timing and cognitive indicators of dyslexia are explored.

Children with dyslexia have been found to show difficulties on both paced and unpaced isochronous timing tasks as well as on tasks with anisochronous stimuli (See Chapter 3; Badian & Wolff, 1977; David, Wade-woolley, Kirby, & Smithrim, 2007; Huss, Verney, Fosker, Mead, & Goswami, 2011; Klipcera, Wolff, & Drake, 1981; Overy, Nicolson, Fawcett, & Clarke, 2003; Thomson, Fryer, Maltby, & Goswami, 2006; Thomson & Goswami, 2008; Waber et al., 2000; Wolff, Michel, Ovrut, & Drake, 1990; Wolff, Cohen, & Drake, 1984; Wolff, 2002). In addition, the intra-subject variability in timing on these tasks can predict around 20% of the variance in literacy and associated literacy component skills (Goswami, 2006; Thomson & Goswami, 2008). This study focuses on children's performance on isochronous timing tasks which engage a less complex neural network of timing control than rhythmic anisochronous tasks (Grahn & Brett, 2007) and for which variability can be readily analysed using the Wing-Kristofferson model (Vorberg & Wing, 1996; see Chapter 2). Children with dyslexia typically exhibit greater variability in inter-response intervals (IRI) on isochronous motor timing tasks, regardless of whether the task is paced (synchronisation tasks) or unpaced (self-paced or continuation tasks). It remains unclear whether this difficulty arises from an inability to appropriately match behavioural responses to stimuli or alternatively, from an impairment in the internal timekeeping systems that facilitate self-paced timing. The present experiment investigates the latter of these two options by assessing performance in the continuation phase of a synchronise-and-continue finger tapping task.

Studies of clinical populations, such as patients with cerebellar lesions or Parkinson's disease, have demonstrated how the use of the Wing-Kristofferson model improves understanding of the individual components of timing control that are disrupted in these populations (Bolbecker et al.,

2011; Harrington et al., 1998, 2004; Ivry & Keele, 1989; O'Boyle et al., 1996; Pastor et al., 1992; Zelaznik et al., 2012). Such statistical methods have also been applied to assess how timing is mediated by individual differences such as age or intelligence (Madison et al., 2009; Vanneste et al., 2001). In the Wing-Kristofferson model, implementation variance represents the random delays generated by the motor system, peripheral to the timing control system. Timekeeper variance represents the relatively invariant outputs of the timing control system, which should only vary with the size of the inter-response intervals (see Chapter 2). Establishing whether the difficulties with motor timing in children with dyslexia are associated with impairments in one or other of these components will help direct future research to particular timing mechanisms and associated brain regions.

Given that the timing difficulties associated with the core deficits of reading and spelling in dyslexia are not typically associated with motor dexterity (Stanford & Barratt, 1996; Thomson & Goswami, 2008), it is expected that timing difficulties in children with reading difficulties will not be primarily attributable to implementation variance. Instead, it is expected that timekeeper variance will be greater in the children with reading difficulties compared to the control groups. The evidence for a difficulty in achieving timed response synchrony in synchronised and self-paced timing tasks described in Chapter 3, suggests that these children have differences in the perceptual registration of tap-timer synchrony or in the representation of temporal stimuli in the pacemaker mechanism of the timekeeping system.

Both chronological-age (CA) and reading-level (RL) matched controls were used here to determine whether any differences between poor readers and chronological-age matched children are merely due to the lack of reading experience in the poor readers or general developmental delay not associated with reading performance (See Chapter 4; Backman, Mamen, & Ferguson, 1984). Furthermore, it was anticipated that the timekeeper component would be correlated with measures of literacy and literacy component skills in the same way that timing variability (standard deviation of IRIs) is typically associated with such measures.

The overview presented in Chapter 3 showed that performance deficits on motor timing tasks are also found in children with ADHD. Some of the earlier studies that assessed motor timing in dyslexia did not control for the presence of attentional impairments (Thomson et al., 2006; Wolff et al., 1984; Wolff, Michel, & Ovrut, 1990) despite the high degree of overlap between the symptoms of dyslexia and ADHD. ADHD symptoms are therefore measured here alongside literacy skills, to examine their relative contribution to the relationship between literacy and timing performance. Some of the general motor impairments commonly found in children and adults with dyslexia have also been attributed to the presence of ADHD symptoms (Denckla et al., 1985; Raberger & Wimmer, 2003; Ramus et al., 2003; Rochelle et al., 2009), and so the timing deficits shown by children with ADHD

and dyslexia may be distinct. For example, reading difficulties may be more strongly associated with increases in timekeeper variance whereas attentional difficulties may be related to motor impairments reflected in implementation variance. However, children with ADHD show brain-based differences across the neural network which controls timing (particularly in the striate and cerebellar cortices), a profile which would be consistent with deficits across both the timekeeper and implementation components (Toplak & Tannock, 2005b). Therefore in contrast to the expected correlations between literacy scores and timekeeper variance, it is anticipated that symptoms of inattention will be associated with increases in both timekeeper and implementation variance.

Typically, motor timing deficits on finger tapping tasks have been found in children with reading difficulties across a range of movement speeds (from 200-1000ms or 5-1Hz; see Chapter 3), although most commonly associations between reading and timing performance have been found when intervals in the 400-600ms range (2.5-1.6Hz) have been used (Thomson et al., 2006; Thomson & Goswami, 2008). Timing of intervals in this range falls into the category of millisecond timing (described in Chapter 2) controlled automatically by a network that includes the posterior cerebellum, sensorimotor, premotor and auditory cortices and the basal ganglia (Buhusi & Meck, 2005; Buonomano et al., 2009; Ivry, 1996; Lewis & Miall, 2003; Mauk & Buonomano, 2004; Meck & Benson, 2002; Rammsayer, 1999; Repp, 2002, 2005; Wiener et al., 2010). In adults, implementation variance should remain stable across the millisecond range whereas timekeeper variance increases as IRIs increase (Vanneste et al., 2001; Vorberg & Wing, 1996; Wing & Kristofferson, 1973a).

To capture the differences in motor timing demonstrated in previous research, stimulus rates similar to previous studies are used (i.e. IOIs of 283-673ms; 3.5-1.5Hz) which will allows examination of whether timing deficits in children with reading difficulties are largely confined to a narrow range of stimulus rates. Examining a number of finger tapping rates also affords an analysis of whether components of timing variability in children are affected by rate in the same way as in adults, with reduced variability and timekeeper variance at faster tapping speeds.

Following the analyses presented in Chapter 7 only the first successful motor timing trials which satisfied the Wing-Kristofferson model predictions are assessed. Furthermore, the additional analyses recommended in that chapter are implemented to better characterise the type of timing control used by participants (i.e. analyses of drift and the presence of closed-loop timing control). Finally, to allow examination of the model fit, the presence of violating trials will be reported. A difference in the number of instances of closed loop timing in the children with reading difficulties is anticipated because the higher variability in output timing shown by children with reading difficulties suggests that they implement a different strategy of correcting any instances of asynchrony. Children with ADHD have previously been found to show more instances of closed-loop timing (Zelaznik et al., 2012) and such findings may explain why children with ADHD often drift away from

the stimulus rate during synchronised performance (Ben-Pazi et al., 2003, 2006). The presence of non-stationarity has not previously been specifically measured or reported in groups with reading difficulties, although they generally are able to achieve a stable and appropriate mean IRI (Badian & Wolff, 1977; Wolff et al., 1984; Wolff, Michel, Ovrut, et al., 1990; Wolff, 2002; but see Klipcera et al., 1981; Thomson & Goswami, 2008). A difference in the amount of drift between children with dyslexia and controls is therefore not anticipated.

8.2. Methods

Further details of methods, measures and analyses were presented in Chapters 4 and 5.

8.2.1. Participants

8.2.1.1. Participant sampling

The sample of children with reading difficulties was drawn from children attending Aston University's Dyslexia Assessment Clinic. Twenty-three children were recruited in the clinical sample; four of these were ultimately excluded as they failed to complete the motor timing task. These children had all received diagnoses of literacy difficulties with associated deficits, for example in numeracy or working memory. For the matched control groups, 53 children were sampled from years four and six of two primary schools. The data from four of the controls was excluded: one for whom English was not a first language, one who was diagnosed and medicated for ADHD, one who failed to complete the motor timing task, and one who was absent during the second testing session, leaving 24 year four children and 25 year five children.

8.2.1.2. Participant matching

On a case-by-case basis, each clinical participant was allocated a reading-level (RL) and a chronological-age (CA) matched control. RL controls were selected using WIAT-II Word Reading raw scores. Suitable matches could not be found for three of the clinical participants (including the youngest and oldest participants sampled). For the remaining 16 children the maximum age difference between any Clinic group member and their CA control was 6 months (mean difference 2.4 months, SD = 1.5 months), and with their RL control was 18 months (mean difference 8.75 months, SD = 4.9). The CA controls all had higher reading scores than the Clinic children (minimum difference 6 raw score points, maximum 26 points, mean = 13.3, SD = 5.3). For the RL controls the maximum reading score difference was 8 points (mean = 2.8, SD = 2.6). In two cases the RL matched control was older than the Clinic participant (by 4 and 5 months respectively) where there was no other suitable match. Reading level comparisons between the groups are presented in Table 8.1.

Table 8.1: Age and reading scores for the Clinic group, and CA and RL matched controls

Group	Age Months		Reading Raw Score		Males (n)	Right Handed (n)
	Mean	(SD)	Mean	(SD)		
Clinic	122.1	(9.3)	96.2	(10.5)	11	13
CA Matched	122.3	(9.1)	109.5	(7.6)	9	13
RL Matched	115.9	(10.9)	98.6	(11.8)	5	10
Clinic-CA Mean difference	2.4	(1.6)	13.3	(5.3)		
Clinic RL Mean difference	8.8	(2.8)	5.0	(2.6)		

8.2.2. Cognitive & behavioural measures

Psychometric measures similar to those used in Chapter 6 were employed to assess the cognitive dimensions associated with dyslexia and ADHD (also described in Chapter 4). In summary, verbal and non-verbal reasoning ability were assessed with the Similarities and Matrices subscales of the WASI (Wechsler, 1999) and literacy skills were measured using the Word Reading and Spelling subtests from the WIAT-II UK (Wechsler, 2005). Age-referenced standard scores for these tests were derived using the published norms. The WISC-IV (Wechsler, 2003) had been administered to the Clinic group during their dyslexia assessment and so the Verbal Comprehension and Perceptual Reasoning Indices were used as measures of verbal and performance IQ for these children. Phonological and orthographic decoding skills were measured with timed non-word and irregular-word reading tests (Castles & Coltheart, 1993). The subtest scores and completion times from these tests were converted to time-error composite scores using a principal components analysis. Higher composite scores indicate poorer aggregate performance on these tests. The non-word composite variable accounted for 64% of the variance in error-time scores and the irregular-word composite variable accounted for 87%. Some of the children in the Clinic group had also completed additional measures in their clinical assessment, including the Working Memory and Processing Speed Indices from the WISC-IV, Pseudo-word naming from WIAT-II UK, the Word Reading Efficiency measures from the Test of Word Reading Efficiency (TOWRE: Torgeson, Wagner, & Rashotte, 1997), and subtests from the Comprehensive Test of Phonological Processing (CTOPP: Torgeson, Wagner, & Rashotte, 1999) including the Elision measure (a test of the ability to segment phonemes in words).

Parents of children in the clinical sample completed the Parent Form of the ADHD Behaviour Rating Scale (Barkley & Murphy, 1998) and class teachers completed the equivalent Teacher Form for children in the control groups, yielding scores for the ADHD-IA and ADHD-HI dimensions. All children completed the Same World/Opposite World task and the Score! task from the Test of Everyday Attention for Children (TEA-Ch; Manly, Robertson, Anderson, & Nimmo-Smith, 1999) which assess attentional control and sustained attention. Age-referenced standard scores were calculated using

the TEA-Ch norms and the increase in time between the Same- and Opposite-World subtests was also calculated.

8.2.3. Motor timing measures

8.2.3.1. Simple reaction time

An auditory reaction time measure (described in Chapters 4 and 6) was included to assess simple motor response speed. Two practice trials were followed by 10 test trials. Finger tap responses were registered on a flat switch plate and response times relative to stimulus onset were recorded. Response times that were less than 150ms or greater than 750ms were assumed to result from errors and were excluded from the data. Mean and standard deviation of reaction time were calculated for participants with at least six valid trials (one child did not meet this criterion).

8.2.3.2. Motor timing

Motor timing performance was assessed using the same synchronise-and-continue finger tapping task described in Chapter 7. Participants tapped in time with auditory pacing stimuli for 10 taps and were instructed to continue tapping once the stimuli ceased. Participants were provided with a practice trial with stimulus inter-onset intervals (IOIs) of 550ms and then completed five blocks of trials. Within each block of trials only one of the five target IOIs was used (673, 508, 402, 331 and 283ms, corresponding to rates of 1.5, 2, 2.5, 3 and 3.5 Hz). The number of trials was minimised to mitigate against effects of fatigue, with two trials in each block. The pacing stimulus comprised an auditory tone of duration 49ms presented via computer speakers at a constant rate through E-prime stimulus presentation software (E-Prime 2.0; Schneider, Eschman, & Zuccolotto, 2002) with onsets timed and corrected for the screen refresh-rate to achieve precision of presentation of ±1ms. The exact duration of intervals differed slightly from those used in Chapter 7 due to a difference in the refresh rate of the experimental computer used. The time of finger tap responses were again recorded with respect to the stimulus onsets on a flat switch plate.

8.2.4. Data analysis

For the finger tapping task, only data from the continuation phase was analysed. The first five responses in this phase were discarded to allow stabilization of responses and the subsequent 30 responses were analysed. Trials where more than 10 IRIs fell outside the range of 50% of the target interval (for example, responses outside the range of 336-1009ms for a base target interval of 673ms) were removed from the analysis as invalid trials which likely resulted from response errors (for example, doubled responses). This criterion was met by 6.5% of trials, with the most exclusions at the slowest and fastest rates of performance (670ms IOI: 2.2% and 283ms IOI: 1.5% of all trials). In the Clinical group a greater number of trials was excluded (n = 22) than either the RL (n = 1) or CA matched groups (n = 8). Mean IRI, standard deviation of IRIs and coefficient of variation (CV) were

calculated for each trial, and were averaged across the two trials completed at each speed. A further 20 trials, spread across the three groups, had high CV values (greater than 15%), indicative of the greater overall variability exhibited by the children in comparison to the adults in the previous study. To avoid the loss of further data such cases were not excluded from the analyses.

Consistent with the Wing-Kristofferson model, lag-zero and lag-one covariance was calculated for each trial, allowing estimation of timekeeper and implementation variance using Equations 6 and 7 (see Chapter 5/Appendix). Variance estimates were subjected to square root transforms to provide estimates in standard deviation units (ms). Lag-one autocorrelation values were examined to establish the extent to which data met the assumptions of the Wing-Kristofferson model. For the main between-group and correlation analyses, the variance estimates were derived from only the first trial which satisfied the model assumptions (a strategy selected based on the evaluation presented in Chapter 7).

Where the effect of tapping rate or group was examined ANOVA were used. Outliers were identified using the methods described in Chapter 4 and unless the removal of outliers affected the outcome of the analyses, outliers are not reported further.

8.3. Results

8.3.1. Matched groups descriptive statistics

Descriptive statistics for the three participant groups are presented in Table 8.2. The Clinic group had also completed additional measures in their clinical assessment; these are presented in Table 8.3. A series of one-way ANOVAs confirmed significant group differences on the measures of single word reading, verbal and non-verbal reasoning, non-word and irregular word reading, and the ADHD-IA and ADHD-HI symptoms ratings. Group differences were not found for the Opposite World Increase, Score!, Digit Span and RAN measures. Post-hoc comparisons were used to further interrogate the group differences, with the alpha level set to 0.01, to control for number of comparisons conducted.

The Clinic group scored significantly below the CA group on the Reading and Spelling measures ($p<0.01$) but did not differ from the RL matched controls ($p>0.40$); this result is expected given that this measure was used for the participant matching. The Clinic group also scored significantly below the CA matched group ($p<0.01$) on the non-word and irregular word reading composites, but at a similar level to the RL matched group ($p=0.22$), further supporting the suitability of the group matching. The difference between the two control groups on these measures was not significant ($p=0.07$).

The CA group showed particularly high verbal reasoning scores (compared to populations norms) and the Clinic group showed particularly high non-verbal reasoning scores. There was no difference between the Clinic group and the RL matched controls on the measure of verbal reasoning ($p=1.00$), but a significant difference between the Clinic group and the CA group on this measure ($p<0.01$). The Clinic group also scored significantly higher on the measure of non-verbal reasoning than both groups of controls ($p<0.01$). This may have resulted from a selection bias in recruiting the Clinic group sample from a clinic setting in which children are privately referred for assessments. For the ADHD-IA symptoms, the Clinic group had significantly higher symptom ratings than either the CA or RL groups ($p<0.01$). For the reports of ADHD-HI symptoms, the difference between the Clinic group and the other two groups did not reach significance ($p=0.07$). Overall, these group differences indicate that the participant matching was appropriate with the Clinic group being different from the CA group but similar to the RL group on a variety of literacy measures. The high non-verbal reasoning scores of the Clinic group mean that caution is required in controlling for reasoning skills in the later analyses.

8.3.2. Motor timing performance

8.3.2.1. Reaction time

The Clinic group had a similar mean reaction time to the CA group (Clinic group mean=348ms, SD=60ms, CA group mean=348ms, SD=87ms) and the RL group were slower to respond (372ms, SD=97ms). An ANOVA showed that there was no significant effect of group on reaction times ($F_{(3,36)}=0.43$, $p=0.65$).

8.3.2.2. Motor timing accuracy and variability

Mean IRIs, standard deviations and CV values across the five response rates are shown in Figure 8.1. Across the rates and groups, mean IRIs were shorter than the target IOIs by 10-55ms.

A series of one-way ANOVAs confirmed that there was no effect of group on mean IRI or IRI SD at any of the five tapping rates ($p>0.26$). However, when outliers were dealt with a main effect of group on SD of IRIs was found for the 673ms IOI condition ($F_{(2,41)}=4.34$, $p<0.05$, $\eta^2=0.18$). Post-hoc comparisons indicated that this resulted from a significant difference in IRI SD between the Clinic group and the CA matched group ($p<0.05$), but not between the other groups ($p>0.38$). All three outliers had high variability, including one participant from the Clinic group and two from the CA group. The CA outliers were relatively young (108 months and 118 months) but did not have outlying values on the literacy or attention variables. On the CV measure, no effects of group were found ($p>0.27$).

The effect of tapping rate on IRI SD (within trial variability) was tested separately for each group and was found to be significant for all three groups (Clinic group $F_{(4,32)}=13.27$, $p<0.01$, $\eta^2=0.62$, CA group

Greenhouse-Geisser Corrected $F_{(1.5,20.8)}$=15.63, p<0.01, η^2=0.53, RL group $F_{(4,60)}$=32.59, p<0.01, η^2=0.69). These effects were in the direction predicted, with lower variability at higher speeds. When variability was normalised by mean IRI using the coefficient of variation measure, the effect of rate was eliminated for all three groups (Clinic group $F_{(4,32)}$=0.37, p=0.83, η^2=0.04, CA group Greenhouse-Geisser Corrected $F_{(2.2,31.7)}$=0.57, p=0.68, η^2=0.03, RL group $F_{(4,60)}$=1.04, p=0.40, η^2=0.07).

Table 8.2: Descriptive statistics for reasoning, literacy and attention measures for the three participant groups

Measure	Clinic Group		CA Controls		RL Controls	
	Mean	(SD)	Mean	(SD)	Mean	(SD)
Verbal Reasoning	108.2[a]	(8.4)	122.9	(13.2)	109.9	(13.3)
Non-verbal Reasoning	110.1[ab]	(10.5)	95.9	(10.4)	97.6	(12.4)
Music Experience (years)	2.3	(1.4)	1.3	(1.2)	0.7	(1.0)
Reading Ability	89.5[a]	(12.4)	105.8	(11.4)	96.4	(14.1)
Spelling Ability	87.3	(13.2)	102.4	(13.4)	89.7	(15.8)
Non-word Reading Score (max = 30)	17.7[a]*	(5.7)	24.4	(5.5)	20.9	(7.5)
Non-word Reading Time (seconds)	66.9	(27.2)	41.8	(15.1)	56.5	(18.6)
Irregular Word Reading Score (max = 30)	15.3[a]*	(4.5)	19.7	(3.7)	17.4	(3.9)
Irregular Word Reading Time (seconds)	55.7	(21.9)	31.8	(12.8)	48.8	(19.5)
Digit Span	97.5	(11.7)	90.9	(13.9)	91.9	(11.5)
Rapid Naming	97.0	(12.9)	103.8	(15.0)	100.9	(10.0)
ADHD-IA (max = 27)	14.1[ab]	(6.2)	4.5	(7.2)	4.3	(5.1)
ADHD-HI (max = 27)	7.8	(5.6)	3.1	(4.6)	3.2	(6.4)
Opposite World- Same World Time Increase	30.3	(13.1)	34.6	(23.1)	34.0	(29.4)
Score! Sustained Attention Task	97.0	(11.1)	91.6	(15.9)	87.5	(16.2)

*All scores are standard scores (Mean = 100, SD = 15) unless otherwise indicated; CA-chronological age matched group, RL-reading level matched group; [a]difference from CA group (p<0.01); [b]difference from RL group (p<0.01); *group comparison conducted on error-time composite measure.*

Table 8.3 Additional descriptive statistics for clinic group

	N	Mean	(SD)
Working Memory Index	15	97.7	11.3
Processing Speed Index	16	93.8	11.2
Pseudo-word Naming	14	87.3	10.2
TOWRE Total	12	87.0	10.4
CTOPP Elision	16	7.9	2.3
CTOPP Composite	12	85.3	14.2

N-number of Clinic participants who had completed these measures in their assessment.

8.3.2.3. Decomposed motor timing variables

This analysis was conducted on the first non-violating trial from each participant (see Data Analysis Section 8.2.4 and discussions in Chapter 7). The presence of violations of the model is explored further in Section 8.3.4. Due to this methodology, for some of the response rates, some children did not have any trials at a particular response rate which were non-violating. Because of this, caution was exercised in this analysis, such that comparisons were only made within any one tapping rate, and not across rates.

The estimates of timekeeper and implementation variance for the three groups are shown in Figure 8.2. One-way ANOVAs demonstrated that there were no group differences at any tapping rate on the measures of timekeeper ($p>0.78$ for all rates) or implementation variance ($p>0.12$ at all rates), or the lag one serial correlation (LOSC) variable (a measure of the ratio between timekeeper and implementation variance) ($p>0.45$ for all rates). In the absence of a statistical analysis of rate, examination of Figure 8.2 suggests an effect of rate was present for timekeeper variance, but to a lesser extent for implementation variance. Only the Clinic group showed a trend towards increased implementation variance at the slower rates.

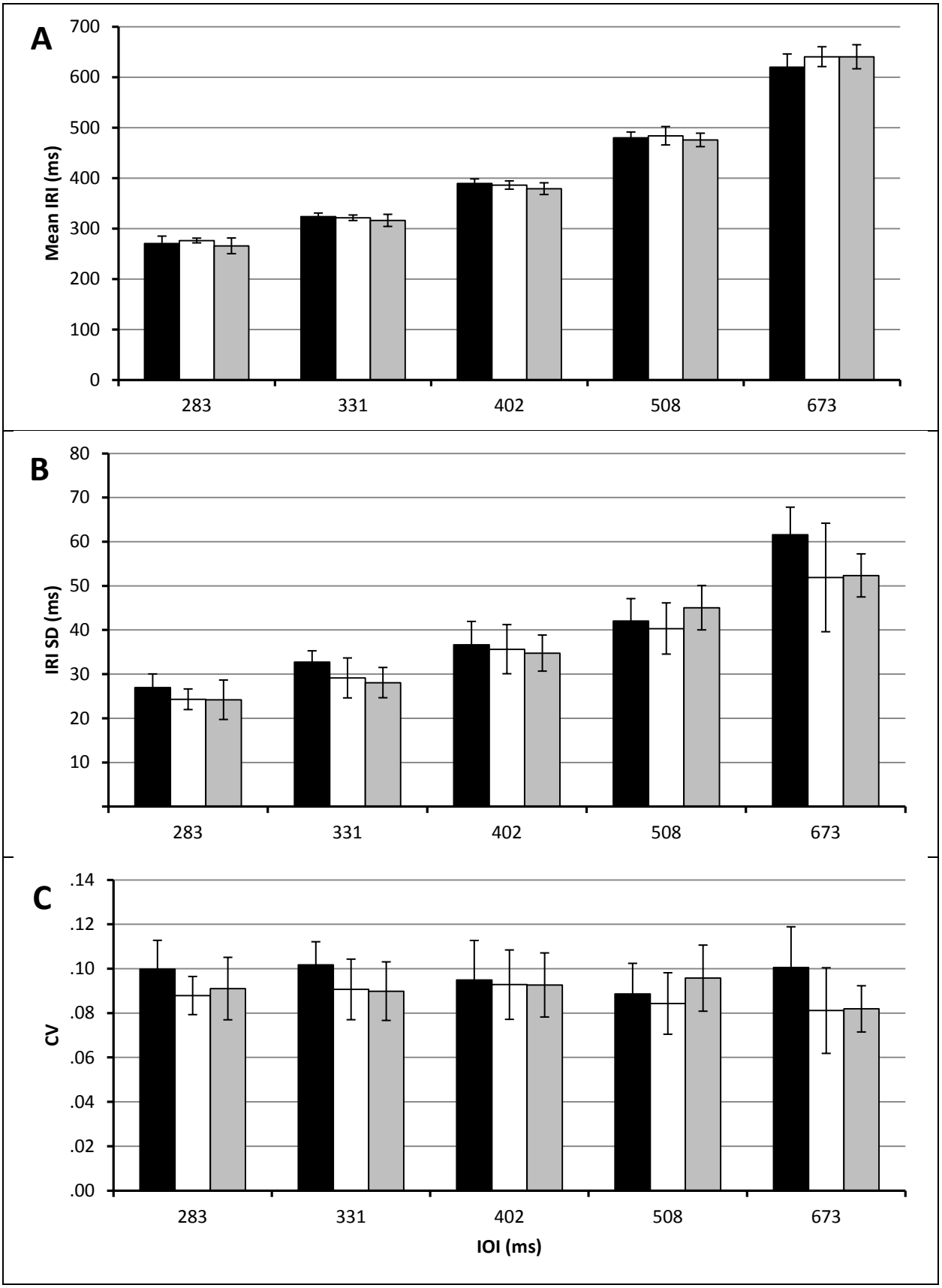

Figure 8.1: Group means for (A) IRI mean, (B) IRI SD and (C) CV

Bars represent groups (black-Clinic group, white-CA group, grey-RA group) and error bars represent 2 SEM, CV measure is dimensionless.

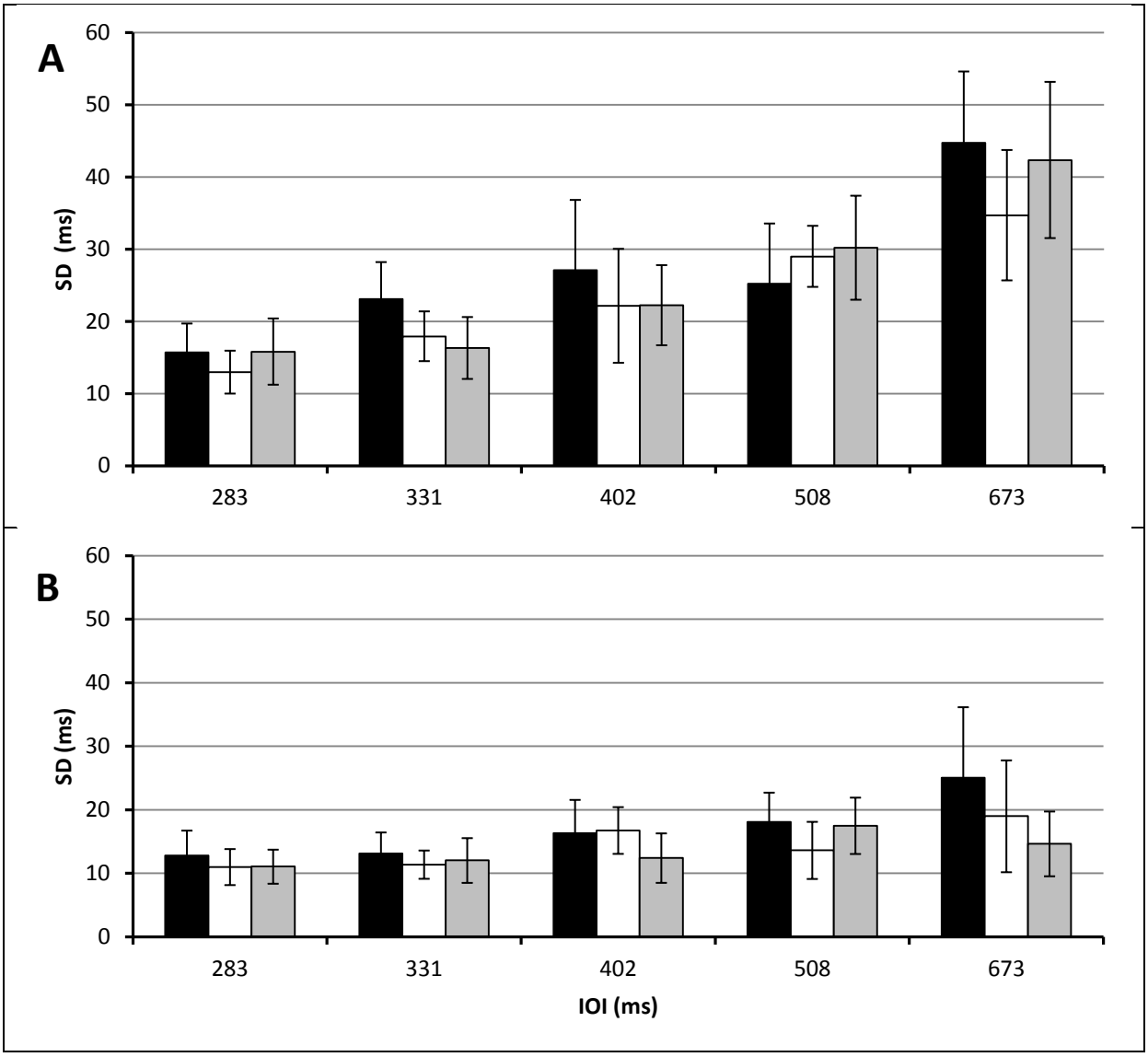

Figure 8.2: Estimated timekeeper (A) and implementation (B) variance (presented as SD) for the three groups across the five response rates.
Bars represent groups (black-Clinic group, white-CA group, grey-RA group) and error bars represent 2 SEM.

8.3.3. Relationships between motor timing performance and cognitive/behavioural predictors

Pearson's product moment correlations were used to examine the relationships between timing performance and predictors of literacy and attention abilities. To reduce the number of comparisons, only the timing variability measure (SD of IRIs) was used to operationalise timing performance. As described in Chapter 3, this variable has been found to distinguish significantly between participant groups in studies of motor timing in dyslexia and has been found to be moderately associated with literacy variables. Correlations were performed across all participants (i.e. across groups). To ensure that data from all participants came from distributions with similar properties and could be combined, scatterplots of the associations between the timing, literacy and attention variables were

examined. These indicated that, across the measures, the groups showed similar distributions of data points and that the distribution for any one group was not responsible for the correlations found. Therefore, performing correlations across all the children in the sample was considered appropriate.

Zero-order and partial correlations, controlling for age and reasoning ability, were performed to examine the relationships between IRI SD across the five tapping rates and the measures of literacy, literacy component skills and attention (Table 8.4). Variables represented by standard scores already accounted for the contribution of age but age was additionally controlled for in the partial correlations to remove its contribution from all variables including the measures of timing performance. All variables had been assessed and corrected for outliers.

Correlations were expected between motor timing variability and measures of literacy. Significant correlations of a moderate size were found between tapping variability on trials with 402ms IOIs and the measures of spelling, rapid naming, digit span and the ADHD-IA and ADHD-HI ratings accounting for 19-34% of the variance in the cognitive/behavioural measures. Rapid naming performance was also significantly associated with timing variability on the 508ms IOI trials and irregular-word naming was associated with variability on the 283ms IOI trials. The direction of these correlations indicates that children with high literacy/component skill scores or low ADHD ratings had less within participant motor timing variability. A number of medium sized correlations between timing variability and the literacy and attention measures (spelling, rapid naming, ADHD-IA and ADHD-HI) were also present, particularly on the trials with 331, 402 and 508 ms IOIs, which did not reach the 0.01 alpha threshold, with rapid naming performance being associated with performance variability at all response rates except the slowest (accounting for 12-33% of the variance in timing variability). The reaction time measure was not related to IRI variability at any of the four rates of tapping.

As described in Chapter 6 (Section 6.5.1) a limitation of conducting a large number of correlations in this manner is that there is a high probability of making Type I errors. If a strict Bonferroni correction were applied to the alpha values on which the significance of these relationships were judged, the accepted p-value would be reduced to 0.0021 (given the number of comparisons). This may be rather conservative given that some of the cognitive and behavioural predictors included in the correlation matrix overlap (with mutual correlations between reading, spelling, rapid naming, irregular-word reading and non-word reading) such that several correlations may come from the same family of comparisons. However, using this conservative corrected p-value several of the correlations would remain significant, in particular those between spelling and tapping variability with 402ms IOIs (where p=0.0003), between rapid naming and tapping variability with 508 and 402ms IOIs (p=0.0003 and p=0.0005) (but not the association with 331ms IOIs, where p=0.005),

between the ADHD-IA ratings and variability with 402ms IOIs (p=0.0005) and that between the ADHD-HI ratings and variability with the 402ms IOIs (p=0.0018).

For the cognitive/behavioural predictors which were significantly correlated with timing variability, the relationships with the decomposed components of timing performance were also examined using further partial correlations, again controlling for age and reasoning ability. Table 8.5 shows the correlations between variance components in the 402ms trials and the literacy and attention variables that were significantly associated with IRI SD. Here, timekeeper variance was not related to spelling, rapid naming or ADHD ratings but implementation variance was significantly associated with both the ADHD-IA and ADHD-HI behaviour ratings (p=0.0022 and p=0.0035 respectively), with high symptom ratings being associated with greater implementation variance.

Similar correlations were performed for the trials with 331ms IOIs and although the rapid naming measure correlated with IRI SD on these trials, neither timekeeper nor implementation variance were significantly associated with rapid naming (r=-0.30, p=0.07 and r=-0.31, p=0.06 respectively). Rapid naming was also correlated with IRI SD for the 508ms trials, but again the correlations with timekeeper and implementation variance at this speed were not significant, although the correlation with timekeeper variance approached significance (timekeeper variance: r=-0.37, p=0.02 and implementation variance r=-0.09, p=0.61). When the associations between irregular word reading and timing variability on the 283ms trials were examined, the associations with timekeeper and implementation variance were also not significant (r=0.30, p=0.07 and r=0.15, p=0.38 respectively). Applying a Bonferroni correction to these comparisons to control for the number of comparisons conducted (resulting in a p-value of 0.0001) would mean that none of the associations described, between the decomposed timing variables and the cognitive/behavioural predictors, would remain significant.

Table 8.4: Correlations between motor timing performance and measures of literacy and attention

	673ms IRI SD	508ms IRI SD	402ms IRI SD	331ms IRI SD	283ms IRI SD	Reading	Spelling	RAN	Digit Span	Non-Word Reading	Irregular Word Reading	ADHD-IA	ADHD-HI	Attention Control	Sustained Attention	Reaction Time
673ms IRI SD		0.53**	0.48**	0.36*	0.46**	-0.15	-0.24	-0.33*	-0.22	0.21	0.26	0.27	0.10	-0.04	0.01	-0.16
508ms IRI SD	0.53**		0.59**	0.55**	0.42**	-0.26	-0.34*	-0.50**	-0.22	0.20	0.29	0.32	0.21	0.13	-0.26	-0.09
402ms IRI SD	0.50**	0.61**		0.58**	0.48**	-0.22	-0.41*	-0.45**	-0.42**	0.23	0.22	0.40*	0.48**	-0.01	-0.32	0.03
331ms IRI SD	0.38*	0.53**	0.61**		0.42**	-0.16	-0.17	-0.41*	-0.24	0.26	0.29	0.30	0.37*	0.06	0.02	0.17
283ms IRI SD	0.49**	0.46**	0.53**	0.43*		-0.12	-0.20	-0.32*	-0.08	0.24	0.27	0.10	0.08	-0.05	-0.14	0.00
Reading	0.01	-0.30	-0.40*	-0.28	-0.23		0.92**	0.52**	0.25	-0.72**	-0.86**	-0.67**	-0.40*	0.01	0.08	0.07
Spelling	-0.12	-0.35*	-0.58**	-0.26	-0.32	0.87**		0.53**	0.38*	-0.70**	-0.82**	-0.65**	-0.37*	0.07	0.12	-0.02
RAN	-0.31	-0.57**	-0.56**	-0.47**	-0.34*	0.45**	0.49**		0.45**	-0.58**	-0.53**	-0.56**	-0.40*	0.05	0.27	-0.09
Digit Span	-0.19	-0.24	-0.44**	-0.29	-0.10	0.16	0.34*	0.43*		-0.29	-0.25	-0.29	-0.21	0.25	0.14	-0.20
Non-Word Reading	0.14	0.16	0.38*	0.27	0.29	-0.63**	-0.63**	-0.54**	-0.30		0.82**	0.56**	0.41*	-0.15	0.08	-0.03
Irregular Word Reading	0.16	0.27	0.36*	0.37*	0.40*	-0.76**	-0.70**	-0.47**	-0.20	0.73**		0.64**	0.38*	-0.11	-0.01	-0.02
ADHD-IA	0.20	0.38*	0.56**	0.41*	0.14	-0.48**	-0.49**	-0.48**	-0.21	0.44**	0.49**		0.69**	0.09	-0.06	0.07
ADHD-HI	0.07	0.24	0.51**	0.42*	0.10	-0.44**	-0.37*	-0.38*	-0.17	0.47**	0.42*	0.74**		-0.05	-0.28	-0.15
Attention Control	-0.07	0.08	-0.07	0.06	-0.02	0.03	0.15	0.01	0.28	-0.16	-0.17	0.13	-0.05		-0.17	0.19
Sustained Attention	0.04	-0.21	-0.27	0.03	-0.18	0.10	0.09	0.35*	0.13	0.08	0.03	-0.09	-0.30	-0.11		0.00
Reaction Time	-0.19	-0.12	0.02	0.18	0.01	0.17	0.04	-0.09	-0.19	-0.05	-0.08	0.06	-0.16	0.17	0.03	

*Pearson's product moment correlations (top right) between psychometric variables of interest and motor timing performance, with partial correlations controlling for verbal, non-verbal reasoning and age (bottom left); *p<0.05; **p<0.01.*

Table 8.5: Partial correlations with timing performance variables on trials with 402ms IOIs

	402 ms IRI SD	TK variance	IMP variance	Spelling	RAN	Digit Span	ADHD-IA
Timekeeper variance	0.55**						
Implementation variance	0.48**	-0.25					
Spelling	-0.39*	-0.05	-0.23				
RAN	-0.41*	0.08	-0.27	0.47**			
Digit Span	-0.47**	-0.09	-0.32*	0.34*	0.33*		
ADHD-IA	0.47**	0.16	0.49**	-0.34*	-0.43**	-0.15	
ADHD-HI	0.38*	0.09	0.47**	-0.27	-0.32	-0.11	0.77**

*Partial correlations controlling for verbal, non-verbal reasoning and age. TK-Timekeeper variance; IMP-Implementation variance; *p<0.05; **p<0.01.*

8.3.4. Sources of violations of the Wing-Kristofferson model

The Wing-Kristofferson model was applied to the data from each trial. A number of trials were found to violate the basic assumption that $-0.5 < \rho_I(1) < 0$ and are detailed in Table 8.6. Violation of the lower bound of this autocorrelation assumption gives rise to negative timekeeper variance and violation of the upper bound gives rise to negative implementation variance. There were no differences between the groups in the number of trials which violated this assumption (χ^2 =1.40, df=2, p=0.50), with 37.5% of trials in the CA matched group, 34.2% in the RL matched group and 30.9% in the Clinic group violating this assumption.

The analyses presented in the Chapter 7 demonstrated that trials which violate the bounds of the predicted autocorrelation value typically contain elements of non-stationarity and/or dependencies between intervals at lags beyond one. It was also discussed how such features of timing behaviour can be used to characterise the nature of timing performance in populations of interest. Therefore, the extent to which the groups of children showed such instances of drift and dependencies was examined. For these analyses, averages were calculated across both violating and non-violating trials to establish average performance characteristics for the children in each group.

Table 8.6: Percentage of trials which did not meet the autocorrelation assumption and remaining trials

IOI (ms)	Clinic group (%)			CA matched group (%)			RL matched group (%)		
	Violations where:		Remaining trials	Violations where:		Remaining trials	Violations where:		Remaining trials
	$\rho_I(1) < -0.5$	$\rho_I(1) > 0$		$\rho_I(1) < -0.5$	$\rho_I(1) > 0$		$\rho_I(1) < -0.5$	$\rho_I(1) > 0$	
283	55.2	3.4	41.4	46.7	0.0	53.3	36.0	0.0	64.0
331	29.0	0.0	71.0	30.0	3.3	66.7	27.6	3.4	69.0
402	27.6	6.9	65.5	19.4	3.2	77.4	6.9	13.8	79.3
508	25.8	3.2	71.0	37.5	6.3	56.3	16.7	16.7	66.7
673	31.3	6.3	62.5	18.8	6.3	75.0	21.7	13.0	65.2

The Wing-Kristofferson model predicts that the lag-one autocorrelation falls within the bounds of zero to minus one half. Violations of the lower limit result from negative timekeeper variance and violations of the upper limit result from negative implementation variance.

8.3.4.1. Autocovariance for lags beyond one

Autocovariance at lags beyond one provides a measure of the extent to which timing is controlled in an open-loop fashion, as predicted by the Wing-Kristofferson model. As described in Chapter 7, autocovariance values averaged across trials at lags two to five were calculated and plotted with 99% confidence intervals. These were compared to expected biased values of autocovariance calculated using Equation 12 (as recommended by Vorberg & Wing, 1996; see Chapter 7) and closed loop timing was considered to be present if the confidence intervals for the observed data did not incorporate the unbiased estimate. Table 8.10 shows the lags at which violations occurred for each of the three participant groups across tapping rates. The Clinic group showed autocovariance at lags beyond one which exceeded the expected bounds on both the fastest and slowest finger tapping rates, but not consistently across all rates. The matched controls also showed instances of closed-loop timing, suggesting that on occasion all children use a closed-loop form of timing control, but not consistently across ISI conditions.

Table 8.7: Instances of autocovariance at lags two to five violating the model assumptions

IOI (ms)	Clinic	CA Matched	RL Matched
283	Lag 2	-	-
331	-	-	-
402	-	-	Lag 3
508	-	-	-
673	Lag 2	Lag 2	-

The lags listed are instances where the predicted estimate of the value of autocovariance exceeded the 99% confidence intervals of the observed autocovariance value for the group.

8.3.4.2. Stationarity

Stationarity in the time series was examined using both linear and non-linear methods for calculating slope. Linear drift was defined as the slope of a regression line between mean IRI and tap number with slope indicating whether responses speeded up (negative slope) or slowed down over time (positive slope). The method described by Madison was used to quantify drift irrespective of its form (linear or otherwise), using a non-parametric estimator (Madison, 2001a, 2001b). This estimator was calculated as a least squares regression line for a plot of median of absolute differences between intervals for lags up to n/2 (i.e. up to lag 15) against lag number $f'(k)$, as described in Chapter 7.

Group medians for linear drift and associated r^2 values are given in Table 8.8 and Figure 8.3. Examination of the table reveals that trials with negative drift had smaller mean IRIs. There was a higher percentage of trials with negative drift at the slow tapping rates, especially for the Clinic group where the majority of trials with 673 and 508ms IOIs had negative rather than positive slope (72 and 82% respectively), despite achieving similar IRIs to the other groups (Figure 8.). At the fastest tapping speeds the number of trials with negative or positive slope was closer to 50%, indicating that drift was of no consistent direction. The r^2 values are all small, such that the linear drift explained little variance in mean IRI. The extent of overall drift across rates and groups was examined using the median slope values, disregarding the sign of the slope. One-way ANOVAs conducted at each response rate showed no significant differences in average slope between the groups ($p>0.10$). A repeated measures ANOVA was conducted for each group to assess the effect of tapping rate on drift and showed an effect of rate for each group (Greenhouse Geisser corrected, CA matched $F_{(2.5,34.9)}=5.23$, $p<0.01$, $\eta^2=0.27$; RL matched $F_{(2.7, 40.9)}=8.45$, $p<0.01$, $\eta^2=0.36$; Clinic group $F_{(2.4,19.3)}=5.67$, $p<0.01$, $\eta^2=0.42$).

Estimates of drift using the $f'(k)$ method are presented in Table 8.9 and Figure 8.4. One-way ANOVAs conducted at each response rate showed no significant differences between the groups in the amount of drift ($p>0.27$ for all comparisons except for the difference between the Clinic and RL

groups at 3.5Hz where p=0.07). Repeated measures ANOVAs showed that there was no effect of tapping rate on drift for either the Clinic group ($F_{(4,32)}$=1.02, p=0.41, η^2=0.11)or the CA matched group ($F_{(4,56)}$=0.24, p=0.92, η^2=0.02), but a significant effect for the younger typically developing (RL) group ($F_{(4,60)}$ = 5.06, p<0.01, η^2= 0.25), with more drift at the slowest tapping speeds.

Given that the presence of drift is not accounted for in the Wing-Kristofferson model, it was important to establish whether drift contributed to the relationships between the cognitive/behaviour predictors and timing performance. Therefore partial correlations were performed, controlling for age and reasoning ability (Table 8.10). These were conducted only for timing performance on the 402ms trials where most frequent relationships with the cognitive/behavioural predictors were found. After controlling for age and reasoning ability, both drift components were significantly associated with timing variability for the 402ms trials. The drift components were not significantly associated with spelling. The association between rapid naming and the linear drift measure approached significance (p=0.05), as did the associations between digit span and ADHD-IA and the non-linear drift measure (p=0.06 and 0.07 respectively).

Rapid Naming ability was moderately associated with both performance variability (SD of IRIs) and linear drift. Therefore a stepwise linear regression was performed to compare the contribution of each measure to rapid naming ability. Reasoning ability and age were entered at the first step. Performance variability and linear drift (on trials with 402ms IOIs) were then added incrementally at the second and third steps to examine their relative contribution to rapid naming. The analysis showed that neither model was significant at step 1 or 2 (p>0.07), despite both performance variability and slope individually predicting variance in rapid naming (performance variability predicted 10%, β=-0.35, t(41)=-2.39, p<0.05 and slope predicted 7%, β=-0.31, t(41)=-2.02, p=0.05).

Table 8.8: Presence of linear drift

IOI (ms)	Group	Trials with Negative Slope				Trials with Positive Slope			For all trials		
		%	Mean IRI	Median Slope	r^2	Mean IRI	Median Slope	r^2	Median Slope	Slope (disregarding sign)	r^2
283	Clinic	46.9	271.6	-0.69	0.07	280.3	0.61	0.07	0.17	0.61	0.07
	CA	37.5	251	-0.18	0.01	274.5	0.36	0.02	0.11	0.24	0.01
	RL	48.0	263.5	-0.21	0.01	282.5	0.74	0.04	0.05	0.44	0.03
331	Clinic	40.0	315.7	-0.46	0.01	328.7	0.55	0.02	0.14	0.51	0.02
	CA	51.6	320.3	-0.49	0.02	322.9	0.42	0.03	-0.09	0.48	0.02
	RL	62.5	311	-0.51	0.03	324.7	0.40	0.03	-0.19	0.47	0.03
402	Clinic	62.1	385.6	-0.75	0.03	399	0.58	0.02	-0.20	0.74	0.02
	CA	62.1	382.2	-0.53	0.03	396.8	0.29	0.01	-0.18	0.43	0.02
	RL	68.8	376.7	-0.53	0.02	386.2	0.73	0.04	-0.28	0.60	0.03
508	Clinic	82.8	479.5	-0.97	0.04	476.6	1.04	0.05	-0.88	0.98	0.05
	CA	54.8	475.8	-0.72	0.02	487.7	0.58	0.01	-0.19	0.65	0.02
	RL	48.4	479.1	-0.93	0.04	475.8	0.63	0.01	0.04	0.78	0.02
673	Clinic	72.0	604.6	-1.60	0.05	658.2	1.01	0.02	-1.12	1.42	0.05
	CA	65.5	620.3	-1.10	0.06	671.6	1.44	0.05	-0.47	1.33	0.05
	RL	60.0	632.7	-1.29	0.07	646.1	1.21	0.04	-0.77	1.29	0.05

The percentage of trials with negative slope is presented; all other trials had positive slope. Mean IRI is shown for trials with drift along with median slope and r^2 coefficients.

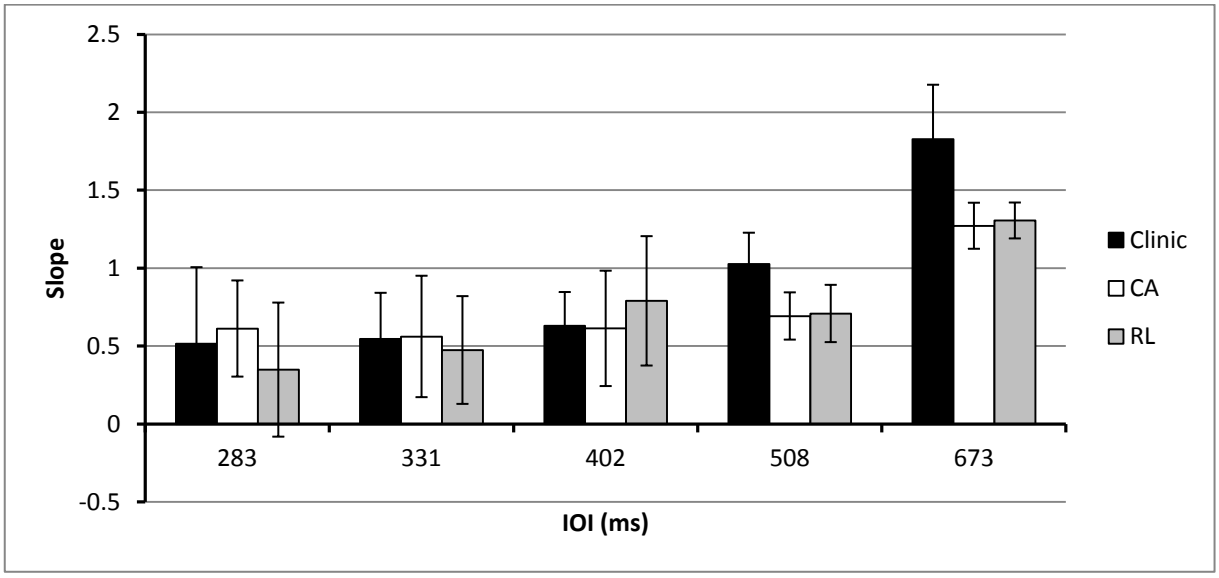

Figure 8.3: Presence of linear slope

Values are median slope values (disregarding sign). Error bars represent ±2 SEM.

Table 8.9: Presence of slope ($f'(k)$)

IOI (ms)	Group	Median Slope	Median Slope (disregarding sign)	r^2
283	Clinic	0.22	0.61	0.12
	CA	-0.08	0.52	0.10
	RL	0.12	0.17	0.08
331	Clinic	0.24	0.26	0.12
	CA	0.30	0.52	0.13
	RL	0.12	0.49	0.09
402	Clinic	0.21	0.44	0.09
	CA	0.00	0.51	0.09
	RL	0.23	0.44	0.10
508	Clinic	0.21	0.96	0.13
	CA	-0.15	0.59	0.08
	RL	0.37	0.63	0.09
673	Clinic	0.32	0.39	0.14
	CA	0.00	0.37	0.11
	RL	0.24	0.67	0.10

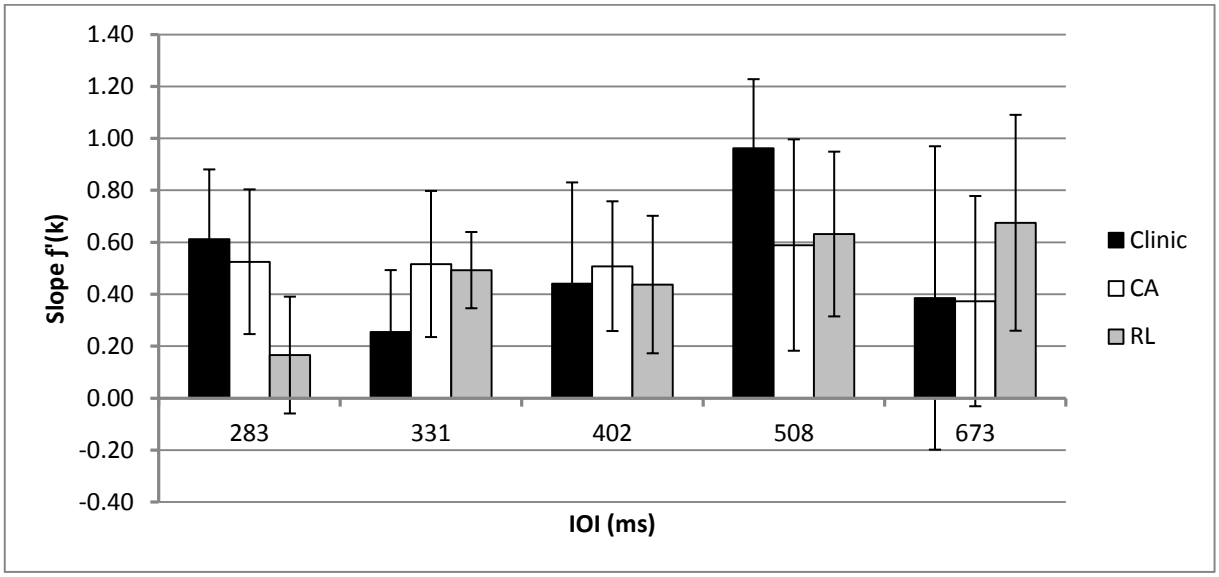

Figure 8.4: Presence of drift ($f'(k)$)

Values are Median slope values (disregarding sign). Error bars represent 2SEM.

Table 8.10: Partial correlations between drift (linear and non-linear), timing performance and cognitive/behavioural predictors

	402 ms IRI SD	Slope (Absolute)	$f'(k)$	Spelling	RAN	Digit Span	ADHD-IA
Slope (Absolute)	0.39*						
$f'(k)$	0.41**	0.22					
Spelling	-0.42	-0.18	-0.25				
RAN	-0.35*	-0.30[a]	-0.11	0.48**			
Digit Span	-0.42**	-0.26	-0.28[b]	0.35*	0.38*		
ADHD-IA	0.51**	0.28	0.29	-0.40**	-0.39**	-0.14	
ADHD-HI	0.43**	0.11	0.26	-0.30	-0.30	-0.10	0.75**

*Partial correlations controlling for verbal, non-verbal reasoning and age * p<0.01; ** p<0.05, a p=0.05, b p=0.06*

8.4. Discussion

The aims of this study were to compare the motor timing performance of children with reading difficulties to the performance of typically developing controls across a range of response intervals. The Wing-Kristofferson statistical decomposition method was used to assess the relative contribution of timekeeper and implementation variance to timing performance in these groups. The relationships between timing variables and measures of literacy and attention were subsequently examined to establish whether the associations between literacy skills and timing ability found in previous studies could be explained by either timekeeper or implementation variance on the tasks. In light of the findings in Chapter 7, the components of timing control which can lead to violations of the Wing-Kristofferson model were also examined.

8.4.1. Accuracy and variability across timing speeds

All three groups demonstrated equivalent response patterns across the different rates of finger tapping, with decreasing variability with increasing inter-stimulus intervals. This effect was accounted for by rate alone being eliminated when variability estimates were rate-normalised using the coefficient of variation measure. This effect of rate is typical in timing research and supports the clock-counter model of timekeeping in which pulses from a hypothetical clock accumulate over time to provide a measure of interval length for behavioural guidance (Gibbon, 1977; Ivry & Hazeltine, 1999; Lewis & Miall, 2009; Pressing, 1999; Wing & Beek, 2002; Wing & Kristofferson, 1973b). In such models higher variability at slower rates is a consequence of the longer period over which signals from the internal clock are accumulated. Here, this was accompanied by a trend for increasing timekeeper variance at slower speeds but no comparable changes for implementation variance.

The observation that the coefficient of variation remains constant across tapping rate suggests that there are no distinct changes in behaviour for any of the groups at any particular speed. Any such abrupt changes can indicate a change in the mechanisms governing responses. For example, large changes in performance variability are found when tapping rates slow down to about 1Hz or 0.75 Hz (1000ms or 1333ms IOIs), where processing is argued to move from automatic or unconscious millisecond timing control mechanisms to more consciously controlled interval timing (Drake & Botte, 1993; Lavoie & Grondin, 2004; Madison, 2001a). No such significant changes were found at any rate across the participant groups in this study.

8.4.2. Group differences in timing performance

The group of children with literacy difficulties performed as well on the motor timing task as the CA and RL matched groups, both in terms of mean IRI and within-participant variability (SD of IRIs). These results contrast with those reported in earlier studies of motor timing in children with dyslexia which found greater performance variability on unimanual timing tasks in children with dyslexia compared to controls (Thomson & Goswami, 2008; Wolff et al., 1984). Here, the only significant group difference in performance variability was found between the Clinic group with reading difficulties and the CA matched group at the slowest tapping rate (673ms IOIs; 1.5Hz) but no difference between the Clinic group and RL controls. Timing performance data across age groups indicates that children may only develop an ability to accurately entrain to tempos as slow as 600ms (1.6Hz) between the ages of 8 and 12 (McAuley et al., 2006). Therefore, participants in the younger age matched group and the Clinic group may have had a less mature timing system and had not yet developed the ability to entrain to slow tapping rates.

Group differences at the slowest tapping speed are in line with the results from Wolff's earlier study which found increased timing variability in a group of children with reading difficulties (who were reading at least 2 years below their expected grade level) when tapping with one hand in the continuation phase at 650ms intervals (1.53Hz) (Wolff et al., 1984). Thomson and Goswami (2008) in comparison found that a group with dyslexia (reading on average 33 standard score points or two SD below controls) had greater variability than controls only on an unpaced tapping task with 400ms intervals (2.5Hz) and not at the slower rates of 500 and 666ms intervals (2 and 1.5Hz). A group difference would be expected in Thomson and Goswami's study if the children with reading difficulties had a developmental delay in the ability to tap at slow speeds as described above. Their results may be explained by neither the experimental nor control group having developed the ability to entrain to slower rates, resulting in the lack of group differences at these speeds. In the current study, entrainment ability at slower speeds in the Clinic group may have been scaffolded by reading experience (Beattie & Manis, 2011), given that the Clinic group were not as severely discrepant in their reading abilities as those in these previous studies. These participants were also drawn from an

assessment clinic where referrals are made privately by parents who are often keen to gain support for their children and may provide additional reading support at home.

The development of the ability to entrain to a rhythm with longer temporal intervals may mirror the development of other skills which require the use of intervals of different lengths (McAuley et al., 2006). For example, early in infancy sensitivity to events with rapid onsets is required to facilitate processing of phonetic categories, and events with slower interval rates become more important later in development as words, phrases and sentences are processed (Werker & Tees, 1999, 2005). This development may be mirrored in other rhythmic entrainment skills, but whether the two are causally linked has not yet been confirmed. Combining the evidence from across timing and language studies does however suggest that by the time children are able process more complex syllabic forms (Kuhl, 2004), that require slow rate processing (Goswami, 2011), they are able to successfully entrain a timed behavioural output comparable frequencies (McAuley et al., 2006).

Longitudinal assessments of timing and literacy skills indicate that timing abilities may support the development of language skills that rely on temporal processing of brief intervals. Rhythmic abilities in the early years of school are related to reading performance at Grade 5 (David et al., 2007) and a similar developmental effect of prosody or rhythm has been suggested elsewhere (Beattie & Manis, 2011; Richardson et al., 2004; Wood & Terrell, 1998). Such evidence supports the hypothesis that rhythmic processing ability is important in building appropriate phonological representations through the use of prosodic temporal cues in speech (Holliman et al., 2008, 2010). Crucially however, particular timing skills, such as the use of rhythm in language processing, may only be important once a certain level of reading has been attained (Wood & Terrell, 1998). Here, the Clinic group may have been slower to develop the ability to entrain to slower rates, behaving more like the RL group than the CA group. However, the potential influence of reduced reading experience could not be confirmed within the present group comparison data, given the limits of matched groups designs (Bryant & Goswami, 1986). Future investigations would benefit from using longitudinal designs to help understand these developmental questions.

Consistent with the results described above, no group differences were present for the implementation or timekeeper variance components estimated by the Wing-Kristofferson model. In addition, group differences in decomposed variance estimates were not present for the trials with the 673ms IOIs (1.5Hz), where a group difference in overall performance variability was present. Therefore the difference in performance variability between the Clinic group and the CA controls at this speed cannot be attributed to the presence of more variable outputs from the internal timekeeper or to greater variability in the motor system's implementation of timekeeper pulses. In line with the maturational hypotheses described above, it is possible that the difference in the Clinic group at the slowest speed was due to general immaturity across the timing system, leading to

greater overall variability rather than the lack of precision in either the timekeeper or implementation components alone.

An alternative explanation for Thomson and Goswami's earlier (2008) finding that children with dyslexia differ from controls in timing variability when tapping at 2.5Hz is that such group differences in timing performance are only discernible when there is a large discrepancy in reading scores between the groups. The reading impaired group in that study were reading on average 2 standard deviations below the control group, a far greater difference than the differences between the groups in this study. This difference in timing performance between the group with reading difficulties and the CA controls was not replicated here at 2.5Hz and casts doubt over the putative relationship between timing difficulties and dyslexia. The correlations discussed below were conducted to further interrogate the presence of such relationships. Such analyses were also conducted by Thomson and Goswami, however, their inclusion of groups with extreme differences in reading abilities may have had the effect of amplifying any statistical association between reading and timing that might be present across the population. In the present study the inclusion of less severely discrepant readers and matched controls allowed the extent of the relationships to be clarified. As described below, these analyses replicated the associations between literacy and timing variability even after severe statistical corrections were applied for multiple comparisons.

8.4.3. Correlations with cognitive/behavioural predictors

Despite not finding the expected group differences in timing variability or decomposed variance, the predicted associations between timing variability and the literacy and attention variables were present and significant. Within-participant timing variability, when tapping with 402ms intervals (2.5Hz), was associated with spelling, rapid naming and digit span performance as well as with (teacher/parent) ratings of inattention and hyperactivity-impulsivity symptoms. These medium sized associations were present even after controlling for age and reasoning performance; variables which have been shown to influence timing performance (Madison et al., 2009; Madison, 2011; McAuley et al., 2006). The associations between variability with 402ms intervals and the literacy variables of spelling and rapid naming, as well as those with the ADHD symptoms also survived Bonferroni correction for multiple comparisons. Children who had lower literacy scores or a greater number of symptoms of inattention or hyperactivity showed more variability in their timed motor outputs, replicating the associations found at a faster tapping speed in the Chapter 6. The correlations indicated that timing variability predicted around 30% of the variance in the literacy and attention measures; associations that are of similar magnitude to those found by Thomson and Goswami (2008). In addition to these associations, similar correlations (with smaller effect sizes) were found between variability and irregular word reading and/or rapid naming performance for the trials with

508, 331 and 283ms intervals (2, 3 and 3.5Hz; although these did not survive correction for multiple comparisons).

In this exploratory study, a number of correlational analyses were performed to examine the pattern of associations across the timing measures, investigating whether clusters of associations were found for particular motor timing rates or cognitive/behavioural predictors and the effect of multiple comparisons was assessed post-hoc. Clusters of significant correlations between literacy and motor timing at 2.5Hz that had been predicted based on prior evidence remained even after these corrections were applied. Unfortunately it is not possible to evaluate whether the correlations found in previous studies would have also survived such controls for multiple comparisons because the authors did not provide exact p-values for their correlations despite carrying out a similar (or greater number) of comparisons (Thomson & Goswami 2006; 2008). Calculations indicate that with the number of comparisons conducted in those studies the accepted alpha value would need to be set to a more conservative value of around 0.0002 (notwithstanding the possibility that Bonferroni corrections are overly cautious in circumstances where several correlations may come from the same family of comparisons; see Section , McDonald, 2009). Thomson and Goswami reported that correlations were present at the $p<0.001$ level (e.g. between paced variability and spelling), but exact values were not reported. So although this study replicates relationships between literacy and timing variability at 2.5Hz found by these authors the analyses remain to be repeated in further hypothesis driven experiments where a smaller number of variables is tested.

Examination of the relationship between cognitive/behavioural measures and the decomposed variance components from the Wing-Kristofferson model showed a medium sized, positive relationship between ADHD symptom ratings and implementation variance at the 2.5Hz tapping rate (402ms IOIs). In addition, a medium sized negative association between rapid naming and the timekeeper variance component was found at the 2Hz tapping rate ($p<0.02$). Such relationships had been predicted due to the associations found in Chapter 6 between literacy variables and timekeeper variance, and between measures of ADHD symptoms and implementation variance. These results therefore provide support for the previous evidence that timing performance is differently related to symptoms of ADHD and dyslexia, with attentional difficulties being associated with greater variance in implementation processes and literacy difficulties being linked to greater variance in the timekeeper system. Unfortunately these associations did not survive an alpha correction to account for the number of statistical comparisons conducted. These findings therefore remain to be replicated in future studies in order to establish the true statistical relevance of the results. In this exploratory study, a large number of correlations were conducted, although to attempt to minimise this the correlations with timekeeper or implementation variance were only conducted for trials where overall variability was associated with cognitive predictors. Future studies may benefit from focusing attention on the motor timing rates found to be of interest here (i.e. 2.5Hz) and maximise

their statistical power by only analysing the contribution of timekeeper or implementation variance at this single response rate.

These results, which provide preliminary support for a difference in the type of timing difficulty associated with attentional and literacy difficulties are not unexpected. Although dyslexia and ADHD co-occur together more frequently than is expected by chance (Pauc, 2005; Willcutt & Pennington, 2000) and share underlying cognitive, neurological and genetic risk factors (Pennington, 2006; Plomin & Kovas, 2005), there are differences in their neural aetiologies. For example, ADHD is associated with differences in frontal-striate cortical regions with associated deficits in executive functions. Some deficits associated with ADHD, such as response inhibition, show improvements with pharmacological treatments that affect the dopaminergic pathways of the striate cortex by maintaining higher levels of dopamine (e.g. the dopamine reuptake inhibitor methylphenidate) (Seidman, 2006). In comparison, children and adults with dyslexia show structural and functional impairments across the neural network responsible for reading in the temporo-parietal regions (Goswami, 2008; Vandermosten, Boets, Wouters, et al., 2012). The evidence from the present studies supports a view that the timing difficulties associated with ADHD and reading difficulties may be dissociable when decomposed variance is examined. The results add to others which have found that children with ADHD show timing behaviours, such as hastening phenomenon and drift (increasing IRI over time; Ben-Pazi et al., 2003, 2006; Zelaznik et al., 2012), that have not been reported in children with reading difficulties.

The preliminary indication reported here of differences in the variance components that are associated with literacy ability and attentional control are also consistent with the proposal that motor impairments in dyslexia should be attributed to the presence of co-occurring symptoms of ADHD rather than to dyslexia alone (Denckla et al., 1985; Raberger & Wimmer, 2003; Ramus et al., 2003; Rochelle et al., 2009). Whether the deficit that leads to motor impairments and elevated levels of implementation variance is separate from that which causes the hastening phenomena reported by Ben Pazi and colleagues is unclear. Children showing co-occurring symptoms of dyslexia *and* ADHD may show impairments in both timekeeper and implementation components or a different profile of performance altogether. Multi-deficit models of these developmental disorders suggest that the overlap between the two conditions may be best accounted for by symptoms which do not predict the single disorders alone (McGrath et al., 2011; Willcutt et al., 2010), indicating that a different profile, rather than a simple aggregate of the individual deficits might be expected in children showing symptoms of and/or receiving diagnoses of both disorders.

Significant associations were found between the ratings of ADHD symptoms and the measures of timing performance, as reported in a previous study that included children diagnosed with ADHD (Ben-Pazi et al., 2006), however, similar associations were not present for the behavioural measures

of attentional control and sustained attention. Furthermore, the ADHD symptom ratings were not themselves associated with the behavioural measures despite the Score! and Same World/Opposite World tasks being designed to capture core behavioural aspects of ADHD (Manly et al., 1999). None of the previous studies of motor timing in ADHD (described in Section 3.3, Chapter 3) have assessed the statistical associations between timing performance and cognitive measures of elements associated with ADHD. It is possible that the factors operationalised with the cognitive tasks used here (inattention and inhibition) are not those which are associated with motor timing difficulties, although the impulsivity captured by the Score! task has been proposed as the behavioural component which explains the motor timing deficits in ADHD (Barkley et al., 2001; Rubia et al., 2009; Smith et al., 2002).

In the case of the correlations between timing performance and the behavioural report measure (ADHD-IA/ADHD-HI) it is possible that these measures from the Barkley and Murphy Report Battery did not give sufficiently continuous or representative measures of symptoms. Particularly in the case of the teacher ratings (for the school children in the control groups), teachers may have only used the higher ratings for children exhibiting the most severe behavioural problems in the classroom and their scores may not have distinguished amongst children with minor symptoms using the lower ratings. In comparison, the parents (using the parent-rating scale for the Clinical group) may have been more likely to report even minor symptoms in the potentially stressful environment of the clinical setting, given that stress has been shown to affect reporting on such rating scales (van der Oord, Prins, Oosterlaan, & Emmelkamp, 2006). It has also been found that the parent and teacher ratings of emotional and behavioural symptoms do not correlate well with each other (with relationships as low as 0.27-0.4 for similar questionnaires such as the CBCL or DSM-IV symptom ratings; Aschenbach, McConaughy, & Howell, 1987; Kolko & Kazdin, 1993) and show poor agreement for diagnoses or predicting later behavioural outcomes (Malhi, Singhi, & Sidhu, 2008; Mitsis, McKay, Schulz, Newcorn, & Halperin, 2000; Verhulst, Koot, & Van der Ende, 1994). The potential for gaining rather dichotomous high-low ratings in the school groups and comparatively high ratings in the Clinic group may have increased the chances of finding statistically significant relationships if there was disproportionate representation at the higher ends of the rating scales for those children with some symptoms of ADHD. In the follow up to this study (Chapter 9), further cognitive measures of ADHD symptoms were sought to more accurately measure difficulties across the participant group. Future studies of motor timing in children may also benefit from the inclusion of similar performance measures that yield continuous measures of ADHD symptoms in order to establish which behavioural components explain the motor timing difficulty. Such methodologies should be increasingly important as disorders are conceptualised in dimensional rather than categorical terms.

8.4.4. A dissociation between the variance components that are associated with features of dyslexia and ADHD?

In light of the high risk of Type I errors in the present study, the associations between literacy and timekeeper variance remain to be replicated, however, were the increased timing variability to be attributed to the timekeeper mechanism, the absence of deficits in overall accuracy of timing may suggest that children with reading difficulties have a difficulty that closely resembles that of cerebellar patients. Harrington et al. (2004) studied patients with focal cerebellar lesions and found the patients had increased timekeeper variance compared to controls in the absence of any difference in implementation variance or accuracy (mean IRI)[2]. They proposed that the lack of impairments of timing accuracy in the patient group indicates that the timekeeper produces outputs at a satisfactory rate, but in the presence of higher timekeeper variance suggests a deficit in the processes that modulate timekeeper outputs. Although the Wing-Kristofferson clock model does invoke the metaphor of a clock or timekeeper, when it is considered within the context of a complex neural system it is important to remember that the timekeeper variance component can encompass changes in a range of control elements (for a discussion see Madison, 2001b). Harrington et al. proposed candidates for processes that may modulate timekeeper variance without affecting the output rate of the timekeeper (i.e. accuracy), including working memory or the coordination between auditory, cognitive and motor output processes, all of which rely on processing occurring in conjunction with the cerebellum (Harrington et al., 2004). Working memory is required to maintain a representation of the timekeeper output during the continuation phase of tapping; coordination of output processes is required to maintain synchrony between predicted and actual events, both of which could affect timekeeper variance (Harrington et al., 2004; Mauk, Medina, Nores, & Ohyama, 2000).

The results from patients with lesions to the cerebellum are in contrast to those found in patients with damage or dysfunction affecting the striate cortex (such as patients with Huntington's disease, Parkinson's disease or bipolar disorder; Bolbecker et al., 2011; Freeman et al., 1996; Harrington et al., 1998). These patient groups show increased implementation variance and/or clock variance as well as alterations in the accuracy of their timing (mean IRI). These groups also have movement related difficulties resulting in changes in force or movement control that may introduce extra variance to their tapping performance (Ivry & Keele, 1989). These profiles are similar to timing performance in children with ADHD who show increased timekeeper and implementation variance (Valera et al., 2010; Zelaznik et al., 2012) together with alterations in the rate of motor timing (mean IRI), with children with ADHD demonstrating hastening phenomena (Ben-Pazi et al., 2003, 2006).

[2] Associations between mean IRI and the predictors of interest were not examined directly here to reduce the number of statistical comparisons performed, however, the groups did not differ on mean IRI and previous studies do not report any group differences and mean IRI is not found to be a prominent predictor of literacy ability.

Here, increased implementation variance was associated with a higher number of ADHD symptoms, but drift (that would be indicative of hastening) was not predictive of either the attention or literacy variables.

A number of authors have suggested that a distinction between timing impairments associated with the striate or cerebellar regions should be drawn because these areas are part of distinct neural networks primarily engaged in different types of timing control (Houk et al., 2007; Kotz, Schwartze, & Schmidt-Kassow, 2009; Penhune et al., 1998). The system involving the pre-SMA and basal ganglia can be thought of as a beat-based system that gives rise to predictable timekeeper pulses needed for rhythmic timing, playing an important role in pattern formation (Grahn & Brett, 2007; Houk et al., 2007; Penhune et al., 1998). In comparison, a separate, so-called automatic timing network that is important for processing millisecond intervals (comprising the primary motor areas, dentate nucleus of the cerebellum, global pallidus (GPi), thalamus and the parts of the SMA that serve primary motor areas) uses feedback from external events to guide behaviour. The cerebellum in particular is important for promoting refinement and amplification of signals, especially in these sensorimotor paradigms (Houk et al., 2007; Penhune et al., 1998).

Typically, patients with Parkinson's or Huntington's disease do not have suitably predictable outputs from the beat-based striate system, leading to greater reliance on the automatic timing system. The correlations found here suggest that children with difficulties with rapid naming have a more variable timekeeper system, but no co-occurring differences in the implementation system. If the timekeeper variance is associated with the automatic timing network, as proposed by Kotz et al., then children with reading difficulties may have impairments in the automatic timing system that lead to over-reliance on the beat-based system. In contrast, patients with damage to the striate cortex can make use of the cerebellar network to compensate for their lack of beat-based timing (Kotz et al., 2009).

In the previous experiment that examined the effects of task modality (Chapter 6), children with poorer literacy skills appeared to rely on a beat-based timing strategy in the visual condition rather than a motor focused strategy that would be based in the implementation system supported by the cerebellar-thalamic-SMA network. The latter strategy is more commonly selected by typically developing individuals and is advantageous for visual timing tasks due to the imperfect pacemaker signals generated under visually guided conditions. For the children with reading difficulties, continued reliance on the beat output system provided by the basal ganglia may occur due to the putative impairment of automatic support systems. Thus, children with poor literacy skills may have a deficit resulting in elements captured by the timekeeper component but are unlikely to be specifically impaired in the beat-based representation. The difficulties are likely to result from peripheral processes such as error correction, monitoring of synchrony between auditory, cognitive and motor outputs, or online memory for pulse rate that contribute to the timekeeper variance.

In the unpaced motor timing paradigm used in this study, the requirement to establish memory traces of the tapping rate may act to increase variability more than in paced tasks. Failures of memory would also be expected to lead to progressive changes in performance with stimulus rate. However, no conclusive evidence was found that the time series of interest were affected by such drift. Furthermore, although the digit span measure of short term memory was strongly and significantly associated with timing variability overall ($r=0.42$, $p<0.01$), it was not associated with timekeeper variance ($r=0.09$, n.s.). Instead it was associated with the implementation component ($r=0.32$, $p=0.05$) and may therefore be related to the difficulties experienced by the children with a higher number of ADHD symptoms who showed higher levels of implementation variance. The evidence that children and adults with dyslexia also show increased variability on unpaced, as well as paced tasks (Thomson & Goswami, 2008; Wolff et al., 1984; Wolff, 2002) where there is less load on working memory, suggests that working memory is unlikely to be a substantial contributor to the timing deficits in children with reading difficulties. A more likely candidate is the mechanism(s) that contribute to maintenance of synchrony within the automatic timing network, such as error correction.

Although the time series model applied here was able to distinguish two components of timing behaviour (i.e. timekeeper and implementation variance), further analysis of these additional processing systems is not possible with the simple model. In the Wing-Kristofferson model *any* non-random variability is attributed to the timekeeper mechanism without distinguishing between underlying components. A number of further mathematical additions to the original Wing-Kristofferson model have been proposed which can examine other components, such as variance in sensory processes (Elliott et al., 2010) and error correction processes with respect to detection of stimulus-response asynchronies (Pressing, 1998; Semjen et al., 2000; Vorberg & Wing, 1996). An alternative approach to understanding the process of error correction within timekeeping behaviour, has been to directly measure processes of error correction (Repp, 2001a, 2001b, 2002, 2005). These methods may be useful to apply in future investigations of motor timing in children, and especially in investigations of motor timing in relation to developmental disorders, to tease apart the control mechanisms involved.

8.4.5. Evidence for a synchronisation deficit

One element of the research which has not often been considered in the field of developmental disorders is the potential differences between synchronisation and continuation paradigms. The results described above indicate that timing performance on unpaced continuation tasks is associated with rapid naming ability. These associations were smaller in magnitude than those found between timekeeper variance and spelling ability in Chapter 6, where only the synchronisation (paced) phase of the finger task was examined. It would also appear from other studies (see Table

3.1, Chapter 3) that children with literacy difficulties have more consistent difficulties with timing on these paced synchronisation tasks.

Analyses have sometimes collapsed across the synchronisation and continuation phases or only analysed one of the segments in motor timing studies examining developmental disorders (Badian & Wolff, 1977; Klipcera et al., 1981; Wolff, Michel, Ovrut, et al., 1990) making comparisons across the tasks difficult. Thomson et al. (2006) analysed the different phases separately and found that paced timing variability, but not unpaced variability, was correlated with spelling and phonological ability in adults after controlling for reasoning ability. Similarly, when Thomson and Goswami (2008) assessed a group of 10 year-olds, paced timing variability explained around 25% of the variance in reading and spelling performance after controlling for age and IQ. Unpaced performance variability only explained 9% of the variance in spelling ability. The unpaced task was selected for use in the present experiment because it gives direct access to timekeeping mechanisms unhindered by the need to monitor ongoing stimuli, which can increase performance variability (Madison, 2001b; Semjen & Ivry, 2001). With more behavioural variability on the paced tasks, it is possible that it may be easier to find associations with cognitive predictors using these tasks and this may account for the differences between synchronisation and continuation data.

An alternative is that the differences appear because literacy variables are more strongly associated with a property of synchronisation tasks that has consequences for literacy development. In paced synchronisation tasks, participants need to become entrained to the repeated stimulus interval, and produce outputs which are perceived as being in synchrony with the pacing stimulus[3]. This process relies on prediction of future stimuli to generate future responses. In light of any variability that may be present in either timekeeper or implementation systems, there is also a need to monitor the success of synchrony so that responses can be updated appropriately when errors are detected. Any variability on a synchronisation task may therefore result from a failure to entrain, or a failure to predict or monitor synchrony appropriately. As described earlier, the profile of increased variability, in the absence of any difference in motor timing accuracy in children with reading difficulties implicate the latter of these options, although both hypotheses are attractive given that entrainment and synchrony are closely linked processes that are both important in speech development (Lakatos et al., 2008).

Some of the earlier studies reported greater asynchrony between stimuli and responses in children with dyslexia (Klipcera et al., 1981; Thomson & Goswami, 2008; Wolff, 2002) suggesting a greater discrepancy between representations of these elements within the timing system (Aschersleben & Prinz, 1995; Stenneken, Prinz, Cole, Paillard, & Aschersleben, 2006). Furthermore, the findings that

[3] Typically, responses actually occur before the onset of the stimulus in order to achieve this perception of synchrony (Aschersleben, 2002); see Chapter 2.

children with dyslexia also experience difficulties on bimanual tasks (described in Chapter 3) suggest a failure to use feedback from sensory sources to appropriately guide synchrony between stimuli and responses. In bimanual tasks the additional sensory feedback from the two hands should assist in synchronisation performance (Helmuth & Ivry, 1996), but did not facilitate timing performance in the children with dyslexia (Badian & Wolff, 1977; Klipcera et al., 1981; Wolff et al., 1984; Wolff, Michel, Ovrut, et al., 1990; see Table 3.1, Chapter 3).

In comparison to synchronisation performance, unpaced continuation performance relies on entrainment being established appropriately during the preceding synchronisation phase to allow the timekeeping system to continue to generate pulses that approximate the target interval rate in order to guide responses even after the stimuli have ceased. In the absence of pacing stimuli, participants must maintain their response outputs based solely on this timekeeping mechanism. The continuation phase should therefore be a more direct measure of the timekeeping system. The inconsistent results with unpaced tasks may be because children with reading difficulties only have problems with the synchronisation phase rather than timekeeping itself.

There is evidence that during unpaced tapping adults rely exclusively on the timekeeper to maintain responses whereas children may not. De Guio and colleagues compared brain activity using fMRI in children and adults during finger tapping tasks and found that children continued to show activity in the auditory cortex even after the auditory pacing stimuli had stopped (De Guio et al., 2012). This continued activity has been attributed to the children generating their own internal reference tone (likened to an echoic rehearsal of the tone) which helps guide responses in the absence of physical stimuli (De Guio et al., 2012). Indeed, during data collection for the present study, several children had to be reminded to remain silent during the task as they attempted to recreate the tones with a spoken “beep” once they had ceased. This idea may explain earlier findings that adults with dyslexia do not show a difficulty with unpaced tapping (Thomson et al., 2006). Adult behaviour is likely guided by timekeeper mechanisms so they rely less heavily on auditory rehearsal. If the echoic rehearsal of stimuli act as a form of pacing stimuli then unpaced tasks may actually measure synchronisation behaviour after all. Another study found that adults also showed activity in the superior temporal gyrus and inferior frontal gyrus during continuation tapping which was attributed to this sub-vocal rehearsal (Rao et al., 1997). If it were being used by adults it may be that they rely on it less to support timing behaviour than children. In light of this, associations found between unpaced or paced timing performance and literacy variables may result from the same deficit in synchronisation to stimuli (whether internally/externally referenced).

The two studies within this thesis that have examined timing performance using the Wing-Kristofferson method have therefore been useful in establishing the different components of timing that are separately associated with literacy and attention variables. However, the Wing-Kristofferson

model used in these studies is not able to break down the variance any further into the feedback components that are used to support synchronisation timing. Despite this the results point towards an impairment located in the automatic timing network described by Kotz et al. (2009), important for monitoring synchrony, prediction and error correction. Furthermore, the profile of increased asynchrony and impairments in synchronisation that are linked to literacy skills suggest a particular failure in the use of sensory feedback to guide motor timing responses. The ability to use sensory feedback can either be measured directly by assessing whether errors in timing are noticed (Repp, 2000, 2001b, 2005), or be modelled indirectly through further adaptation of the Wing-Kristofferson model to estimate feedback components (Elliott et al., 2010; Helmuth & Ivry, 1996; Pressing & Jolley-Rogers, 1997; Semjen et al., 2000; Vorberg & Wing, 1996). In the next study the first method was selected as more appropriate, given the time available, to assess the use of sensory feedback through the introduction of errors into the time series.

8.4.6. Evaluations of the Wing-Kristofferson model

Following the analyses of the Wing-Kristofferson model performed in Chapter 7, the decomposed timekeeper and implementation variance components used presently were based on estimates drawn from the first trial completed by each participant which satisfied the assumptions of the model. An alternative option would have been to remove violating trials from the analyses, but this would have resulted in unequal quantities of data for each participant. By analysing the presence and causes of violations it was possible to confirm whether the groups differed in the extent to which their tapping behaviour satisfied the assumptions of the time series model. The data from the Clinic group did not consistently contain any more violations of the model assumptions than either of the control groups, such that the factors contributing to violations (e.g. non-stationarity or closed-loop timing) were equally present across the three groups. Such factors can affect the estimates produced by the Wing-Kristofferson model, depending on the particular model parameters used. Because the violations were evident to a similar extent across the groups of children here, it is likely that the same results would have been found regardless of the method of calculating estimates of variance components.

All of the groups showed some evidence of closed-loop timing control that should not be a feature of time series governed by lag one autocorrelations. This means that the children were occasionally using information from previous intervals (at lags greater than one: i.e. 2-5) to update their responses. All three groups also showed some non-stationarity in their time series (linear and non-linear), but particularly the RL matched group at the slowest tapping rate. Drift may be an indicator of the extent to which children have developed the ability to entrain to different tapping rates, with the youngest children having most difficulty with the slowest rates. However, the cases of violations of the model were not present consistently across the groups or across interval rates.

This type of analysis of the model assumptions has recently been used to suggest that children with ADHD use more of this closed-loop feedback than control children when completing a motor timing task with 500ms intervals (2Hz) (Zelaznik et al., 2012). Here, however, where violations were assessed across a range of intervals, it would appear that most children occasionally use closed-loop timing control or drift from the target IOIs.

The results of the regression analysis showed that linear drift and timing variability (IRI SD) contributed unique variance to rapid naming performance but that drift was no better predictor than overall timing variability. Drift likely represents a component that is different from the standard measure of variability. Children with ADHD typically show a greater tendency to drift from the stimulus rate than controls (Ben-Pazi et al., 2003, 2006; Zelaznik et al., 2012) and therefore this feature of performance may relate to attentional deficits in this group, but in this group which was not selected for attention difficulties, the measures of drift were not significantly correlated with the ADHD ratings. Including a detailed analysis of time series to confirm whether they conform to linear models is also certainly useful to characterise behaviour (Madison, 2001b; Vorberg & Wing, 1996). The children assessed here were able to generate some time series which satisfy this type of model and so the results should not preclude the use of extended time series models in future which account for sensory or error correction components (e.g. those described by Elliott et al., 2010). Although only time series that satisfied the model were analysed presently, it is worth noting that a greater number of trials from the Clinic group was rejected prior to analysis, where more than 10 IRIs fell outside the range of 50% of the target IOI (e.g. outside the range of 335-1009ms for a target IOI of 673ms). Failures to match the trial IOI may have resulted from excess variability of the type measured in the study above, or other differences such as self-generated errors or complete failures to register the target response rate. The present study did not aim to examine such trials but rather the format of timing behaviour under circumstances where the assumptions of the Wing-Kristofferson model were met. However, these data suggest that the children with reading difficulties find this task more challenging than other children and future analysis of the causes of such performance failures could add to understanding of the full spectrum of their timing difficulties.

9. Experimental chapter 3: Error correction in motor timing tasks

9.1. Introduction

Previous chapters have shown that children with reading difficulties tend to have increased variability on motor timing tasks (paced and unpaced) that appears to be attributable to mechanisms associated with maintaining synchrony between stimuli and associated responses (Chapters 3, 6 and 8; Badian & Wolff, 1977; Klipcera, Wolff, & Drake, 1981; Thomson & Goswami, 2008; Wolff, Cohen, & Drake, 1984; Wolff, 2002). These processes contribute to the timekeeper variance component measured using the Wing-Kristofferson model of time series analysis that was found to be higher in children with poor literacy skills (Chapter 8; Harrington, Haaland, & Hermanowicz, 1998). In normal motor timing behaviour, spontaneous variability in the millisecond range is present. To maintain an inter-response interval (IRI) rate without drift, this variability has to be monitored such that responses remain timed to the pacing stimuli. The ability to account for this variability can be measured by assessing error detection and correction for errors in millisecond timing (Drewing & Aschersleben, 2003; Madison & Delignières, 2009). Creating disturbances (or perturbations) of known magnitude in the pacing stimuli during a synchronisation task allows measurement of the ability to correct errors rapidly and return to the pre-perturbation response rate. Artificially created disturbances of this kind in stimuli on synchronised motor timing tasks produce a similar neural response to self-generated errors on these tasks (Bijsterbosch, Lee, Hunter, et al., 2011). This task has been widely applied by Repp and colleagues to measure error correction behaviour in adults (Repp & Steinman, 2010b; Repp, 2001b, 2002, 2005). When created experimentally, small perturbations of this type are known as phase shifts (see Figure 1.); the inter-onset interval (IOI) of a single stimulus is altered, without affecting the interval rate (or period).

The contribution of phase correction to motor timing performance has not previously been investigated in children. Wolff (2002) tested the effect of a change in the rate of pacing stimuli and found that when the rate changed (from 1.5 to 2 to 2.5 Hz and back again) children with dyslexia took longer to correct to the new rate. This type of period change creates a step change in the speed of stimuli (see Figure 1.). Typically, children with reading difficulties are able to maintain a steady finger tapping rate (mean IRI) over time, but show increased interval-to-interval variability in the order of milliseconds around their mean IRI, of the kind that is influenced by phase correction mechanisms (Repp, 2001a). It is therefore unlikely that the period change paradigm used by Wolff was representative of the small magnitude interval-to-interval changes in IRIs that contribute to higher within participant variability in participants with reading difficulties.

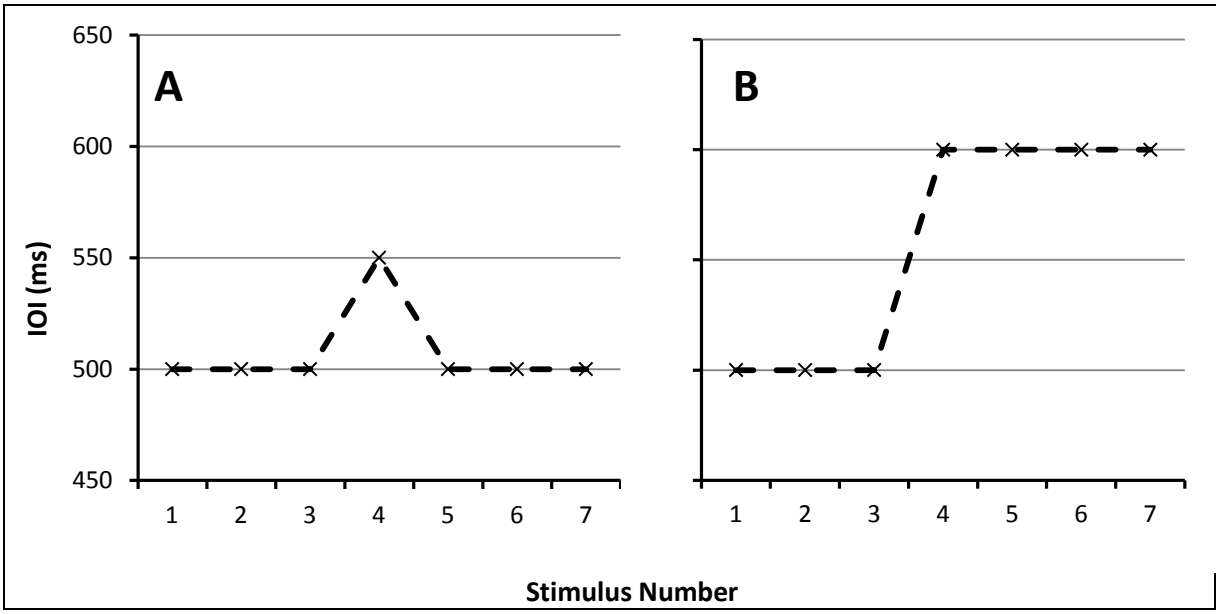

Figure 9.1: Phase and period shift paradigms
Representation of inter-stimulus intervals with a 50ms phase change (A) and a 100ms period change (B).

During paced motor timing tasks, participants tend to tap before the stimulus presentation (negative asynchrony) in order to achieve a perception of synchrony (see Chapter 3 and Aschersleben & Prinz, 1995; Repp, 2005). When phase changes in stimuli occur, they affect response asynchronies, such that positive phase changes (an increase in the length of a single IOI, as shown in Figure 1.) the cause the response asynchrony to increase momentarily. Correspondingly, when a negative asynchrony is introduced (a decrease in an IOI), response asynchrony decreases. Both types of change affect the synchrony between stimuli and responses and tend to be compensated in a way that does not alter the rate of tapping (or the period of the internal timekeeper) but instead rapidly corrects the local asynchrony (Repp, 2000, 2001a).

This compensation reduces asynchrony back to the pre-shift baseline and may be based on comparing the expected sensory consequences of a movement (forward models) to the actual sensory consequences of responses (Miall & Reckess, 2002; Stenneken et al., 2006). When the expectation of the model and the perception of stimuli or responses do not match, then correction can be implemented. Positive phase shifts, which give rise to increases in asynchrony, are more efficiently corrected than negative shifts. The latter are not noticed as readily because of the negative asynchrony that already exists in tapping behaviour, meaning that larger negative shifts are required before a response is perceived as being late with respect to the stimulus (Bijsterbosch, Lee, Hunter, et al., 2011; Repp, 2000). The degree to which correction occurs (the phase correction response; PCR) is generally measured by examining the compensation function, averaged over a number of trials (Praamstra, Turgeon, Hesse, Wing, & Perryer, 2003; Repp, 2000). This function demonstrates the extent and rate at which the asynchrony is corrected to baseline levels over the finger tap responses that follow the phase shift.

Within the phase correction paradigm, a distinction has been drawn between small phase shifts (<4% of the IOI, e.g. for 500ms IOI a shift of <20ms) and larger phase shifts (>5%). Small phase shifts are typically below the perceptual threshold for detection yet are corrected unconsciously and as rapidly as shifts that are consciously detected (Repp, 2000, 2001b). These small shifts (in the range of 5-50ms) are often smaller than the average within-participant variability exhibited in finger tapping behaviour, but are distinguishable from this noise when analysed over repeated trials. When larger shifts (e.g. of 50ms) are implemented, participants are able to report their presence and the phase correction response is not as rapid as for small shifts, leading to the conclusion that a different mechanism of compensation governs sub- and supraliminal shifts (Repp, 2005).

Small phase shifts of the kind which contribute to the ongoing within participant variability in motor timing tasks tend to be processed within an automatic timing network that is associated with processing temporal information in the millisecond range (Mauk & Buonomano, 2004). Small changes in the synchrony of stimuli and responses are able to be corrected rapidly despite the changes not being consciously detected, due to the coupling between sensory and motor regions in the network (Repp & Su, 2013). This network incorporates pre-motor and sensorimotor areas and the cerebellum (particularly dentate nucleus) (Bijsterbosch, Lee, Hunter, et al., 2011; del Olmo, Cheeran, Koch, & Rothwell, 2007; Repp, Keller, & Jacoby, 2012). Activity across this network, enables feed-forward predictions to be made to optimise performance when changes in stimuli are encountered (Stein, 2009; Stoodley & Schmahmann, 2009; Tesche & Karhu, 2000; Wolpert, Miall, & Kawato, 1998). As discussed in Chapter 8, the motor timing difficulties experienced by children with reading difficulties are characteristic of a difference in the same automatic system of timing control that operates through this SMA-thalamic-cerebellar network and which contributes to the monitoring of synchrony and error correction processes that maintain synchrony.

In paced motor timing tasks, timekeeper mechanisms work to maintain synchrony. Feedback from sensory events can also influence the timed response (e.g. feedback from movements and external stimuli). When pacing stimuli contain uncertainties (for example in visually paced tasks, as described in Chapter 6, or where there are instabilities in the stimuli), then synchronisation is primarily governed by a strategy which places less emphasis on the sensory events (Jäncke, Loose, et al., 2000; Kolers & Brewster, 1985; Villing et al., 2011). Following the results presented in Chapter 6 (under visually paced conditions) and those presented in Chapter 8 (using unpaced finger tapping) it was proposed that children with poorer reading or attentional abilities tended to rely on sensory events, regardless of their validity due to instabilities within the stimuli. In addition children with reading difficulties tended to fail to synchronise their responses to external stimuli appropriately. Children with poor reading abilities are therefore expected to be insensitive to shifts in the pacing stimuli.

9.1.1. Overview of experiments

First, to confirm that the computer based error correction task was successful in generating a phase correction response, a pilot study was conducted with a group of adults (Experiment 1). Following this, a pilot study was conducted with a group of children (Experiment 2) to establish whether they respond to phase shifts in the same way as adults and to select the size of the phase shift to be used in the final experiment. In the final study, a group of children were assessed on the error correction task and their phase correction behaviour was compared to performance on literacy, attention and other cognitive tasks (Experiment 3).

The IOI rate selected for the phase correction paradigm was 500ms (2Hz). This rate is frequently used in phase correction paradigms (Bijsterbosch, Lee, Dyson-Sutton, Barker, & Woodruff, 2011; Repp, 2000), produces a low coefficient of variation and represents the preferred tapping rate of children at which performance is likely to be most stable (Y. Chen et al., 2002; McAuley et al., 2006). This rate was also within the range of tapping rates where difficulties in timing stability are frequently reported in children with reading difficulties (Chapter 8). Only positive phase shifts that cause an increase in asynchrony were implemented.

9.2. Experiment 1: Pilot study with adults

The aim of the pilot study was to trial the phase correction methodology in order to generate phase correction response functions, replicating those demonstrated in previous studies of phase correction in adults (Praamstra et al., 2003; Repp, 2000).

9.2.1. Methods

9.2.1.1. Participants

Five adults (one male) drawn from the university student and staff population aged 23-39 completed the task. Adults provided consent for taking part following the ethical principles described in Chapter 4.

9.2.1.2. Phase correction motor timing task

The phase-shift correction task (Figure 9.2) included a synchronisation phase, followed by a subliminal phase shift created by lengthening one interval, and a resynchronisation phase of 20 tones to allow any phase correction behaviour to be recorded. Fifty percent of trials also contained a supraliminal distracter shift occurring after the fifteenth resynchronisation tone, where one interval was lengthened by 90ms. The initial subliminal phase shift was created by adding 6, 9, or 12ms to a single IOI. The pacing stimuli had 500ms inter-onset-intervals (IOIs) and were auditory pure sinusoidal tones (440Hz), 50ms in length with rapid smooth onsets and offsets. The number of stimuli in the initial synchronisation sequence prior to the phase shift was varied on each trial (with 15, 16,

17, 18, 19 or 20 stimuli) so that the position of the initial phase shift was jittered, thereby mitigating against any form of entrainment to the location of the shift. Varying the number of initial synchronisation stimuli had the additional effect of jittering the position of the distracter shift. Participants completed 12 trials at each shift magnitude (two at each jitter position: one with the distracter shift, one without). Two practice trials were followed by 36 experimental trials, presented in a random order in 9 blocks of 4 trials. Stimulus and response onset times were recorded.

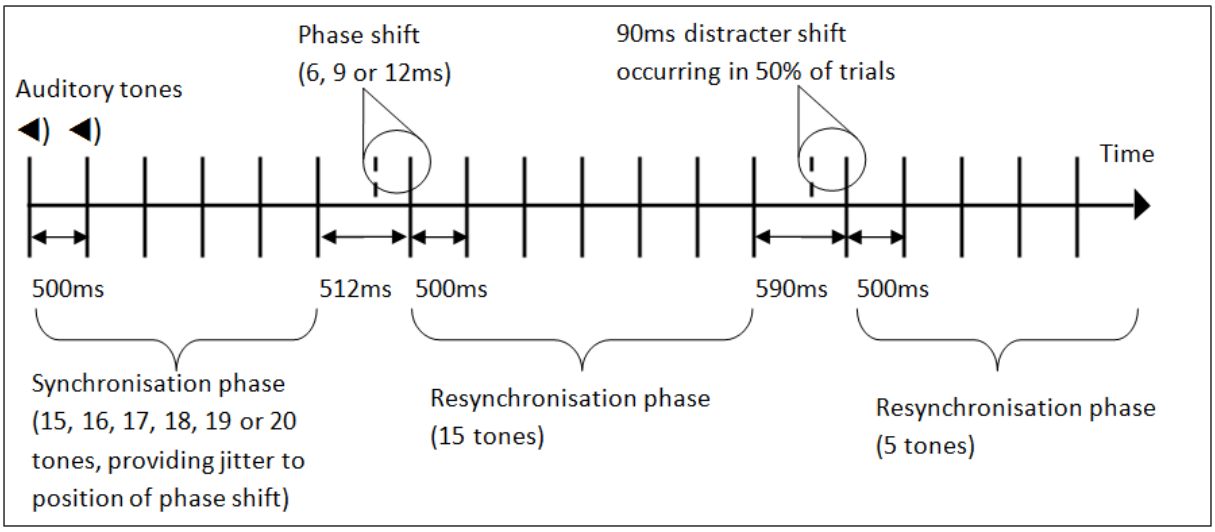

Figure 9.2: Phase correction task

Representation of the stimuli presented in a single trial of the phase correction task. Vertical bars represent time of occurrence of auditory tones at 500ms ISIs. The synchronisation phase was 15, 16, 17, 18, 19 or 20 tones in length, providing jitter to position of phase shift. The phase shift was then presented as a single lengthened interval (506, 509 or 512ms ISI). This was followed by 15 resynchronisation stimuli also with 500ms ISIs. A distracter shift was then presented, with one stimulus interval lengthened by 90ms. The trial was completed with 5 further resynchronisation stimuli with 500ms ISIs. The participant's task was to tap in time with the stimuli and report whether they had heard the distracter shift.

The phase correction paradigm was presented to participants as a game in which they were acting as the operator of a bottle-labelling machine (Figure 9.3). They were asked to tap their finger on the response pad in time with the auditory "beeps" so that the machine labelled the bottles correctly. The end of each trial was signalled by a different tone. The diagram in Figure 9.3 was a static image that appeared on the screen and did not have any moving visual elements. Participants were also told that sometimes the machine might have a problem such that the beeps might sound like they were out of time (i.e. the supraliminal distracter shift). If this happened they were instructed to keep tapping in time and to "inform the engineers" by selecting the appropriate yes/no response at the end of the trial. Participants received a feedback score after each trial, calculated as mean IRI (in milliseconds) for responses made between the third stimulus in the trial and the fifteenth resynchronisation stimulus (where the distracter stimulus occurred). This was presented alongside the target score of 500 and a traffic light indicator (green for scores within ±25ms of the target, orange for up to ±50ms and red for scores more than ±50ms from the target mean IRI). Participants were encouraged by the experimenter to improve the accuracy of their responses by tapping in time

with the beat in order to gain "green" scores (so the labels were placed in the correct location on the bottle). Participants placed their wrist on a foam rest and made finger tapping responses with their first-finger on a Wacom Bamboo Touchpad (CTH 460) that is sensitive to finger touches of minimal force and gives rise to minimal auditory feedback.

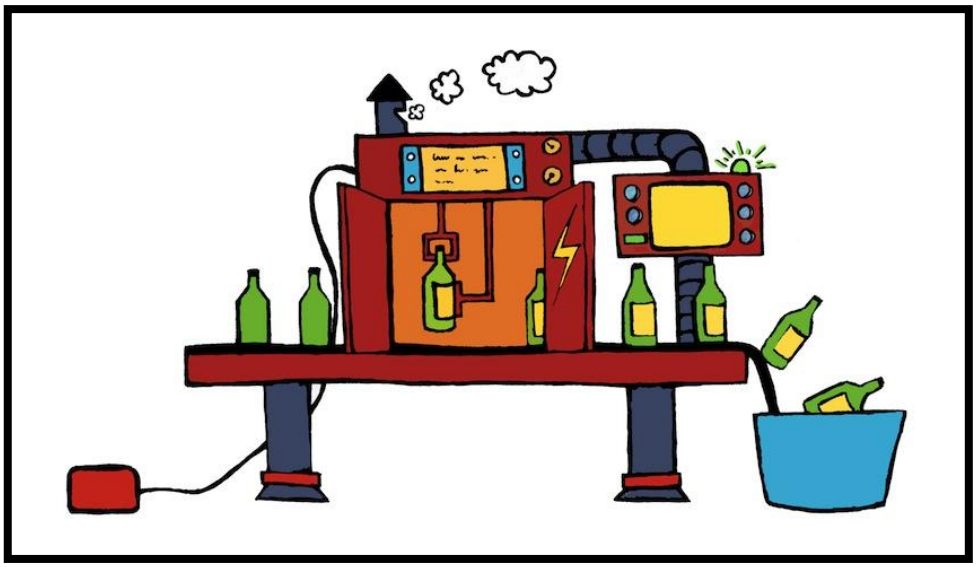

Figure 9.3: Phase correction game
Screenshot of the display used during the finger tapping task.

9.2.1.3. Data analysis

Responses were aligned with the nearest stimulus onset time to identify any missed responses or double bounces. Where such errors occurred in the three responses prior to the phase shift or the 10 responses following the shift, the entire trial was removed from the analysis. Such errors may interrupt any response to the shift. Inter-response intervals (IRIs) were then calculated for each pair of responses. In a similar exclusion process, where IRIs falling outside the range of 50% of the target interval (greater than 750ms or less than 250ms) occurred in the 3 responses before or 10 responses after the shift, the entire trial was removed from the analysis. Response asynchronies were then calculated for each response for the remaining trials (response time minus stimulus onset time).

The position of the target phase shift was considered point T (target). To deal with the jitter in the shift position across trials, asynchronies were aligned relative to the target. To account for individual differences in average response asynchrony, asynchronies were normalised within each trial by calculating a within trial average of the three asynchronies occurring prior to the target (T-1 to T-3) and subtracting this from each asynchrony in the trial. This resulted in normalised asynchronies that were artificially close to zero immediately preceding the perturbation. For each position in the sequence (T-3 to T+8), an average of these normalised asynchronies across all trials and all participants was calculated and plotted as a compensation function. Mean IRI and IRI SD were calculated for the 10 responses occurring before the shift to measure the accuracy and variability of

synchronised performance prior to the phase-shift. For this first pilot study accepted alpha values were set to 0.05 due to the relatively small number of trials recorded.

Across trials it was observed that asynchronies were larger than expected (magnitudes of 100ms asynchronies). Re-examination of the data collection programme revealed that a delay was added by the computer software at the start of each trial, which acted to increase the asynchrony. This delay was different for each trial, but is adequately dealt with through the normalisation process such that the compensation functions are still valid.

9.2.2. Results and discussion

At the end of each trial participants were asked whether they had noticed a problem with the machine (the distracter shift). On average the participants correctly identified the presence or absence of the distracter 80.1% of the time (SD 10.3). This value was likely not higher because the adults often reported that they had forgotten whether they had heard the shift at the end of the trial. However, this task did serve to focus the attention of participants at the end of the trials prior to receiving feedback on their synchronisation performance. Of the 180 trials completed by the participants, 23% were deleted where double bounces or missed taps occurred within 3 intervals of the target shift. Participants produced responses that were close to the target IOI of 500ms (mean IRI for the 10 responses preceding the perturbation = 499ms, SD 3ms).

The compensation functions (Figure 9.4) show the average responses across trials and participants at each shift magnitude. The larger asynchrony at point T results from the presence of the phase shift in the stimulus train and should therefore be equal to the size of the phase shift. Correction can be observed where the post-target responses return to the pre-shift baseline. The function in Figure 9.4 illustrates that variability was present across trials in the asynchronies produced before the shift. This likely resulted from participants who were novices to the tapping paradigm compared to the highly practised participants (including musicians) often assessed in previous investigations of error correction (Repp, 2000, 2002). To account for this variability, the post-target responses were analysed relative to a less conservative baseline value than zero (2 standard errors of the mean asynchrony before the shift below zero, shown as the dotted line in Figure 9.4). The variability in the baseline data used for normalisation of asynchronies also acts to increase the size of the shift at point T. This was evident for all three phase shift magnitudes, where asynchronies at point T were larger than expected magnitude of the shift (6, 9ms, and 12ms), and is likely due to the small samples of data assessed here.

For all three shift magnitudes, asynchronies at T were significantly different from the adjusted baseline in all cases ($p<0.05$). Examination of the functions suggests that some correction towards the baseline did occur following the phase shift, however, t-tests showed that the asynchronies after

point T were not significantly different from either the expected shift magnitude, or from the adjusted baseline value ($p>0.05$ for all comparisons at T+1 to T+8). The large error bars on the functions demonstrate the between-trial variability which precluded demonstration of a phase correction response in this small sample.

Although the small sample of pilot data only illustrated a trend towards the phase correction response, the results were sufficient to confirm that the paradigm elicits an appropriate compensation function. In previous studies, larger samples of data (n trials = 400-600) have been collected to generate functions which show significant error correction responses (Praamstra et al., 2003; Repp, 2000). Therefore, for the second pilot study, we aimed to collect more trials per participant and shift magnitude to improve the reliability of the phase correction function. Thus, data collection for the second experiment was focused at two shift magnitudes (9ms and 12ms). Previous studies find a robust phase correction response at these magnitudes of shift (Repp, 2000, 2001a) and the children were expected to show a similar response function. However, due to the lack of evidence about how children might respond to this paradigm, trials were also included at two additional magnitudes (6ms and 15ms) to ensure a compensation function was captured.

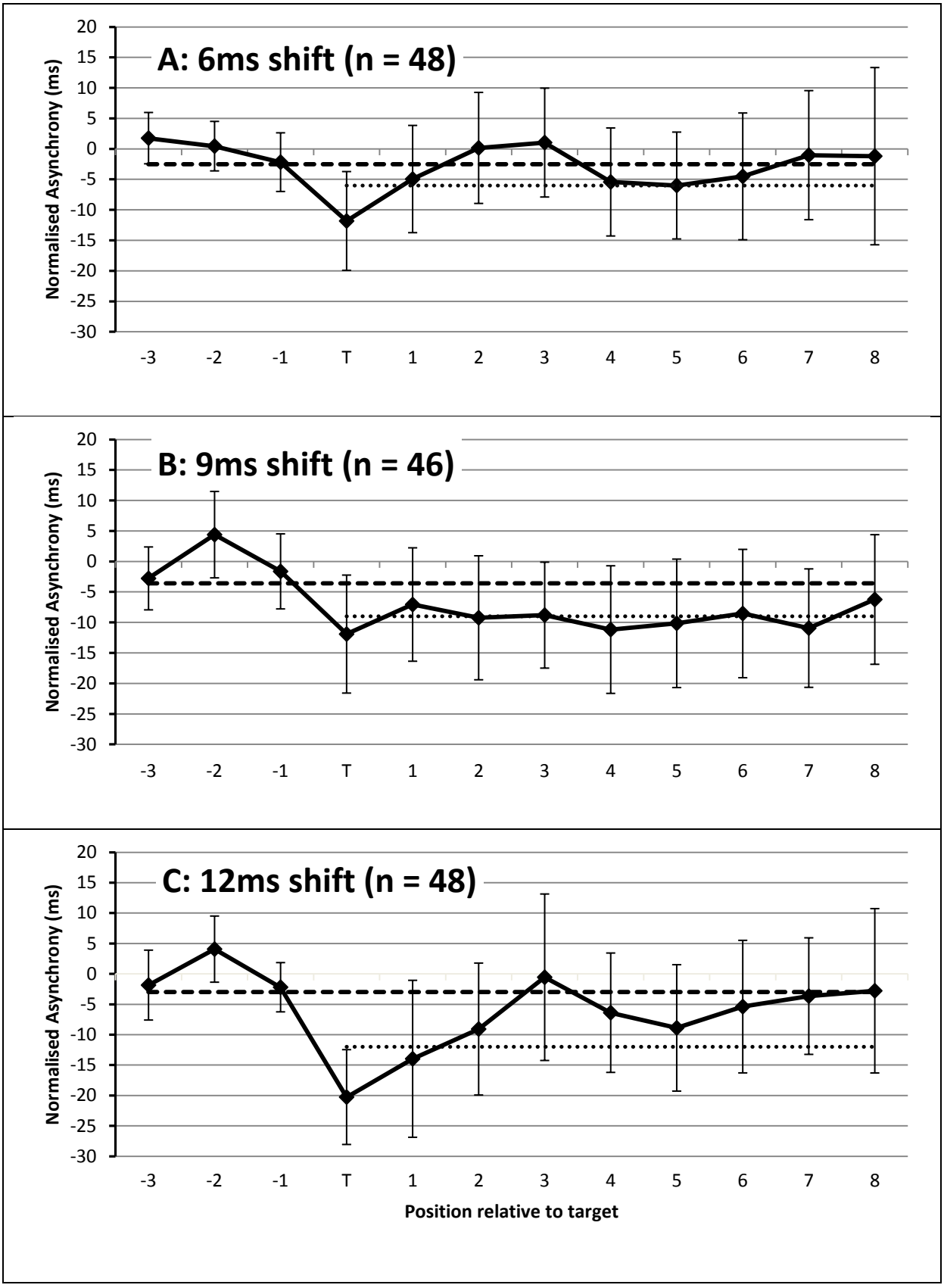

Figure 9.4: Average compensation functions

Functions show group mean asynchrony across the sequence relative to the position of the target phase shift (T). Individual asynchronies were normalised based on the three shifts before the target so that the pre-shift baseline lies at zero (error bars represent 2 SEM). A: 6ms shift (n trials=48), B: 9ms shift (n=46), C: 12ms shift (n=48). The upper dotted line (- - -) represents a conservative lower limit of the pre-shift baseline average (2 SE below the pre-shift baseline mean). The lower dotted line (....) represents the expected asynchrony following the target shift if no correction has occurred.

9.3. Experiment 2: Pilot study with children

The second pilot study tested the phase correction method with children to establish whether they show the same compensation response to phase shifts as shown by adults and to allow selection of an appropriate phase shift magnitude for implementation in Experiment 3. It was desirable that the magnitude of the phase shift would result in a consistent compensation function with some between participant variability, such that individual differences in error correction ability could be identified in Experiment 3. As described above, the data collection was focused at the 9ms and 12ms phase shifts where consistent phase responses are demonstrated in adults (Repp, 2000, 2001a). Additional trials with 6 and 15ms shifts were also included. In the first pilot study, a substantial amount of data (23%) was lost due to missed finger taps or double bounces immediately before or after the phase shift. To reduce this problem, trials containing errors near the phase shift were repeated by participants where possible.

9.3.1. Methods

9.3.1.1. Participants

The sample of 20 children (including 10 boys, and one left-handed participant, mean age 119 months) was drawn from a Year 5 class group from a state junior school. The school was situated in a large Derbyshire town and the total school population was around 350 pupils. Initial consent was gained from the head teacher and parental consent was gained via an opt-out letter of the kind already described in Chapter 4. Prior to commencing the experiment, all children were informed of the purpose of the experiment and about their right to withdraw following the ethical procedures described in Chapter 4.

9.3.1.2. Phase correction motor timing task

The phase correction task was similar to that described in Experiment 1 (See Section 0). Half of the sample completed trials with 6, 9, and 12ms phase shifts and half completed trials with 9, 12 and 15ms shifts, so that all participants completed trials with 9 and 12ms shifts. Each participant completed twelve trials at each of these phase shifts (50% with and 50% without the additional 90ms distracter stimulus, see Figure 9.2). The 36 trials completed by each participant were randomised and presented in 9 blocks of 4 trials. Breaks were encouraged between trials and blocks as required. Children completed the trials across two or three testing sessions on separate days. Any trials containing errors were repeated during later sessions. Responses were recorded with the same touchpad as in the first pilot experiment. For ease of movement and comfort, the children held the touchpad on their knees.

9.3.1.3. Data analysis

The data analysis procedure followed that described in Section 9.2.1.3. With a larger sample, the alpha value for all comparisons was set to a more conservative level of 0.01 to account for the large number of statistical comparisons completed. Of the 720 trials completed in total, 14% were eliminated where response errors occurred within three intervals of the target shift (17% of trials with the 6ms shift, 20% of trials with the 9ms shift, 11% of trials with the 12ms shift, and 12% of trials with the 15ms shift). Across the remaining 619 trials (collapsed across shift magnitude) the average IRI for the 10 taps preceding a shift was 500ms (SD 36ms), indicating that participants achieved the stimulus rate appropriately.

9.3.2. Results and discussion

The children correctly identified the presence or absence of the distracter shift in 80.7% of trials (SD 14.9) which was similar to the percentage for adults in Experiment 1. The children appeared to be engaged by and interested in this part of the task.

The compensation functions (Figure 9.5) show the average of normalised asynchronies across all trials and participants at each shift magnitude. The change in asynchrony at point T results from the introduction of the phase shift; if the post target responses return to baseline following the shift, correction has occurred. The variability present in the pre-target baseline data used for normalisation of asynchronies acts to increase the size of the asynchrony at point T (evident with the 6 and 15ms shifts where the least data was collected). As in Experiment 1, the variability in the pre-shift baseline responses meant that it was appropriate to use a less conservative baseline value in examination of the correction responses post-target (calculated as 2 SE below the baseline, shown as the upper dotted line in Figure 9.5). For trials with the 6ms phase shift, the variability in the baseline data meant that the average asynchrony at point T was larger than the expected -6ms, but was not significantly different from the conservative baseline ($t=-1.57$, $df = 99$, $p=0.12$). Following the shift, average normalised asynchronies were also not statistically different from either the baseline (all comparisons n.s. with smallest p value = 0.13 at T+3 and T+5), or the expected shift value of -6ms (all comparisons n.s. with smallest p value = 0.33 at T+5). This suggests that a correction response was not discernible from the variability in responses.

For the trials with the 9ms shifts, the mean asynchrony at point T was significantly different from the baseline ($t=-3.56$. $df = 211$, $p<0.01$). The difference between the average asynchronies and the baseline was also significant at T+1 ($p<0.01$) and approached significance for T+2 to T+4 ($p<0.02$) suggesting that correction did not occur immediately. At points T+5 to T+7 responses were not significantly different from baseline or the shift level of -9ms (all comparisons n.s.). Only at T+8 were average asynchronies significantly different from -9ms ($p<0.01$) but not from the baseline indicating that correction to the pre-shift baseline asynchrony had occurred by the eighth tap.

As shown in Figure 9.5C, the 12ms shift resulted in asynchronies that were significantly different from the baseline at the time of the shift (t=-3.25, df = 210, p<0.01) and at point T+1 (t =-3.26, df = 210, p<0.01) but were not significantly different from the target shift magnitude of -12ms (n.s. p>0.6). At T+3, T+4 and T+6 and T+8 the average normalised asynchronies were significantly different from the shift magnitude of -12ms (p<0.01), with responses at T+5 and T+7 also approaching significance (p<0.03). In addition, responses from T+2 onwards were not significantly different from the baseline (all comparisons n.s. with smallest p value = 0.1 at T+2) suggesting that correction had occurred from the second or third tap onwards.

For the 15ms shift (Figure 9.5D) the phase correction response appears similar to that in the 12ms shift condition. However, many of the comparisons were not statistically significant because fewer trials were completed at this shift magnitude. Normalised asynchronies at the time of the shift (T) and at T+1 were significantly different from the conservative baseline value of -2.58 (p<0.01), but not significantly different from the target shift magnitude of -15ms. Responses from T+2 onwards were not significantly different from baseline (all comparisons n.s. with smallest p value = 0.31 at T+2), but were also not significantly different from the shift magnitude of 12ms (although at T+2, T+5, T+6 and T+8 the difference approached significance, p<0.02).

The results indicate that where sufficient trials were collected with the 9 and 12ms shifts, phase correction responses were apparent (n>200). It took longer for the 9ms shift to be corrected compared to the 12ms perturbation which was corrected after two or three taps. For the 6 and 12ms shifts the correction response was indistinguishable from the variability in the data. Repp (2000) found that adults rapidly corrected small shifts (e.g. 4-8ms) in comparison to larger shifts of 10ms, whereas Praamstra et al. (2003) found that 15ms shifts were rapidly corrected by adults. The children assessed here, appear to correct the 12ms shift most effectively, with preliminary evidence that the 15ms shift is also rapidly corrected. Therefore, the 12ms shift, which showed a correction response with some variability across participants, was selected for use in the third experiment.

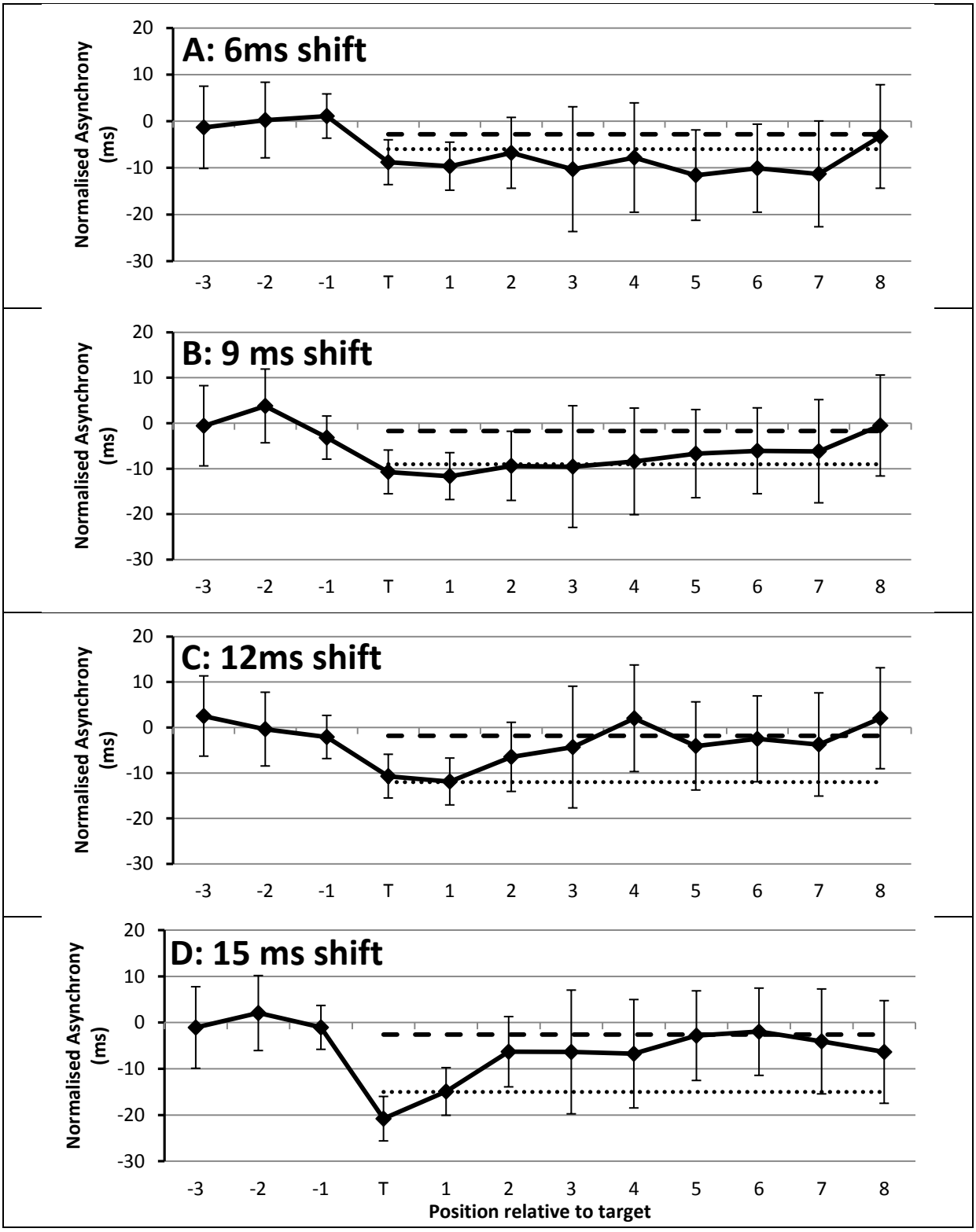

Figure 9.5: Average compensation functions

Functions show group mean asynchrony across the sequence relative to the position of the target phase shift (T). Individual asynchronies were normalised based on the three shifts before the target so that the pre-shift baseline lies at zero (error bars represent 2 SEM). A: 6ms shift (n trials=100), B: 9ms shift (n=212), C: 12ms shift (n=211), D: 15ms shift (n=96). The upper dotted line (- - -) represents a conservative lower limit of the pre-shift baseline average (2 SE below the pre-shift baseline mean). The lower dotted line (....) represents the expected asynchrony following the target shift if no correction has occurred.

It was clear that a large amount of data was required to enable individual differences in these responses to be distinguished from general motor timing variability. In light of the need to collect sufficient data whilst minimising the number of tapping responses the children had to provide, only one shift magnitude was selected for the final experiment. In addition, the number of taps required in any single trial was reduced by decreasing the number of responses in the initial synchronisation phase to eight. This number was also comparable the number used in studies of error correction in adults where the position of the shift in the sequence has been found to have no effect on the correction response (Repp, 2000). Examinations of the number of stimuli required before synchronisation occurs suggest that in adults there is no significant change in performance after 5 responses (Kolers & Brewster, 1985).

9.4. Experiment 3: Phase correction, reading and attention

9.4.1. Introduction

This experiment was conducted to examine the extent to which error correction ability in children varies with measures associated with reading and attention difficulties. These analyses were conducted with a sample of typically developing children because it was anticipated that trends would be present even in children without specific diagnoses given the correlations between reading/attention measures and timing performance found across samples of good and poor readers in previous studies (Chapters 6 and 8; Thomson & Goswami, 2008; Waber et al., 2000a). As well as completing the phase correction task with 12ms shifts, participants also completed synchronisation and continuation tapping tasks with isochronous stimuli of the kind used in the previous chapters in addition to the phase correction task. In the present study, only the overall variability in performance on these tasks (SD of IRIs) was analysed to give an indication of how phase correction behaviour relates to standard motor timing behaviour.

In Chapters 6 and 8, measures of spelling and rapid naming were moderately and significantly associated with motor timing. Spelling and rapid naming tasks encompass a rather broad range of component skills and so more specific measures of literacy component skills were utilised here to target reading efficiency and word fluency. Measures of processing speed were also used to assess whether motor timing is more related to a more general speed of processing factor than to fluency only on literacy tasks. In earlier chapters, a simple tone-response reaction time measure was used to control for the effect of motor speed in the analyses. Here, a more precise measure of motor reaction time and movement speed was included as a potential control for the influence of motor speed.

Behavioural measures of attention related abilities used in the previous studies (Score! and Opposite Worlds) were not consistently related to motor timing, nor were they correlated with parent or

teacher ratings of ADHD symptoms. Here, a computerised measure of behavioural inhibition, the Stop Signal Task, was included to assess component abilities frequently associated with the behavioural phenotype of ADHD. Deficits in response inhibition have been found in a majority of studies examining ADHD in children and adolescents and can be measured effectively using a continuous performance test such as the Stop Signal Reaction Time task (Nigg, 2001; Schachar, Tannock, Marriott, & Logan, 1995; Walshaw, Alloy, & Sabb, 2010). Response inhibition has also been suggested as an appropriate phenotypic marker for familial ADHD (Crosbie & Schachar, 2001). Furthermore, the timing impairments found in ADHD may be representative of a premature response style associated with impulsivity symptoms that is measured by inhibition tasks (Rubia et al., 2009).

9.4.2. Methods

9.4.2.1. Participants

Children for this study were drawn from another inner city Derbyshire primary school. The school had a total population of around 300. Thirty-two children from Year 5 participated in the study and were drawn from three mixed ability classes: one class of mixed year 4 and 5 children (n = 13), and two classes of mixed year 5 and 6 children (n = 19). The school operated a mixed class system to accommodate student numbers within their available classroom space. Seven children in the group had already taken part in a cognitive remediation programme within the last 9 months and were not included in the study. Following participation, a further 3 children were excluded from the analyses: one due to the disclosure of frequent ear infections and resulting partial deafness, one child who had received speech and language therapy in previous years and still showed evidence of speech related difficulties, and one child with a verbal reasoning score more than 2 standard deviations below the population mean. The remaining 29 children (17 females) had a mean age of 120 months (SD 3 months). All but four children had previously received some formal music tuition and all students participated in weekly class-based music sessions.

9.4.2.2. Cognitive / behavioural measures

Participants completed the Wechsler Abbreviated Scale of Intelligence subscales of verbal and non-verbal reasoning described in Chapter 4 (Similarities and Matrices subtests; Wechsler, 1999). Reading ability was assessed using the sight-word and phonemic decoding efficiency measures from the Test of Word Reading Efficiency (TOWRE; Torgeson, Wagner, & Rashotte, 1997). In each of these tasks children are given 45 seconds to read aloud as many words as they can, measuring the accuracy and fluency of reading words and novel non-words. The TOWRE has norms based on a US sample of 1500 children and young people and is commonly used in assessments of reading ability in clinical settings in the UK and Europe. Age-referenced standard scores for each child were calculated and the sum of the two subtest scores was converted into a Total Word Reading Efficiency Standard Score (TWRESS).

Three fluency tests (semantic, alliteration and rhyme) from the Phonological Assessment Battery were also administered to provide a measure of phonological ability (PhAB; Frederickson, Frith, & Reason, 1997). These tests require children to generate as many words as possible in 30 seconds that either come from the same category (semantic), start with the same sound (alliteration) or that rhyme with a test word (rhyme). The alliteration and rhyme subtests measure the ability to retrieve phonological information from memory. The PhAB has UK norms from a sample of 629 children aged 6-14; the age-referenced norms were used to generate standard scores.

As in the previous studies, ratings of ADHD symptoms were obtained using the ADHD Behaviour Rating Scale-Teacher Form (Barkley & Murphy, 1998), yielding separate scores for inattention (ADHD-IA) and hyperactivity-impulsivity (ADHD-HI) dimensions. The Stop Signal Task (SST), a measure of response inhibition, from the Cambridge Neuropsychological Testing Automated Battery (CANTAB eclipse^{TM}, Cambridge Cognition), was administered as a behavioural measure of inhibition. The stop signal reaction time measure is often used as a behavioural indicator of the presence of ADHD (Nigg, 2001; Schachar et al., 1995; Walshaw et al., 2010). This continuous performance task requires participants to press a left or right button in response to an arrow on the screen and withhold responses if they hear an auditory tone (the stop signal). Five blocks of 64 trials were presented, each divided into 4 sub-blocks for analysis purposes. Each sub-block comprised 12 Go trials and 4 Stop trials, presented in a random order with feedback provided after each block. The CANTAB algorithm derives the time between the arrow and the tone on Stop trials from four adaptive staircases starting at 100, 200, 400 and 500ms respectively. The Stop Signal Reaction Time (SSRT) generated is the latency of the stop signal where the probability of participants being able to inhibit their responses is 50%, i.e. the time required to inhibit a response.

The child version of the Simple Reaction Time (SRT) task from the CANTAB was also administered. In this task participants held down a button with the forefinger of their dominant hand until a spot appeared on the screen. On seeing the spot participants are required to release the button and touch the spot as fast as possible, generating a reaction time (time to release the button) and a movement time (time to move from button to screen).

The Symbol Search and Coding subtests, forming the Processing Speed Index (PSI), of the WISC-IV were administered as measures of speed of processing of visual information (Wechsler, 2003). The Symbol Search task required children to determine whether either of two target symbols appears in a group of 5 symbols. The coding task involved copying symbols that are paired with numbers in the key into response boxes, completing as many symbols as possible in the 2 minute time limit. For both tests, the number of items correctly completed in two minutes was recorded and scores were converted to standard scores using the appropriate age-referenced norms from the WISC-IV.

9.4.2.3. Motor timing tasks

9.4.2.3.a. Phase correction task

The phase correction paradigm (Figure 9.6) was similar to that described in Section 9.3.1.2 with a shift magnitude of 12ms. The jittered position of the phase shift followed either 8, 9, 10, or 11 synchronisation stimuli. This was followed by 15 resynchronisation stimuli and on 50% of trials a distracter shift of 90ms. Five further pacing stimuli followed the distracter shift. All participants completed six trials at each jitter position (three with distracters, three without) and trials were randomised. The 24 trials were completed over at least three testing sessions on different days, with breaks taken between trials. Where possible, any trials containing errors (missed taps or double bounces) were repeated during subsequent testing sessions. All other parameters of the task were identical to those described in the pilot study. As in Experiment 2, any trials with missing data were repeated by participants.

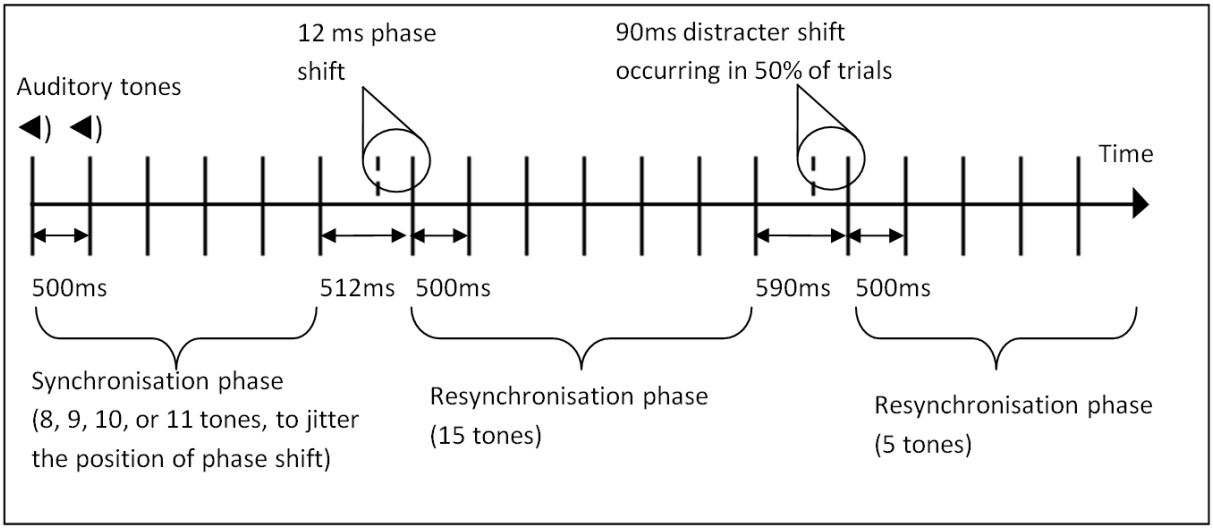

Figure 9.6: Adjusted phase correction task

Representation of the stimuli presented in a single trial of the phase correction task. Vertical bars represent time of occurrence of auditory tones at 500ms ISIs. The synchronisation phase was 8, 9, 10, or 11 tones in length, providing jitter to position of phase shift. The phase shift was then presented as a single lengthened interval (512ms ISI). This was followed by 15 resynchronisation stimuli also with 500ms ISIs. A distracter shift was then presented, with one stimulus interval lengthened by 90ms. The trial was completed with 5 further resynchronisation stimuli with 500ms ISIs. The participant's task was to tap in time with the stimuli and report whether they had heard the distracter shift.

9.4.2.3.b. Synchronise and continue motor timing task

A synchronise-and-continue motor timing task, similar to that used in Chapters 7 and 8 was used to measure paced and unpaced timing performance. After completing 8 phase correction trials, participants completed a block of three synchronise-and-continue trials. The latter task was presented as an extension of the game used for the phase correction task (see Section 0). Children were told that they had been successful enough to become a "master operator" and could operate the machine without the sounds being present. Children were presented with the auditory synchronisation tone (IOI 500ms) and instructed to tap in time with the beat. After 35 synchronisation stimuli, the tones stopped and participants were asked to continue tapping at the

same speed. Time was provided for 35 continuation responses to be recorded. Participants completed up to nine synchronise-and-continue trials with the aim of collecting at least three error free synchronise-and-continue trials for each participant.

9.4.2.4. Data analysis

9.4.2.4.a. Phase correction task

For the phase correction task, the analyses were the same as in Experiment 1 (Section 9.2.1.3). The accepted value of alpha was set to 0.01 to adjust for the high number of statistical comparisons carried out. From the data collected, only 26 trials had to be eliminated where response errors occurred within three intervals of the target shift. Across the remaining 670 trials, the average IRI for the 8 taps preceding a shift was 497 ms (SD 37 ms).

The compensation functions were constructed as described in Experiments 1 and 2, with asynchronies normalised on the average asynchrony of the three taps before the target (i.e. T-1, T-2, and T-3). When the phase correction response was plotted, the group average asynchrony at the time of the target (T) should have been at -12ms, however there was an overshoot to -20ms (±1.5ms) (Figure 9.7). This overshoot was a product of the variability in the three taps before the target on which the asynchronies were normalised. As described in Experiments 1 and 2, this variability makes it difficult to assess the functions relative to a common baseline or the expected change of 12ms. Therefore to provide a common baseline for the analysis, asynchronies were normalised relative to the asynchrony occurring at the time of the target. This yielded a zero baseline at the point of the target, and for correction to have occurred responses had to return to +12ms after point T (i.e. correcting for the 12ms shift). This type of normalisation has been conducted in previous studies (Repp, 2011, Experiment 3A) and has been found to result in similar phase correction estimates to the first methods, with no statistical difference in the function produced when correcting small shifts like those used here. The function normalised on the response at point T, collapsed across all trials and participants, is shown in Figure 9.8. As a whole the group did not appear to correct the 12ms shift completely.

The initial aims of the experiment were to generate a phase correction function for each child, to provide an individual measure of error correction ability. However, it was found that the degree of within-participant variability in the data meant that such modelling was not satisfactory and individual values could not be calculated with such a small number of trials. Rather than using error correction ability to predict performance on psychometric variables, only group comparisons were conducted to provide an examination of the differences in error correction ability in different ability groups. Due to the associations found in the previous chapters, groups were created based on median splits for reading, attention and reasoning, allowing comparison of the error correction performance of participants with good and poor abilities on these measures.

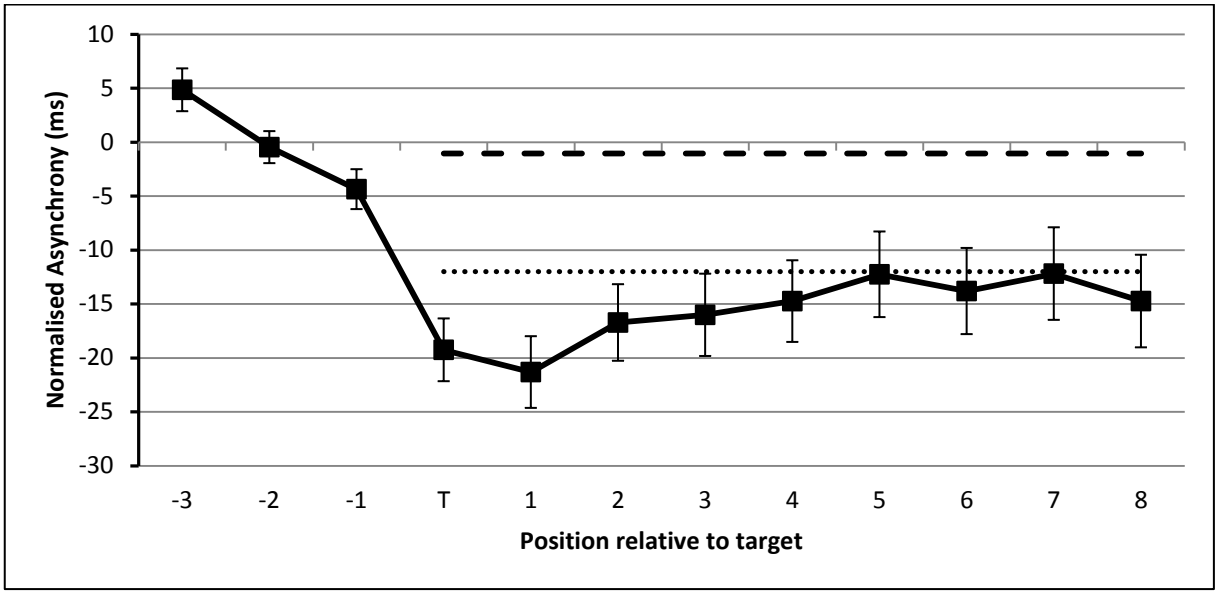

Figure 9.7: Phase correction response (normalised on asynchrony before shift)
Group mean asynchrony across the sequence relative to the position of the target phase shift of 12ms (T). Individual asynchronies normalised based on the participant's average asynchrony for the three responses before the target 12ms phase shift so that the pre-shift baseline lies at zero (error bars represent 2 SEM). The upper dotted line (- - -) represents a conservative lower limit of the pre-shift baseline average (2 SE below the pre-shift baseline mean). The lower dotted line (....) represents the expected asynchrony following the target shift if no correction has occurred.

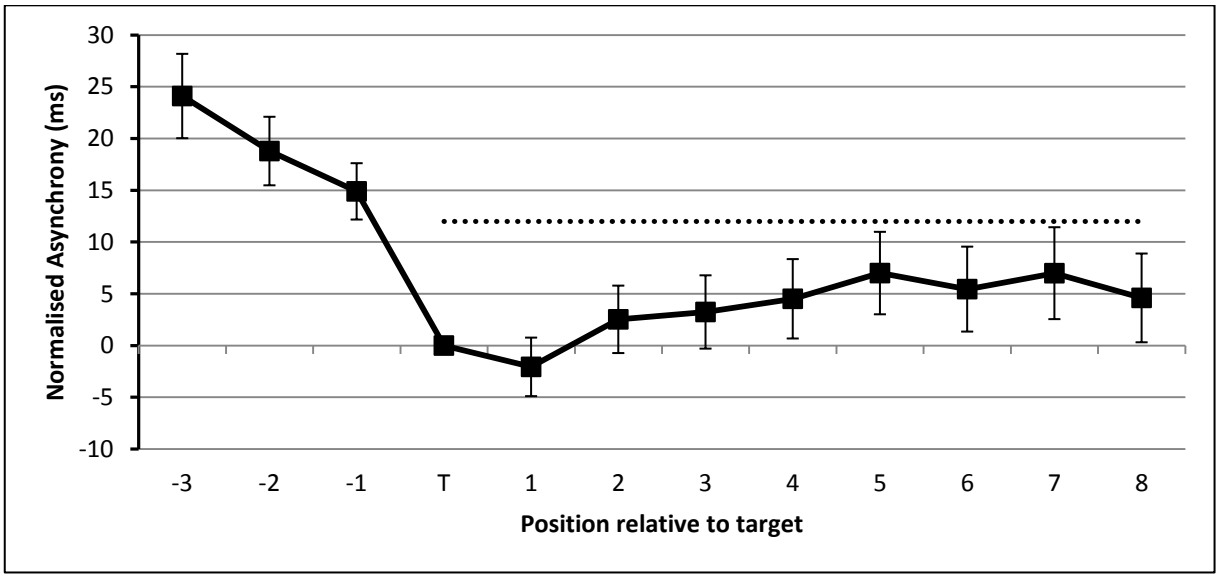

Figure 9.8: Phase correction response (normalised on asynchrony at point T)
Group mean asynchrony across the sequence relative to the position of the target shift of 12ms (T). Individual data normalised relative to the participant's average asynchrony at point of target 12ms phase shift so that the pre-shift baseline lies at zero (error bars represent 2 SEM). The dotted line (....) represents the expected asynchrony following the target shift if correction has occurred.

9.4.2.4.b. Synchronise-and-continue task

Both the synchronisation and continuation phases of the task were analysed to give Mean IRI and IRI SD. The first five responses at the start of each trial were eliminated from the start of each phase to allow stability to be achieved. All participants completed three trials with minimal tapping errors (average number of taps missing was zero, and maximum for one participant in one trial was six).

9.4.3. Results

9.4.3.1. Descriptive statistics

Descriptive statistics for all participants are shown in Table 9.1. The children had verbal and non-verbal reasoning abilities in the average range as well as average word efficiency (TOWRE) and fluency (PhAB) scores. On the phase correction task participants noticed the 90ms distracter shift on average in 87.5% of trials (SD 13.6).

Table 9.1: Descriptive statistics for all participants

	Mean (standard deviation)	
Age (months)	120.3	(3.0)
Verbal Reasoning	54.7	(8.9)
Non-verbal Reasoning	50.4	(6.7)
Total Word Reading Efficiency	102.9	(11.2)
Alliteration	104.1	(11.8)
Rhyme	102.8	(11.6)
Processing Speed Index	104.5	(8.8)
ADHD-IA Rating (max rating 27)	5.3	(5.8)
ADHD-HI Rating (max rating 27)	3.6	(5.6)
Simple Reaction Time (ms)	333.9	(56.9)
Simple Movement Time (ms)	361.1	(107.4)
Stop Signal Reaction Time (ms) (last 16 trials)	211.3	(63.6)

Notes: All scores are standard scores unless otherwise indicated. ADHD-IA and ADHD_HI are inattention and hyperactivity-impulsivity subscale scores.

9.4.3.2. Group comparisons

9.4.3.2.a. Error correction responses and reading ability

A median split was performed on the TOWRE total efficiency measure (TWRESS), resulting in a group of 14 poor readers (mean TWRESS = 94.1, SD 9.2) and 15 good readers (mean TWRESS = 111.1, SD 4.7). Examining the performance of these groups on the other cognitive measures showed that the poor reader group also scored significantly below the good readers on the Alliteration subtest of the

PhAB measure (poor readers = 97.5, SD 7.2; good readers 110.2, SD 12.0; t=-3.42, df = 27, p<0.01) but did not differ significantly in age or reasoning ability scores.

A compensation function with asynchronies normalised on asynchrony at point T were plotted for each group (Figure 9.9) and t-tests were used to examine whether correction had occurred. Firstly, the difference from the zero baseline was assessed at points from T onwards. For the poor reader group the asynchronies were not significantly different from zero at any point (the asynchrony at T+5 only approached significance, t = 2.04, df = 319, p=0.04, all other comparisons n.s., minimum p value = 0.2). The asynchronies for the good readers showed more correction over time, with responses at T+3 and T+5 to T+8 that were significantly different from zero (T+3: t = 2.75, df = 349, p<0.01; T+4: t=2.39, df = 349, p=0.02 and p<0.01 for comparisons from T+5 to T+8). These tests were repeated to assess the degree to which the 12ms shift was corrected (i.e. in comparison to the target pre-shift baseline of 12ms). These analyses showed a lack of correction for the poor readers, with asynchronies being significantly different from 12ms at all points (p<0.01; except T+5 (p=0.07) and T+7 (p=0.03)). In contrast, the good readers showed a correction response; asynchronies from T+5 onwards were not significantly different from 12ms (all comparisons n.s., minimum p-value = 0.06 at T+8).

A final test of the degree of correction of the two groups assessed the slope of the averaged asynchrony values from T to T+5 using a least squares regression procedure. Both groups showed a linear trend towards the target correction value, but the slope for the group of good readers exceeded that for the poor readers, and accounted for a greater proportion of the variance in the data (good readers slope = 1.96, intercept = -1.74, r^2=0.79 and poor readers slope = 1.17, intercept = -1.08, r^2=0.67).

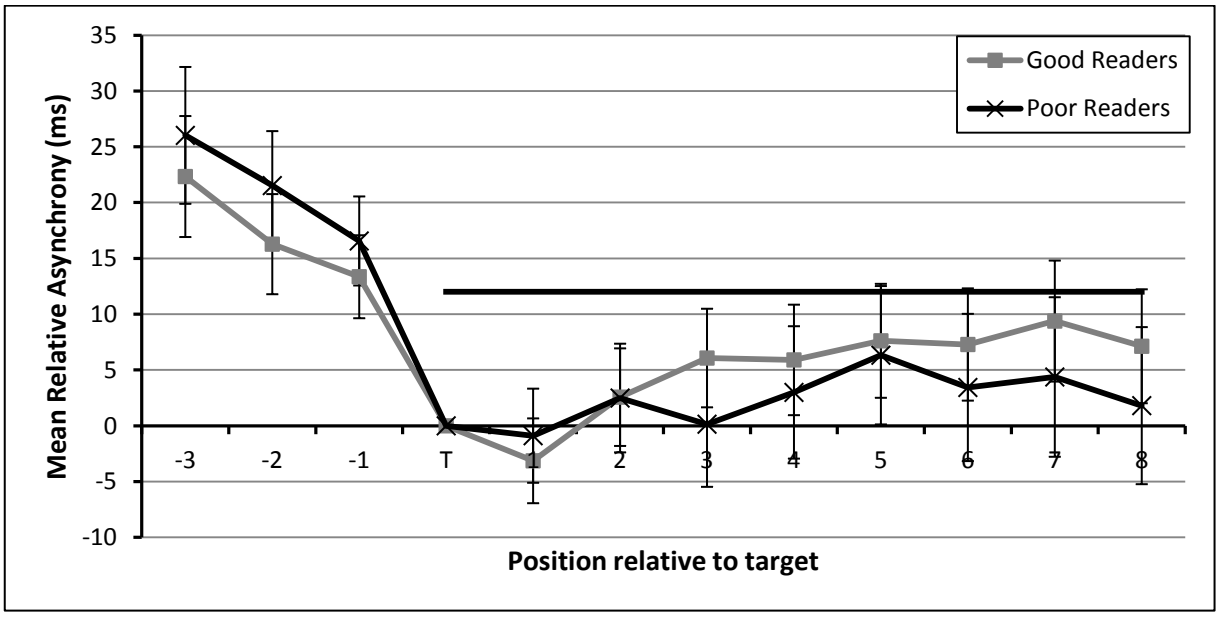

Figure 9.9: Phase correction responses for good and poor readers
Asynchronies are normalised on the asynchrony at point T (error bars represent 2 SEM). The dotted line (....) represents the expected asynchrony following the target shift if correction has occurred.

9.4.3.2.b. Error correction responses and attention

To provide groups with good and poor inhibition performance a median split was performed using the SSRT measure of attentional control. The correction responses for the groups are shown in Figure 9.10. There were 14 children in the group with longer reaction times on the SSRT measure (good inhibition; mean RT = 260ms, SD 55) and 15 in the group with shorter reaction times (poor inhibition; mean RT = 166ms, SD 26). These two groups did not differ on any of the other psychometric measures.

The children with better stop signal performance showed a steady correction of the phase shift, with averaged asynchronies from T+2 onwards being significantly different from zero ($p<0.01$ for all comparisons), and asynchronies at points T+4 onwards not differing significantly from the target of 12ms (T+4 onwards smallest p-value = 0.11). The children who had shorter SSRTs did not correct to the same extent. At no point were the asynchronies significantly different from the zero baseline (smallest p value at T+1 = 0.10) and the asynchronies were significantly different from the target correction value of 12ms at all points except at T+7 (T+7: $p<0.04$, all other comparisons $p<0.01$). It would appear that the poor attention group were slower to correct to a level that was not significantly different from the pre-shift level. The degree of linear slope in the functions from T to T+5 also indicated a group difference in error correction (good SSRT performance slope = 1.92, intercept = -2.50, $r^2 = 0.83$, poor SSRT performance slope = 1.28, intercept = -0.44, $r^2 = 0.62$).

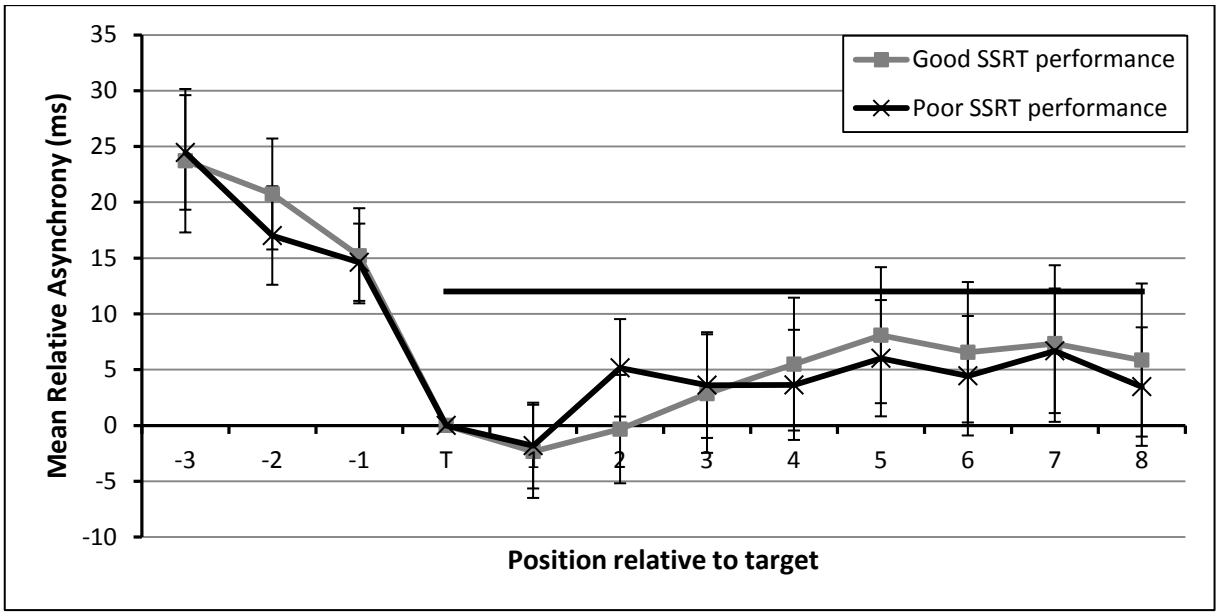

Figure 9.10: Phase correction responses for SSRT groups

Asynchronies are normalised on the asynchrony at point T (error bars represent 2 SEM). The dotted line (....) represents the expected asynchrony following the target shift if correction has occurred. Slope function after target shift measured with least square regression line plotted from T to T+5.

9.4.3.2.c. Error correction responses and reasoning ability

Based on evidence that timing performance is affected by reasoning ability (Madison et al., 2009; McAuley et al., 2006), the phase correction response was also compared across groups split on

reasoning ability. A median split based on non-verbal reasoning was performed to create groups with low (n=16, mean reasoning score = 93.3, SD 5.9) and high reasoning abilities (n=13, mean reasoning score = 109.6, SD 6.0). These groups did not differ on any of the other psychometric measures, with only the group differences in verbal reasoning and movement times approaching significance (p=0.02 and p=0.03 respectively). The average compensation functions for the reasoning groups are displayed in Figure 9.11.

The group with high reasoning scores, did not show any correction in the first three or four taps after the phase shift (with mean relative asynchrony not significantly different from zero at T+1 to T+3, p>0.2 and T+4, p=0.06). The mean asynchronies at T+5 and T+7 were significantly different from zero, and those at T+6 and T+8 approached significance (all at p=0.02). The mean asynchrony values differed significantly (p<0.01) from 12ms until T+4 after which they were not significantly different from 12ms (p>0.04) indicating that correction towards the pre-shift level had occurred. The group with low reasoning scores had asynchronies that were not significantly different from zero at all points from T onwards ($p \geq 0.04$ for all comparisons; smallest p value of 0.04 at T+5) and were significantly different from 12ms at all points (p<0.01) except T+5 and T+7 (p=0.02) indicating a lack of correction to the pre-shift level. Again, the slope and r^2 values suggest that more correction occurred in the group with high reasoning abilities (high reasoning ability slope = 1.90, intercept = -2.91, $r^2 = 0.76$, low reasoning ability slope = 1.25, intercept = 0.18, $r^2 = 0.56$).

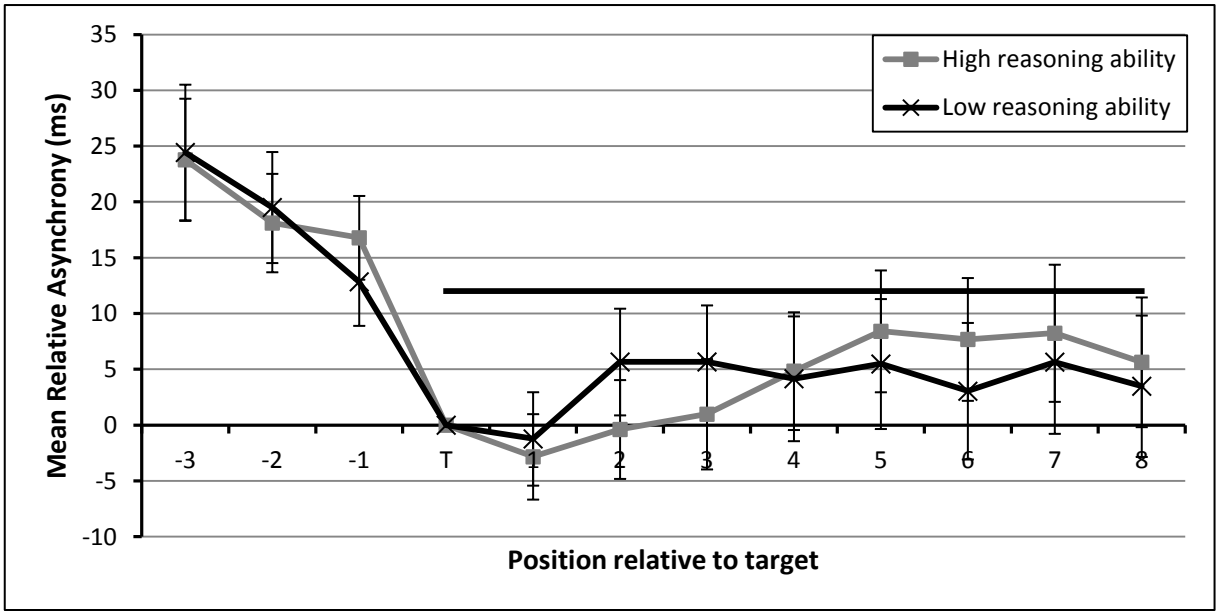

Figure 9.11: Phase correction responses for high and low reasoning groups
Asynchronies are normalised on the asynchrony at point T (error bars represent 2 SEM). The dotted line (....) represents the expected asynchrony following the target shift if correction has occurred. Slope function after target shift measured with least square regression line plotted from T to T+5.

9.4.3.2.d. Error correction responses and motor timing performance

To evaluate the relationship between overall performance on motor timing tasks and error correction ability, the final group splits compared participants who had high or low variability (IRI SD)

on the synchronisation and continuation motor timing tasks (Figure 9.12 and Figure 9.13). The IRI SD measure was used to operationalise timing performance in the previous chapters. This resulted in a group of 14 children with high variability (synchronisation variability = 49ms SD 5.9; continuation variability = 49ms, SD 5.0) and a group of 15 children with low variability (synchronisation variability = 32ms, SD 5.0; continuation variability = 31ms, SD 4.1). The PCR functions are shown in Figure 9.12 and Figure 9.13.

For the children with high variability on the synchronisation task, the mean relative asynchrony was not significantly greater than zero at any point from T+1 to T+8 (all comparisons n.s., smallest p value at T+7 = 0.03). All values were significantly different from the target correction magnitude of 12ms ($p<0.01$) at all points except T+5 ($p=0.03$) and T+7 ($p=0.24$). This suggests correction was slow and incomplete, even eight taps after the shift. In comparison, the children with low synchronisation variability showed rapid correction to the shift. Their mean asynchrony was significantly greater than zero ($p<0.01$) at T+3, T+5 and T+6 and comparisons approached significance for T+4, T+7 and T+8 ($p<0.02$). The comparison with the pre-shift level of 12ms indicated that correction had occurred by T+5 and T+6 ($p=0.16$ and $p=0.06$), but this was not maintained with the difference at T+7 and T+8 approaching significance ($p=0.03$ and $p = 0.02$).

For the children with high variability on the continuation task, the mean relative asynchrony was not significantly greater than zero at any point from T+1 to T+8 (all comparisons n.s., smallest p value at T+5 = 0.09). The values were also significantly different from the target of 12ms at T+1 to T+4 ($p<0.01$), but not from T+5 onwards ($p>0.03$). It appears that these children rapidly corrected about five taps after the shift had occurred. The group with low variability on the continuation task also showed rapid correction, with values from T+3 onwards being different from zero (T+3: $p=0.01$, T+4 onwards: $p<0.01$), although the values were only significantly different from the target of 12ms at T+4, T+5 and T+7, indicating incomplete correction by T+8. The degree of linear slope in the PCR function was calculated for each group for responses from T to T+5 and results are shown in Table 9.2. The slopes were steeper and accounted for more of the variance in the data in the groups with low variability (both synchronisation and continuation).

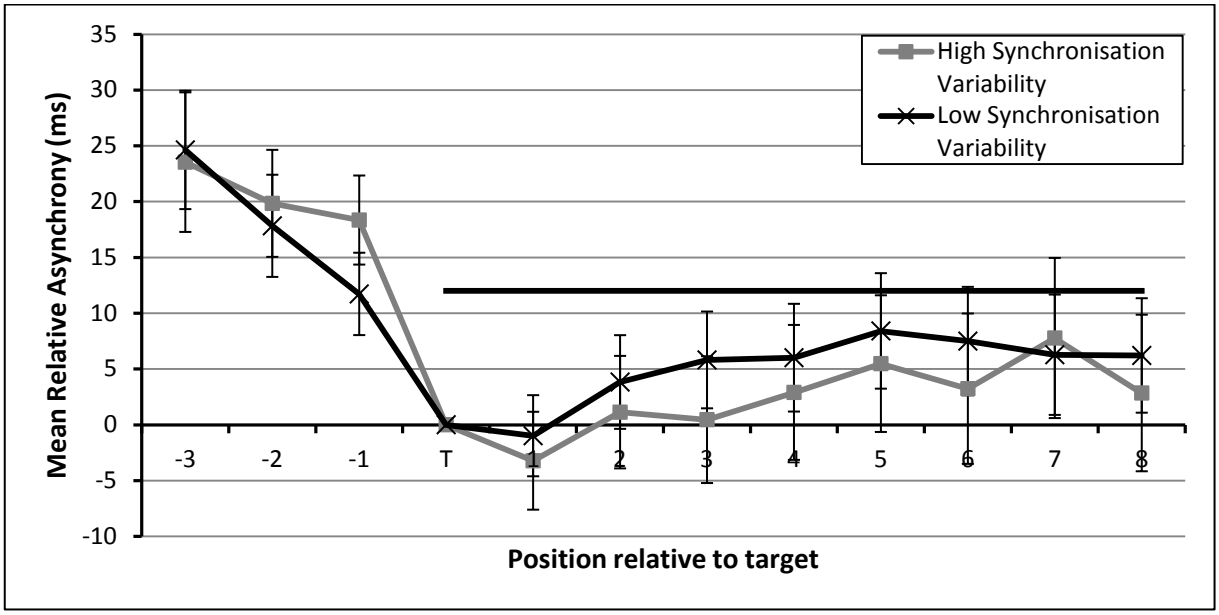

Figure 9.12: Phase correction responses for synchronisation groups
Asynchronies are normalised on the asynchrony at point T; error bars represent 2 SEM.

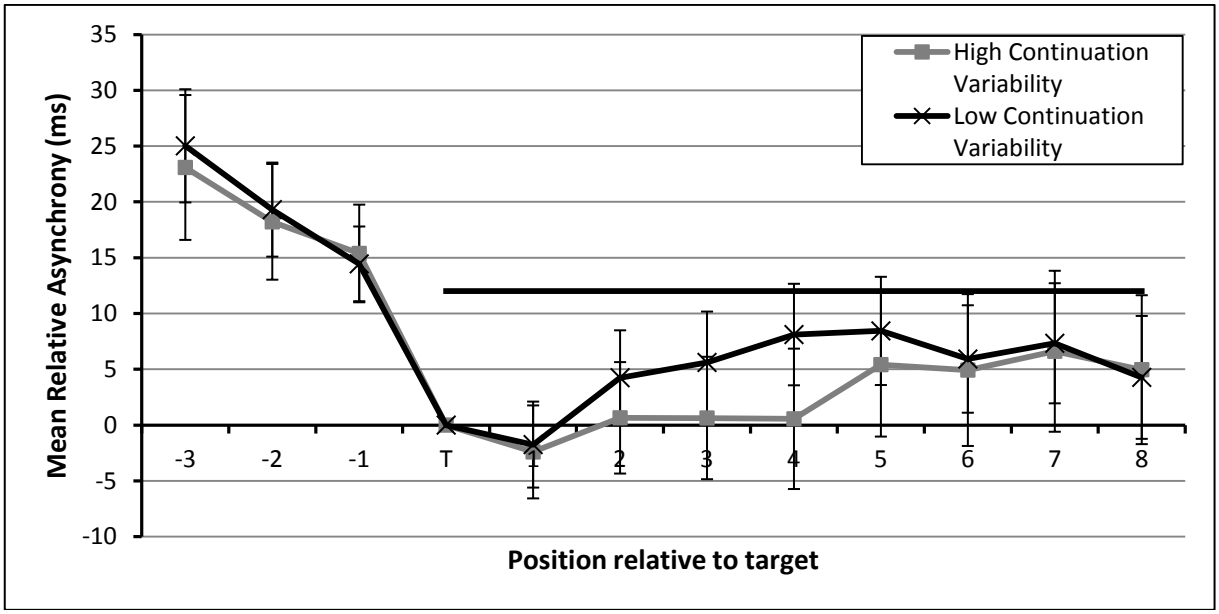

Figure 9.13: Phase correction responses for continuation groups
Asynchronies are normalised on the asynchrony at point T; error bars represent 2 SEM.

Table 9.2: Slope of function after target shift: Motor timing groups

Group	Slope	Intercept	r^2
High Synchronisation Variability	1.29	-2.10	0.68
Low Synchronisation Variability	1.86	-0.80	0.89
High Continuation Variability	1.03	-1.76	0.57
Low Continuation Variability	2.09	-1.12	0.87

Slope calculated using least square regression from T to T+5

In previous chapters, it was reported that children with poor reading or attention scores had greater variability in their timing response. Here it is apparent that groups with high variability take longer or fail to correct errors in timing behaviour when compared to groups with low variability. The relationship between timing variability and the psychometric variables may have accounted for the difference in error correction performance in groups divided based on measures of reading or attention. To test this, the groups created for the examinations of error correction above (high/low readers, high/low attention, high/low reasoning scores) were compared on synchronisation/continuation ability (Table 9.3). This showed that the reading and attention groups differed in synchronisation variability with children with low scores on these measures having significantly more variability on the synchronisation task than children with high scores. This difference in variability may have contributed to the differences in error correction that were recorded. The groups divided on reasoning scores did not differ in synchronisation or continuation variability.

Table 9.3: Motor timing performance across groups

Group		**Synchronisation Mean IRI SD (ms) (SD)**			**Continuation IRI SD (ms)**		
Reading	Low	46	(9.7)	**	43	(9.9)	n.s.
	High	36	(8.3)		38	(10.8)	
SSRT Inhibition	Low	44	(11.9)	*	43	(12.2)	n.s.
	High	37	(6.8)		38	(8.5)	
Non-verbal Reasoning	Low	41	(10.9)	n.s.	41	(11.6)	n.s.
	High	40	(9.4)		39	(9.3)	

*Group differences: *p<0.05, **p<0.01, n.s. non significant*

9.4.3.3. Relationships between timing performance and cognitive predictors

To examine the same relationships between timing performance and the psychometric measures reported in the previous chapters in the thesis, Pearson's product moment correlations controlling for reasoning ability were performed (Table 9.4). Timing performance was operationalised using the measures of variability (IRI SD) from the paced and unpaced motor timing tasks that were administered. Measures of phonological ability, reading fluency and processing speed were used to examine which components of reading ability are related to motor timing performance. Measures of reaction time and movement speed (from the CANTAB battery) were included to confirm previous findings that general movement ability is not related to timing performance.

In the zero-order correlations paced synchronisation performance was strongly and significantly correlated with total word reading efficiency (TWRESS) and with performance on the SSRT measure of inhibition. Performance on the unpaced continuation task was also strongly correlated with

processing speed. Correlations between the scores on the alliteration measure of phonological ability and motor timing variability were not significant. In the partial correlations controlling for reasoning ability, the same relationships remained significant although the association between unpaced timing variability and processing speed only approached significance (p=0.06). Significant correlations were found between SSRT performance and timing variability on both the paced and unpaced tasks and remained significant after controlling for reasoning ability. The reaction and movement time measures were not significantly associated with the measures of timing performance.

Table 9.4: Correlations between timing performance and cognitive predictors

	Paced Variability	Unpaced Variability	PhAB Alliteration	TWRESS	PSI	SSRT	Reaction Time	Movement Time	Verbal Reasoning	Non-verbal Reasoning
Paced Variability		0.70**	-0.12	-0.43*	-0.2	0.42*	0.07	0.35	0.03	-0.06
Unpaced Variability	0.70**		-0.26	-0.25	-0.41*	0.47*	0.18	0.26	-0.02	-0.17
PhAB Alliteration	-0.14	-0.29		0.43*	0.10	0.09	-0.11	0.33	0.18	-0.01
TWRESS	-0.43*	-0.25	0.43.*		0.41*	-0.02	-0.13	-0.23	0.13	0.08
PSI	-0.18	-0.39[A]	0.17	0.46*		0.05	-0.36	-0.25	-0.15	0.22
SSRT	0.42*	0.45*	0.10	0.00	0.10		-0.04	0.14	-0.13	-0.21
Reaction Time	0.04	0.13	-0.19	-0.16	-0.26	-0.08		0.20	0.20	-0.18
Movement Time	0.35	0.24	0.34	-0.22	-0.24	0.11	0.19		-0.09	-0.16
Verbal Reasoning										0.47*

*Pearson's product moment correlations (top right) between psychometric variables of interest and motor timing performance on the paced and unpaced phases of the synchronisation-continuation task, with partial correlations controlling for verbal, non-verbal reasoning and age (bottom left); *p<0.05; **p<0.01 [A]p=0.06. PhAB=Phonological Assessment Battery, TWRESS=Total Word Reading Efficiency Standard Score, PSI=Processing Speed Index, SSRT=Stop Signal Reaction Time.*

9.5. General discussion

The purpose of this study was to provide an initial examination of the relationship between phase correction ability in paced timing tasks and cognitive predictors associated with reading and attention, with the aim of explaining the higher variability recorded in children with reading difficulties. The evidence from the previous chapters in the thesis indicates that the motor timing difficulty experienced by children with reading difficulties is likely due to differences in mechanisms within the automatic timing system which is responsible for error monitoring and correction (Lewis & Miall, 2003; Repp, 2005). Therefore, a failure in the ability to correct errors may underlie the increased variability reported in these children.

The phase correction paradigm employed here relied on averaging the phase correction response over multiple trials. Although each participant carried out 24 trials, this was ultimately not sufficient to generate a consistent correction function that could be used to provide a measure of error correction at the individual level. This precluded a direct analysis of the relationship between error correction, motor timing and the psychometric measures at the individual level. Instead, participants were divided into ability groups based on their scores on tests of reading, attention and reasoning, and the error correction ability in each group was assessed. Such group-wise analyses allowed a preliminary examination of error correction variables and should act to stimulate further research examining this component of timing performance in populations with developmental disorders.

Previous studies have shown that adults correct positive phase shifts of 8-15ms within 2-5 taps of the occurrence of the shift (Praamstra et al., 2003; Repp, 2000). The group-wise statistical comparisons showed that children with better performance on the reading, attention and non-verbal reasoning tasks corrected timing errors in a manner which resembles that of adults. The correction response was slightly slower than in adults, but correction of the phase shift typically occurred by the fifth tap following the shift. The children with better performance on the psychometric measures showed a consistent incremental increase in the degree of correction between the point at which the error was created (the target phase shift) and the fifth tap, suggesting that they were iteratively correcting a proportion of the asynchrony with each subsequent response. In contrast, the children with poorer performance on the reading, attention and reasoning measures did not correct the asynchrony created by the phase shift to the same extent. There was some evidence of a degree of rapid correction, with about half of the asynchrony created by the phase shift (about 5ms) corrected within two taps of the shift, but no further correction occurred subsequently in the children with low scores on the psychometric measures. In the absence of further correction of the asynchrony, this initial rebound may be due to the general fluctuations within the tapping response, rather than a specific correction response.

Given that children with reading difficulties (Thomson & Goswami, 2008; Wolff, 2002; Chapter 8) and poor attentional control (Ben-Pazi et al., 2006; Toplak & Tannock, 2005b) show increased variability in their motor timing behaviour, the difference in error correction between the groups divided by relevant psychometric variables was expected. The difference found between the good and poor reasoning groups may be explained by the evidence that higher general reasoning ability is associated with less variable motor timing performance (Holm et al., 2011; Madison et al., 2009; McAuley et al., 2006; Waber et al., 2000).

Due to the difficulties already described in generating individual measures of error correction performance it was not possible to measure the relationship between phase correction ability and measures of general timing performance on the paced and unpaced tasks. However, the correlations

between general timing performance variability (IRI SD) and the psychometric variables replicated results from previous chapters with motor timing performance associated with literacy and attention related variables in the same direction and with similar effect sizes to those reported earlier. The use of additional measures of reading components allowed the relationships with literacy to be examined further. Reading efficiency, a measure of reading fluency was associated with timing variability under paced conditions. In comparison, processing speed was associated with timing variability under unpaced conditions. This fits with the notion that it is components of the synchronisation task, such as the combination of multiple elements in time that is related to reading. In comparison, processing speed, as a measure of general cognitive efficiency may better reflect the processes necessary for maintaining timing in the absence of stimuli. Although studies have been conducted to examine how general ability relates to timing performance (Holm et al., 2011; Madison et al., 2009; McAuley et al., 2006; Waber et al., 2000), the effect of general reasoning ability on different components of timing behaviour has not been investigated. If the present results were to hold, then general ability should be associated with the elements of timekeeping that are more heavily relied upon in unpaced tasks (e.g. the beat-based system and/or auditory rehearsal).

That behavioural inhibition was, in comparison, associated with timing performance across both the synchronisation and continuation phases is again indicative of a difference in the types of timing skills associated with reading and attention related elements. It may also support the suggestions that the phenotype of ADHD is related to impairments in the beat-based clocking component of the timekeeping system, as described in Chapter 8. As in the studies in Chapter 6 and 8, the variability in motor timing was not strongly correlated with reaction time, or with the additional measure of movement time that was introduced in this study. This indicates that motor timing variability is unlikely to be accounted for by general movement ability, concurring with results from earlier studies (Thomson & Goswami, 2008).

In previous chapters it was proposed that children with reading difficulties have an impairment within the automatic part of the timing system that is relied upon for maintaining synchrony in millisecond timing tasks. This part of the timing system (operationalised as the timekeeper component in the Wing and Kristofferson model) is essential for maintaining accurately timed responses during synchronisation tasks. Pulses from the timekeeping system act as an internal representation of tempo and allow predictions to be made about the sensory consequences of behaviour (Mates, 1994). Anticipatory and predictive representations may then be compared to the sensory feedback received following stimuli and responses in order to assess the success of timing behaviour (Kotz et al., 2009; Stenneken et al., 2006). The error correction task measures the efficiency of this system in dealing with disturbances in synchrony. Due to the difficulties in producing an individual measure of error correction performance, the present results require replication, but they do provide tentative support for poor readers having an impairment in such a

tempo maintenance system. The group-wise analyses of error correction in good and poor readers suggested a trend towards less effective correction of errors in poor readers compared to good readers. These results also correspond to previous evidence that children with reading difficulties have larger anticipation times during motor timing tasks (tapping further from the stimulus onset) than control children (Wolff, 2002) and are similarly suggestive of a mismatch between the coordination of action and perception required to achieve synchronisation.

These results may also help to explain why associations have been found in previous studies between motor timing performance variability and sensitivity to the location of p-centres (Thomson et al., 2006; Thomson & Goswami, 2008) (further explanation of p-centres was provided in Chapter 3, Section 3.2.2). Children and adults with reading difficulties typically show a lack of sensitivity to auditory p-centre locations when manipulated by altering the rise time of the amplitude envelope of a stimulus (Goswami, Gerson, et al., 2010; Goswami et al., 2002, 2011; Hämäläinen et al., 2005). In the typical rise-time task used in these studies, participants are asked to decide whether a sound sequence resembles a rhythmic beat or not. By altering the rise times of the sinusoidal tone the "beat" in the stimulus becomes more or less detectable. These authors propose that a p-centre deficit would affect sensitivity to the prosodic features of speech during language development.

Speech processing requires the prediction of elements occurring over time and relies on expectancy based feed-forward models similar to those required for the maintenance of synchronised motor timing behaviour (Arnal & Giraud, 2012; Arnal, 2012; Luo et al., 2010). Two studies have measured the relationship between p-centre sensitivity and variability in motor timing synchronisation performance and found that they were moderately correlated (r^2=0.1-0.4) (Thomson et al., 2006; Thomson & Goswami, 2008). Regression analyses in these studies indicated that rise time sensitivity and synchronisation ability were independently related to performance on literacy measures. However, it is possible that the associations between these two measures and reading/spelling performance can both be accounted for by the same mediating variable; the ability to detect asynchrony (i.e. insensitivity to small changes within temporal stimuli like phase shifts).

The success of synchronisation behaviour is mediated by the judgement of the synchrony of p-centres in the stimulus train (Pöppel, 1997; Vos et al., 1995). Thus, judgements of isochrony and synchrony are not dependent on the physical onsets or peak stimulus intensities, but on the prediction and judgement of the time at which the next stimulus will be perceived (Villing et al., 2011). Villing et al. (2011) recently demonstrated that the correction response to phase shifts in a synchronisation motor timing paradigm (finger tapping to speech syllable stimuli) can be used to generate an estimate of the location of a p-centre within a stimulus. More commonly, the p-centre location is estimated by asking participants to adjust stimuli until they are judged as being synchronous. Villing et al. found that the estimate generated using the phase-correction paradigm

corresponded well with that derived from the adjustment paradigm, and was equally precise. This indicates that there are similarities in the processes measured by the rise time and synchronisation paradigms that may explain the associations previously found between motor timing behaviour and p-centre sensitivity.

The overlap between such paradigms is not surprising given that both error correction and the learning of frequently encountered sounds during language development rely heavily on the degree of coupling between sensorimotor events (Repp et al., 2012; Westermann & Miranda, 2004). Use of the phase correction task alongside the rise time judgement task may provide a useful alternative method for estimating p-centre sensitivity in children. The phase correction task, administered through a synchronisation task does not rely on verbal reports from children regarding the perception of synchrony (or the presence of "a beat") and so may allow more accurate measurements of these abilities in studies of populations where verbal reports cannot always be relied upon. In order to generate a greater number of phase shift responses more efficiently, multiple shifts can be introduced within a single trial (e.g. one every 10 taps; Repp, 2000).

Whilst the two difficulties present in children with reading difficulties (with motor timing variability and p-centre sensitivity) may be related, the origins of the deficits remains unclear. One explanation is that these task performance impairments arise from a failure to identify the locations of p-centres. Such a mechanism could be present early in life and affect the judgement of isochrony in both the motor timing and p-centre tasks which rely on the judgement of the synchrony of p-centres in the stimulus train (Pöppel, 1997; Vos et al., 1995). Alternatively, the difficulties may be due to the existence of greater variability in the timing system used to perform these tasks. Such variability would produce greater tolerance of uncertainty around the occurrence of sensory events with temporal characteristics. Children with reading difficulties would therefore be less aware of asynchronies in temporal stimuli. The amount of variability occurring around temporal events affects the ability to compensate for both internally or externally generated asynchronies (Vroomen & Keetels, 2010). If variability were present in the timing systems of these children early in childhood they may, over time, develop a lower coupling strength between sensory and motor events; coupling that is necessary for judgment of p-centres and accurate synchronisation performance.

As described in Chapters 3, 6, and 8, symptoms of ADHD are also found to be associated with increased variability on motor timing tasks (Ben-Pazi et al., 2003, 2006; Rubia et al., 2003; Rubia, Taylor, et al., 1999; Valera et al., 2010; Zelaznik et al., 2012). The timed tapping response of children with ADHD is also characterised by the presence of hastening phenomenon where children tapped faster than the target mean ISI (Ben-Pazi et al., 2003, 2006; Jucaite et al., n.d.). These deficits have been explained with reference to the lack of inhibition typically shown by children with ADHD (Nigg, 2001; Schachar et al., 1995; Walshaw et al., 2010). A lack of behavioural inhibition typically affects

performance on time judgement tasks because children respond prematurely or fail to inhibit responses (Noreika, Falter, & Rubia, 2013b). Here, children who were less able to delay their responses on a behavioural measure of inhibition were found to show less correction of phase shift asynchronies than children with better performance on the inhibition measure. As with the hastening phenomenon reported by Ben Pazi and colleagues in which children with ADHD maintain an inappropriate response rate, the children with poor inhibitory control may have been attempting to tap at a single response rate, irrespective of the presence of the phase shifts in the stimuli. Zelaznik et al., however, argue that children with ADHD implement a closed loop approach to timing tasks in which they cannot inhibit the use of sensory feedback, and therefore always attempt to use any sensory feedback available to them. This is incompatible with the present data which indicates that children with low scores on the inhibition measure failed to use information about asynchronies to update responses. Whilst the correction of errors in millisecond timing tasks occurs unconsciously, there is a requirement in motor timing synchronisation behaviour for an *intention* to maintain synchrony (Repp, 2005; Schwartze et al., 2011). The children with difficulties with inhibition may have failed to attend to the requirements of the task (i.e. to tap in time) which moderated their response to phase shifts.

Conditions affecting the basal ganglia, like ADHD and Parkinson's disease, are found to affect the ability to maintain an accurate timekeeper response controlled within the pre-conscious timing system (Freeman et al., 1996; Harrington et al., 1998; O'Boyle et al., 1996; Valera et al., 2010; Zelaznik et al., 2012). However, they also impact upon temporal processing that demands overt attention to stimuli. Studies of patients with basal ganglia damage indicate that these patients have difficulties dealing with the correction of period changes (see Figure 1.) which require the registration of interval size and memory for these intervals (Grahn & Brett, 2009; Schwartze et al., 2011). In comparison, the phase correction task used here relies upon pre-attentive mechanisms and making comparisons between the times of occurrence of stimuli rather than any registration of the size of subsequent intervals. Thus, although children with attention difficulties (particularly impulsivity) appear to show difficulties on a phase correction task, this may be due to a separate mechanism in failing to inhibit their ongoing response, or similarly a failure to pay attention to stimuli. These results mirror those from Chapter 6 in which children with impaired sustained attention selected a less appropriate strategy for dealing with the stimuli presented to them (under visually paced task conditions; see Section 6.4.3.2).

This study has been useful to demonstrate the ease of use of the motor timing paradigm in measuring error correction performance in children and the potential for advancing understanding of the relations between error correction and p-centre sensitivity in dyslexia. However, there were some limitations of the methodology which will need to be addressed in applying the paradigm in future. All participants showed a trend of decreasing asynchrony in the three responses before the

shift suggesting that they had not stabilised their performance by this time. This may have occurred because the number of stimuli (and therefore responses) prior to the shift was reduced to a minimum of 8 (with the phase shift occurring in a jittered position after 8-11 stimuli) in order to reduce the trial length. To analyse errors in the presence of this pre-shift trend, responses were analysed relative to the asynchrony at the time of the shift as described by Repp (2011, Experiment 3A) so that the success of phase correction could be assessed by examining the degree to which the 12ms asynchrony was reduced. Whilst this strategy yields almost identical results to other strategies (e.g. those used in the pilot experiments where responses were analysed relative to asynchronies occurring before the shift) (Repp, 2011), ensuring that children have fully entrained to the stimulus rate prior to the shift would be beneficial in future experiments of this kind. This would ensure that error correction was not affected by the degree to which participants had already achieved a steady response output. Due to the amount of data that is required for generating phase correction functions, it is also recommended that data is collected from children over several testing sessions over a number of days and that multiple phase shifts be introduced into each trial (as used previously by Repp, 2000).

10. Discussion

10.1. Aims and rationale

This thesis was first motivated by observations that motor timing difficulties are present in children with reading difficulties and that these have not previously been considered in the context of models of typical human timing behaviour. The review of research in the field of timing in Chapter 2 demonstrated the range of methods and approaches available for assessing time based processing and the state of understanding of typical human performance on time related tasks. With a focus on motor timing tasks, statistical modelling methods were examined that enable analysis of the mechanistic elements underlying performance on motor timing tasks.

Chapter 3 re-examined the evidence for a motor timing difficulty in dyslexia in the context of the timing perspective. Discussion of the relevance of such a difficulty in light of the involvement of temporal processing in the development of reading building on the acquisition of appropriate sound-based representations of language was also presented to illustrate the importance of examining temporal processing deficits in dyslexia. Typically dyslexia is associated with increased variability in motor timing responses, especially when performance is synchronised to pacing stimuli (Badian & Wolff, 1977; Klipcera et al., 1981; Thomson et al., 2006; Thomson & Goswami, 2008; Wolff et al., 1984; Wolff, Michel, Ovrut, et al., 1990; Wolff, 2002). In addition, children with dyslexia are typically able to maintain an appropriate tapping rate but show greater asynchrony between stimuli and responses in motor timing tasks (Thomson & Goswami, 2008; Wolff, 2002). Evidence for greater motor timing performance in children with ADHD was also reviewed because of the overlap commonly observed between symptoms of dyslexia and ADHD. It was judged appropriate to investigate timing in relation to the symptoms of these two developmental disorders in order to establish the extent to which the timing deficit (or aspects of it) was specifically related to symptoms of each disorder or the overlap between them. Such research helps to establish whether cognitive factors which overlap are related to the high degree of diagnostic overlap between the disorders. Understanding shared and unique elements of the disorders is particularly relevant given the current recognition of the multi-factorial nature of the biological and cognitive risk factors that underlie these disorders (Cramer et al., 2010; Snowling, 2008; Thomas & Karmiloff-Smith, 2002).

This review led to the development of the research aims which were to apply the Wing-Kristofferson linear model of time series analysis to motor timing data from children to assess the relative contributions of timing components to motor timing variability in children with reading and attention difficulties. Use of this model throughout the thesis allowed variance in timing to be separated into that attributable to mechanisms within the timekeeping system and that attributable to implementation systems. The review of motor timing studies conducted with populations of children with dyslexia and ADHD also revealed inconsistencies in the application of stimulus characteristics in

studies that had examined timing performance in children with dyslexia and ADHD. Therefore additional research questions were posed, to examine the effects of auditory, visual and bimodal stimuli on timing performance and to investigate whether timing performance under each stimulus modality was equally related to symptoms of dyslexia and ADHD.

10.2. Overview of experimental chapters and key findings

10.2.1. Chapter 6: Effect of stimulus modality on motor timing performance

Motor timing behaviour had previously been measured using acoustically paced motor timing tasks with participants with dyslexia (Badian & Wolff, 1977; Thomson et al., 2006; Thomson & Goswami, 2008; Wolff et al., 1984; Wolff, Michel, Ovrut, et al., 1990; Wolff, 2002) and using visually or bimodally paced tasks with participants with ADHD (Ben-Pazi et al., 2003, 2006; Rubia et al., 2003; Toplak & Tannock, 2005b). This chapter therefore examined whether the different stimulus modalities produce comparable motor timing behaviour in samples of children and adults. The Wing-Kristofferson model (adjusted using the method recommended by Kooistra et al., 1997) was applied to the time series data to further interrogate the components of task performance under the different stimulus conditions and assess the contribution of the different underlying timing mechanisms to literacy ability and attention skills.

The findings replicated previous studies, showing that adults are less accurate at producing synchronised motor responses when pacing stimuli are presented visually than when they are presented bimodally (combined visual and acoustic) or acoustically (Fendrich & Corballis, 2001; Jäncke, Loose, et al., 2000; Kolers & Brewster, 1985; McAuley & Henry, 2010; Patel et al., 2005). For the first time this modality effect was also replicated in a sample of children and results demonstrated that auditory tasks are most appropriate for analysing motor timing performance in studies of children. In comparison, when timing was visually paced, synchronised finger tapping performance was characterised by shorter intervals and greater asynchrony between stimuli and responses. Bimodal performance accuracy and stability was found to be generally intermediate to the other two conditions, as expected given that auditory stimuli tend to dominate over visual stimuli in bimodal conditions (Fendrich & Corballis, 2001) yet the presence of visual stimuli leads to a reduction in stability compared to unimodal auditory conditions (Repp & Penel, 2002).

When the relative contribution of implementation and timekeeper variance was examined differences were identified in the manner in which tasks were performed. The results supported previous proposals that visual task performance is guided using the internal timekeeping systems, relying less heavily on feedback from the visual stimuli, given that visual stimuli provide information with low temporal resolution (Grahn, 2012; Jäncke, Loose, et al., 2000; Jantzen et al., 2005; Repp & Penel, 2002) thus resulting in reduced timekeeper variance. Under visual conditions, children with

better performance on literacy and attention measures appeared to use this same strategic approach to the task as adults. In comparison, children with poor literacy and attention skills maintained a focus on the stimuli regardless of the inappropriateness of this strategy (giving rise to increased timekeeper variance and reduced implementation variance).

The key findings from this study were therefore that when visual stimuli are used to drive motor timing behaviour, less stable motor timing behaviour is produced than with auditory or bimodal stimuli. Secondly, children with attention or reading difficulties may be more susceptible to the constraints placed on performance under visual conditions due to the different manner in which they approach the task. In light of these results, studies that have previously used visual or bimodal tasks to assess timing performance in developmental populations with ADHD (Ben-Pazi et al., 2003, 2006; Rubia et al., 2003) may have been measuring the ability to select an appropriate timing strategy rather than the true capacity of the timekeeping system itself. Results from this study and Chapters 8 and 9 do however indicate that a greater number of symptoms of ADHD may also be associated with less effective timing performance on auditory tasks. Therefore should these previous studies be repeated using auditory tasks it is expected that group differences would still be found in performance between children with and without ADHD.

10.2.2. Chapter 7: Assessing the validity of the Wing-Kristofferson model

In the previous chapter (6) the Wing-Kristofferson time series analysis model was applied using the adjustments suggested by Kooistra et al. (1997). These adjustments accounted for events that would lead to the model assumptions being violated, such as negative variance estimates, limited time series sample sizes and the presence of drift in the data. Based on previous studies using these models with other non-expert populations (Carroll et al., 2009; Duchek et al., 1994; Harrington et al., 1998, 2004; Kooistra et al., 1997; O'Boyle et al., 1996; Pastor et al., 1992; Simmons et al., 2009; S. J. Wilson et al., 2002), such breaches of the statistical basis of a linear time series model were not unexpected and the frequency of failures to match the model were no more common in the experiments in Chapter 6 than in other studies. Because the presence of such factors can cause the timekeeper and implementation variance estimates to be over- or under-estimated an analysis was conducted to assess the different methods of dealing with these statistical inconsistencies in Chapter 7. This allowed an appropriate analysis strategy to be selected for Chapter 8 and increased confidence in the estimates calculated.

Four methods were compared, using time series data drawn from the unpaced phase of a synchronise-and-continue motor timing task completed by a sample of adults: (i) setting negative variance estimates to zero, (ii) eliminating trials which did not satisfy the model, (iii) selecting the first valid trial which satisfied the model, and (iv) using the adjustments proposed by Kooistra et al. (1997). Particularly at slow speeds of tapping, differences were identified between the methods,

speeds at which the presence of drift can have a greater impact on estimates. Methods (i) and (iv) were found to overestimate timekeeper variance and underestimate implementation variance. In comparison, the estimates drawn from methods (ii) and (iii) were not affected in this way due to the exclusion of many causes of violations. Method (iii) was therefore selected for use in Chapter 8. These results provided a replication of previous work that has tested the different analysis methods (O'Boyle et al., 1996) with the additional benefit of extending the validation analysis to include the Kooistra et al. adjusted model.

The finding that the Kooistra model tends to overestimate timekeeper variance and underestimate implementation variance, especially at slower speeds should be beneficial to future studies considering which methods of adjustment to apply. It was judged that this disparity in variance estimates calculated using the Kooistra model would have had minimal effects on the results from Chapter 6, where that model was applied, because of the relatively fast rate of motor timing employed in that study (3Hz). The recommendation from this study is that future studies actively monitor elements that contribute to the differences in variance estimates produced by different adjustment methods of the time series model, for example assessing whether participants do perform using open-loop timing control or whether they are able to maintain steady outputs overtime and not drift from the target IRI. This will not only help researchers to understand why violations of the model have arisen but also provide additional information about the manner in which participants control their timing behaviour.

10.2.3. Chapter 8: Assessing the nature of motor timing in children with reading difficulties

This study applied the Wing-Kristofferson model of time series analysis (using adjustment method iii described above) to examine the different components of timing variability that contribute to motor timing performance in children with reading difficulties compared to age and reading level matched controls. As well as assessing group differences, relationships between the components of timing and cognitive indicators were explored. Timing performance was measured across five tapping rates (from 1.5-3.5Hz; IOIs from 670 to 283ms) with many previous studies of timing in relation to dyslexia focusing on a narrower range of performance speeds, with indications that deficits in the 2Hz range (500ms intervals) should be key for the appropriate development of language skills (Dauer, 1983; Goswami, 2011; Talcott et al., 2000; Thomson & Goswami, 2008; Witton et al., 1998; Wolff, 2002).

A group difference in general performance variability (IRI SD) was only found at the slowest tapping rate of 1.5Hz (670ms IOIs) where the Clinic group were significantly more variable than the same age controls and it was suggested that differences at slower rates may be attributable to maturational effects. The groups also did not differ significantly in the amount of timekeeper or implementation variance demonstrated. Group differences had been expected given that the clinic group had poorer reading performance (overall, and on non-word and irregular reading tasks) than the age-matched

controls and that groups with dyslexia have previously been found to show impairments on this task. The clinic group selected for reading difficulties also had higher non-verbal reasoning skills than the other groups. Higher scores on reasoning tasks have been shown to be associated with improved performance on motor timing tasks (Madison et al., 2009; Madison, 2011; McAuley et al., 2006), and may account for the present failure to find group differences. In addition, it was noted that some of the previous studies have compared groups of dyslexic participants who had reading scores that were far more discrepant from those of the control participants than in the present study (Thomson & Goswami, 2008). Such differences between groups may mean that any group effects are more apparent against the background of generally high levels of performance variability in children.

Correlation analyses that controlled for reasoning ability and age indicated that poorer performance on measures of literacy ability (in particular, spelling, rapid naming and digit span) and the presence of a greater number of ADHD symptoms were associated with increased timing variability when finger tapping at 2.5Hz (402ms IOIs), correlations which survived controls for multiple comparisons. These findings are important because they replicate the statistically significant findings from earlier studies which had much greater variance in reading scores to be explained, with dyslexic participants being more severely discrepant in their reading scores than the control groups (Thomson & Goswami, 2008). When the associations with decomposed timing variance were examined, a difference was found in the way literacy and attention variables were related to timing components. Timekeeper variance was associated with a literacy component skill (rapid naming) and implementation variance was associated with ADHD symptoms. When conservative Bonferroni corrections for multiple comparisons were applied these associations did not survive. These results can therefore only be interpreted as preliminary evidence that the outputs from different components of the timing system are separately associated with symptoms of ADHD and dyslexia. Similar dissociations are found when different clinical populations are compared, such as those with disruption to the cerebellum or basal ganglia (Grahn & Brett, 2009; Harrington et al., 2004; Ivry & Keele, 1989) with the former affecting timekeeper variance alone and the latter affecting timekeeper, implementation variance and IRI accuracy. As such, it is proposed that dyslexia may be associated with a failure in the central timekeeping mechanism whereas ADHD is related to failures in the implementation system. Analysis of the presence of any violations of the assumptions of the Wing-Kristofferson model (e.g. the presence of non-stationarity, or closed-loop timing) showed that children with reading difficulties were no more likely to show drift in their time series and this factor did not account for the association between timing variability and rapid naming.

Although the result must be interpreted with caution due to the exploratory nature of the correlational analyses, the results can be considered in conjunction with those from Chapter 6, where poor reading was also associated with over reliance on the timekeeping system in conditions of temporal uncertainty (i.e. the visually paced task) where the output from the timekeeper would

typically be ignored. From this evidence it is anticipated that children with literacy difficulties have a failure in the automatic timekeeper system; likely not in the beat detection system, but in that responsible for predicting, monitoring and maintaining synchrony (as described further below, Section 10.3.1). In contrast to this, the associations between symptoms of ADHD and impairments in the implementation system indicate that although motor timing deficits in children with dyslexia and ADHD appear quite similar, once performance variability is decomposed using time series analysis methods differences between the disorders become apparent. Reading component skills were not, however, associated with implementation variance that reflects the function of the motor output systems and similarly reaction time was not associated with timing performance. This evidence supports previous evidence that the motor timing impairments in dyslexia are not attributable to a motor coordination impairment (Thomson & Goswami, 2008) and corroborates hypotheses that motor impairments are instead associated with the presence of ADHD symptoms in children with reading difficulties (Denckla et al., 1985; Raberger & Wimmer, 2003; Ramus et al., 2003; Rochelle et al., 2009).

10.2.4. Chapter 9: Error correction in motor timing tasks

On the basis of the results described above it was hypothesised that the motor timing impairments in dyslexia may be attributable to elements within the automatic timing system that contribute to the maintenance of synchronised timing outputs, more specifically in the systems that allow errors to be corrected. The studies in Chapter 9 therefore examined the extent to which good and poor readers were able to correct small synchronisation errors and whether this was associated with general motor timing variability. Synchronisation errors were created by introducing phase shifts within the ongoing train of synchronisation stimuli and responses to these errors were recorded. The paradigm, which has previously only been used with adults, was first piloted to establish its success in measuring phase correction in adults and children (Experiments 1 and 2). These experiments helped to develop the paradigm for the final experiment (3) in which typically developing children completed the error correction task and a synchronisation-continuation motor timing task.

Unfortunately, the small number of trials completed by each participant was insufficient to produce an individual measure of error correction performance. Instead, group comparisons were made across groups divided on reading, attention, reasoning and motor timing abilities. For each of these comparisons, results were in the expected direction with children who had higher scores on reading, inhibition and reasoning tasks showing more rapid and more complete error correction than children with lower scores on these measures. Similarly, children who had less variable performance on synchronisation or continuation motor timing tasks showed better error correction performance. Finally, general motor timing performance on the synchronisation and continuation tasks was found to be associated with measures of literacy and attention in the same direction as in the previous

chapters. Some dissociations were found between the types of literacy components that were associated with performance, with reading fluency correlating with paced motor timing and processing speed with unpaced performance. The results from this experiment provide preliminary evidence that error correction may be a likely contributor to the timing variability shown by children with reading difficulties.

The potential overlap between these difficulties and other temporal processing deficits, for example in detecting the perceptual-centres of stimuli was described (Goswami, Gerson, et al., 2010; Hämäläinen et al., 2005; Huss et al., 2011; Thomson et al., 2006; Thomson & Goswami, 2008). If replicated the differences in error correction abilities in poor readers may be explained by greater overall variability within the timing system, that cause increased tolerance to phase errors. Further research is required to confirm whether individual measures of error correction can explain the differences in performance in motor timing tasks in children with reading difficulties. Despite the methodological challenges the study demonstrated the utility of applying this paradigm to research in the field of timing in developmental disorders, particularly given the putative overlap between error correction performance and sensitivity to perceptual-centres of stimuli (Villing et al., 2011).

10.3. Implications for broader theory and recommendations for future research

10.3.1. Components of timing behaviour and reading ability

The results of the studies conducted here confirmed that reading difficulties are associated with increased variability in motor timing behaviour and suggested that this may be attributable to greater timekeeper variance estimated using the Wing-Kristofferson model. Furthermore, poor readers tended to rely less heavily on timekeeping mechanisms (especially during conditions of temporal uncertainty such as the visual task). The decomposition afforded by the Wing-Kristofferson model quantified two variance components. In simple terms, implementation variance is the variability that changes with each response across the series of responses due to the random delays in implementing each motor response through the peripheral motor system (not dependent on previous responses). Timekeeper variance is the remaining variability, or that which remains constant across the entire train of responses (due to the lag-one statistical dependencies within the system). Timekeeper variance, has been modelled as that attributed to a clock-like mechanism that outputs temporally stable pulses used to determine the duration of intervals (Church, 1984; Hinton & Meck, 1997; Wing & Kristofferson, 1973a). In practice however, the timekeeper variance component is a metaphor for a number of processes (that may utilise temporally dependant neural oscillatory activity) that enable regularly timed intervals to be produced (Madison, 2001b; Penhune et al., 1998). As such, the Wing-Kristofferson model is limited in pinpointing the origin of any increased variance any more precisely than to one or other of these processes. Figure 10.1 illustrates a number of the processes that contribute to the timekeeper variance component in the Wing-Kristofferson model,

prior to implementation of responses through the motor system. In order to reach more precise conclusions about the putative origin of the motor timing impairments associated with reading difficulties the various strands of evidence described in this thesis can be re-examined with reference to the elements shown in Figure 10.1.

Within the timing system there is firstly, an internal representation of the stimulus tempo that must be derived from perceptual inputs, a process attributed to the SMA and its connections through to the striate cortex (Grahn & Brett, 2007; Kotz et al., 2009; Schwartze et al., 2011; Wiener et al., 2010). For millisecond timing, the registration of stimulus intervals is thought to occur by recognition of the moments of occurrence of stimuli rather than registration of the interval duration itself (Repp, 2005). Initially, timekeeper outputs are based on this sensory detection but once a representation is established, future stimuli are anticipated and predicted using forward-models processed through sensorimotor-cerebellar networks (Drewing & Aschersleben, 2003; Kotz et al., 2009). The ongoing representation of time intervals is then maintained in the SMA and IFG (Buhusi & Meck, 2005; Grahn & Brett, 2007; Grahn & McAuley, 2009; Wiener et al., 2010). When this initial registration of intervals is disrupted, for example in patients with Parkinson's or Huntington's disease, changes in the rate of responses are found (i.e. in mean IRI) as well as increased variability in timing (Buhusi & Meck, 2005; Grahn & Brett, 2009). It is unlikely that this process is disrupted in children with reading difficulties because they typically are not found to have differences in mean IRI, generally maintaining an appropriate tempo, but with greater variability around the mean as described in Chapters 3 and 8 (Badian & Wolff, 1977; Klipcera et al., 1981; Thomson & Goswami, 2008; Wolff et al., 1984; Wolff, Michel, Ovrut, et al., 1990; Wolff, 2002).

There is more evidence that the disruption in children with reading difficulties may be localised within the components that contribute to the maintenance of synchrony. The registration of feedback (from stimuli or responses) and the prediction/anticipation of future events allow the success of synchronisation or timed unpaced outputs to be judged (Drewing & Aschersleben, 2003). Specifically, the synchrony between current anticipatory models (of future stimuli) are compared to somatosensory inputs through interactions between the motor cortex (that codes "when" events should occur), the primary and association sensory cortices (providing representations of stimuli/tactile feedback about responses) and the cerebellum (providing the comparison between models and events by maintaining a trace of recent events) (Arnal, 2012; Doya, 2000; Mauk et al., 2000; Miall & Reckess, 2002; Tesche & Karhu, 2000). These elements fall within the so-called "automatic" timing network necessary for processing stimuli with intervals in the millisecond range (De Guio et al., 2012; Gibbon et al., 1997; Jantzen et al., 2004; Lewis & Miall, 2003; Mauk & Buonomano, 2004; Meck, 2005; Tesche & Karhu, 2000). It is likely that differences in this automatic timing control system are responsible for the associations found between timekeeper variance and

component reading skills. In considering other evidence for difficulties associated with dyslexia, potential causes of disruption in this system are considered below.

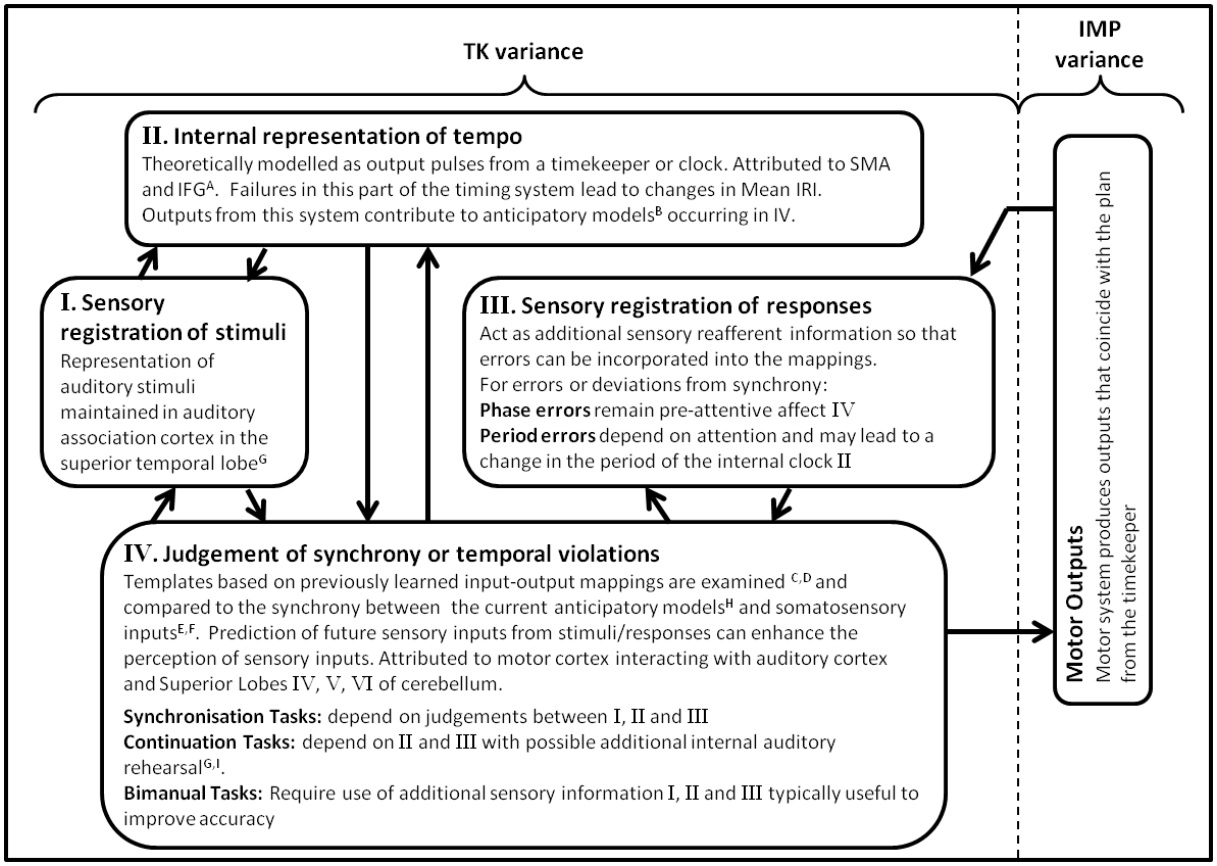

Figure 10.1: Components of the timing system

A representation of the different processes contributing to timekeeper (TK) and implementation (IMP) variance. [A]Grahn & Brett, 2009; [B]Arnal, 2012; [C]Doya, 2000; [D]Mauk et al., 2000; [E]Miall & Reckess, 2002; [F]Tesche & Karhu, 2000; [G]De Guio et al., 2012; [H]Drewing & Aschersleben, 2003; [I]Rao et al., 1997.

In Chapter 6, it was reported that under visually paced synchronisation conditions children with poor reading skills rely less on stimulus driven timing strategy and use the timekeeper to guide synchronisation behaviour, instead focusing on a stimulus-driven strategy. The timing system of these children likely did not judge the timekeeper to be any less accurate under these conditions of temporal uncertainty. Visual stimuli have a longer and more variable latency of transmission to the central processing areas (Brebner & Welford, 1980; Ng & Chan, 2012) and examination of bimodal performance and tasks where jitter has been introduced into pacing stimuli suggest that stimuli giving more reliable or accurate temporal information are normally weighted more heavily and are used to guide behaviour (Elliott et al., 2010; McAuley & Henry, 2010; Recanzone, 2003; Repp & Penel, 2002; Roach et al., 2006). Internal models which use knowledge of the typical correspondences between stimuli and responses allow guidance of future behaviour (Friston, 2005; Lochmann & Deneve, 2011; Roach et al., 2011). In noisy situations (e.g. a noisy room or in visually paced tasks) where the accuracy of stimulus inputs can be obscured, models can be used to select the most appropriate aspects of sensory input to rely on (Roach et al., 2006). If, however, sensory

sources of temporal information or the internal predictions regarding temporal events (based on judgements of synchrony) contain noise then the prediction made will be less temporally accurate. Subsequently, models will contain a greater tolerance of uncertainty for temporal events.

The poor readers may therefore have a greater tolerance for temporal uncertainties in the anticipation and prediction of future stimulus-response correspondences. Such a difference would result in a failure to judge the sensory information received from visual stimuli or from the timekeeper as being irregular or to judge that responses themselves were implemented with a high degree of variability. The reduced correction response in poor readers suggested by results in Chapter 9, if replicated by future research, are similarly indicative of a lack of awareness of changes in the times of occurrences of sensorimotor events or failures to notice errors of synchrony.

Evidence from other temporal processing tasks supports this proposal. For example, children with dyslexia have been found to show impaired detection of simultaneity between two events (Farmer & Klein, 1995; Rosen, 2003) and show impairments across a range of tasks which assess the ability to discriminate small changes in stimuli (Hämäläinen, Salminen, & Leppänen, 2012; Schulte-Körne & Bruder, 2010). There is also evidence for differences in the neural responses to temporal changes in children and adults with dyslexia. For example, neural event related potentials known as mismatch negativities, which should occur following deviant stimuli are delayed or have reduced amplitudes in groups with dyslexia (Hämäläinen, Salminen, et al., 2012; Schulte-Körne & Bruder, 2010). Individuals with dyslexia also have weaker coherence or synchrony between populations of neurons activated by stimuli occurring in rapid succession (Nagarajan et al., 1999).

The proposal of a difference in judging synchrony or making predictions based on temporal information fits well with other hypotheses about temporal processing in dyslexia. For example, it has been suggested that children with dyslexia are unable to make use of repetitions of stimuli that would normally improve predictive models and therefore perception under circumstances of noise (Ahissar, 2007; Hämäläinen, Salminen, et al., 2012). Instead it is suggested that they base each response on current comparisons of sensory events rather than predictions meaning that children with dyslexia do not benefit from learning. Ahissar relates this deficit to the Sluggish Attentional Shifting hypothesis (Ahissar, 2007; Hari & Renvall, 2001) in which typical task performance should be based on repeated predictions that can reduce attentional load, but in dyslexia this does not occur. In terms of phonological development these deficits would affect categorical perception of language elements (e.g. syllables) that particularly depend on temporal processing of prosodic components (Peelle & Davis, 2012; Peelle et al., 2012). Similarly, under Goswami's Temporal Sampling Hypothesis, the difficulties summarised could stem from less well defined temporal reference points due to impaired phase locking of neuronal oscillations to temporal stimuli in dyslexia (Goswami, 2011). Goswami proposes that this is a cause of the deficits in rise time detection and rhythmic

production (i.e. motor timing tasks) as well as those in frequency modulation detection (Talcott et al., 2000; Witton et al., 1998) with consequences for the development of appropriate grapheme-phoneme representations.

Developmentally, it is likely that these differences in temporal processing systems are present from an early age if we assume that they contribute to the development of literacy deficits (rather than being a consequence of literacy deficits). Indeed, there is evidence for infants who are at familial risk of developing dyslexia showing differences in their neural responses to auditory temporal stimuli before language acquisition has occurred (Guttorm et al., 2005; Leppänen et al., 2010; Lyytinen, Guttorm, Huttunen, & Vesterinen, 2005; Plakas, van Zuijen, van Leeuwen, Thomson, & van der Leij, 2012) and that they show differences in processing speech sounds based on temporal characteristics (Richardson, Leppänen, & Leiwo, 2010). Any early disruption in making or using judgements of synchrony would make predictions noisier. Over time this would lead to the proposed higher tolerance of variability in the temporal processing system. The greater variability or impaired phase locking may be related to the disruption of the integrity of communication pathways within the brain in dyslexia as evidenced by diffusion tensor imaging of white matter tracts (Klingberg et al., 2000; Nagy et al., 2004; Steinbrink et al., 2008; Vandermosten, Boets, Wouters, et al., 2012; Vandermosten, Boets, Poelmans, et al., 2012).

Any increased tolerance to uncertainty in children with dyslexia may have greater functional consequences in tasks where multiple temporal events have to be reconciled. For example, the results from this and other studies (Thomson et al., 2006; Thomson & Goswami, 2008) suggest that associations between literacy and timing performance are stronger for performance on paced synchronisation tasks rather than unpaced continuation tasks. In unpaced timing tasks, fewer demands are placed on the temporal control system because there is no need to update responses with respect to externally occurring stimuli, only to deviations between timer outputs and feedback from responses. In comparison, synchronisation tasks where temporal signals from several sources (stimuli, responses and timekeeper) have to be resolved may pose additional problems for children with dyslexia (a conclusion also reached elsewhere Vandermosten et al., 2011).

This discussion demonstrates that the location of the timing difficulty associated with dyslexia could be isolated to a particular area of the timing system. However, it also demonstrates a limitation of the original Wing-Kristofferson model in which elements I-IV of Figure 10.1 all fall under the timekeeper variance component. In future it will be beneficial to apply statistical adaptations which extend the original time series decomposition model and will allow confirmation of the reasons why children with dyslexia fail to synchronise their timing performance appropriately. For example, models exist which can measure: the use of feedback from responses through bimanual synchronisation paradigms and provision of supplementary sensory information (Drewing &

Aschersleben, 2003) or through the inclusion of terms in the Wing-Kristofferson model that account for this use of sensory information (Elliott et al., 2010); the use of prediction or forward models by measuring responses to synchronisation stimuli that are more or less predictable (Acerbi, Wolpert, & Vijayakumar, 2012; Miall & Reckess, 2002; Wolpert et al., 1998) or the degree to which errors are corrected (Repp et al., 2012).

10.3.2. Relationships between timing and components of literacy and attention

Associations between timing performance and behavioural/cognitive indicators associated with dyslexia and ADHD have been examined throughout this series of studies. Temporal processing deficits occurring early in development may have varying affects on different cognitive/behavioural tasks due to the skills encompassed by such tasks. Here, timekeeper variance was significantly associated with rapid object naming ability, spelling performance and digit span performance after controlling for age and IQ. Rapid naming ability in particular was associated with timing performance across the range of interval rates assessed in Chapter 8. This task requires multiple skill elements to be brought together for successful performance, namely fluency, phonological processing, articulation, visual scanning, pacing, memory and planning (Wolf et al., 2000). Poor rapid naming performance in children with dyslexia is usually not accompanied by visual or articulatory deficits and is not typically attributable to the presence of ADHD (Raberger & Wimmer, 2003) (and supported by the regression analyses conducted in Chapter 8). The associations between this factor and timing may therefore reflect impairments in the development of phonological representations that result from temporal processing deficits (although phonological ability is usually dissociable from rapid naming performance; Wolf et al., 2000), or a deficit in rapidly and fluently combining multiple sensory perceptual elements (Raberger & Wimmer, 2003).

In Chapter 9 synchronisation performance was more specifically related to reading fluency but not to general processing speed. The reading fluency measure reflects a number of processes including word decoding (that relies on phonological and orthographic awareness) and the ability to integrate information from multiple internal processes across the reading network (such as symbol activation, word recognition and lexical access) efficiently and automatically (Wolf & Katzir-Cohen, 2001). Therefore, which of these elements are related to timing performance remains unclear. Future research may examine whether the timing deficits in poor readers are particularly related to failures of integration and synchrony across this reading network.

Measures of attentional elements (both subjective ratings of symptoms and objective measures of inhibitory control) were also related to timing performance but were associated with implementation variance rather than timekeeper variance. Rubia and colleagues suggest that an inhibitory control deficit affecting regulation of behaviour may affect the setting up of the representation of stimulus intervals or the output of responses (Rubia, Smith, & Taylor, 2007). Here

we did not measure the association between mean IRI and predictors of ADHD, but other studies have found that children with ADHD are unable to match their IRIs to the target ISI during motor timing tasks (Ben-Pazi et al., 2003, 2006) which is indicative of a failure of the clocking mechanism to set up a representation of the stimulus. The associations between increased implementation variance and a higher number of symptoms of ADHD suggest that there is an additional impairment in implementation of responses later in the timing network (see Figure 10.1). Other investigations indicate that children with ADHD have impairments with the later (attention dependent) evaluation of errors such that they fail to respond to feedback and correct for timing errors (Van De Voorde, Roeyers, & Wiersema, 2010), consistent with the present findings. The associations found between digit span and general timing variability may therefore reflect an overlap between working memory and attention deficits. Any future applications of the motor timing or error correction paradigms will therefore require careful monitoring of attentional factors in order to measure their influence over the quality of timing behaviour, error detection or correction.

In the review presented in Chapter 3, only one previous study was found that had investigated period correction in children with reading difficulties. The poor readers showed slower responses to period changes (like those shown in Figure 9.1) (Wolff, 2002). Although the presence of other learning difficulties was controlled in this study it is feasible that the difficulty in responding to period changes was mediated by sub-clinical symptoms of impulsivity which affected the conscious detection of period changes. As already advocated, such results highlight the need to measure symptoms of ADHD in future studies of motor timing behaviour in children with reading difficulties in order to monitor their supplementary influence on timing performance.

10.3.3. Rate of processing

The correlations described above, between timing variables and cognitive/behavioural predictors, were most consistently present in trials with the 402ms IOIs (2.5Hz), with some associations also being found on longer and shorter (508 and 331ms) IOI trials (2 and 3Hz). The importance of rates in the 2-3Hz range replicates previous results where tapping performance in this range tends to be disrupted in groups of children with dyslexia compared to controls (Badian & Wolff, 1977; Thomson et al., 2006; Thomson & Goswami, 2008; Wolff et al., 1984; Wolff, Michel, Ovrut, et al., 1990; Wolff, 2002), or is found to be associated with literacy ability (Thomson et al., 2006). Some of these studies found group differences at a slower rate of 1.5Hz (Klipcera et al., 1981; Thomson et al., 2006; Wolff et al., 1984; Wolff, 2002), which were not present in this study (although see discussion on maturation in Chapter 8). In the analyses conducted by Thomson and Goswami (2008), performance was collapsed across rates (from 1.5-2.5Hz) making it difficult to assess the importance of specific rates.

Evidence for impairments around a small range of frequencies may point towards specific timing mechanisms that are disrupted in dyslexia. In the context of robust associations with literacy ability, the impact of this specific mechanism would be predicted to affect the development of reading ability (notwithstanding the possibility of reverse causation, namely that disrupted reading acquisition causes the timing difficulty). This line of reasoning follows that of the temporal processing hypothesis (e.g. Tallal, Miller, & Fitch, 1993) in which temporal processing deficits lead to disruptions in processes underlying language and reading acquisition.

Recently, Goswami collated evidence (Luo & Poeppel, 2007; Poeppel et al., 2008; Poeppel, 2003) to establish that the sampling of different temporal rates is carried out by neuronal oscillations of different frequencies (Goswami, 2011). In the context of reading development, Goswami highlights particular oscillatory frequencies that contribute differently to the processing of features of the speech signal. For example, Theta band neuronal oscillations (4-10Hz) are important in coding the syllable rate in speech and Delta frequency oscillations (1.5-4Hz) allow sampling of rates relevant for speech prosody. Phase locking to different rates within the speech signal is important for comprehension of speech (Peelle & Davis, 2012; Peelle et al., 2012) and a failure to phase lock would prevent integration of the acoustic features of language (Goswami, 2011). As in speech processing, neuronal oscillations are important in motor timing. To enable motor outputs to follow predictable millisecond timing stimuli, oscillations in the delta-theta range that are entrained to the stimulus rate are required (Arnal & Giraud, 2012; Nozaradan et al., 2011).

With the motor timing impairment in children with reading difficulties appearing to fall in the 2-3Hz range, this would implicate the lower frequency oscillations in the Delta range which are important for sampling prosodic features of speech. These slow rates of neuronal oscillation do occur across both cerebral hemispheres, but those associated with speech perception tend to be preferentially dominant in the right hemisphere (Poeppel, 2003). That a range of frequencies is implicated in dyslexia is not surprising because the systems that are important for the temporal analysis in speech operate over a range of frequencies to allow analysis of temporal features, like prosody. These features are not static, but vary according to the speaker and acoustic context (Arvaniti, 2009).

Processing of frequencies in the range of 2-3Hz has been implicated in previous investigations of temporal processing in dyslexia. For example, frequency modulation perception has been found to be impaired in children with dyslexia when the frequency modulations occur at a rate of 2Hz, but not at faster rates of 240Hz (Talcott et al., 2000, 2003; Witton et al., 1998). Performance on the slow modulation rates in these tasks is also associated with reading ability (Hulslander et al., 2004; Talcott et al., 2002). As with motor timing and prosodic or syllabic elements of speech, these slower frequencies require temporal analysis rather than the spectral analysis needed to process high

frequencies of the kind required for the analysis of pitch or voicing patterns (Rosen, 1992; Witton et al., 1998).

Infants rely on slow frequency syllabic units earlier than they utilise more rapid phonetic transitions (Poeppel et al., 2008; Telkemeyer et al., 2011). Thus, entrainment to the slow temporal aspects of speech are important from an early age, with the rate of babbling movements and vocalisations also falling into the range of 1-3Hz (Dolata, Davis, & MacNeilage, 2008; Petitto et al., 2001). As described in Chapter 3, perception and production of temporal elements of speech, such as the synchrony between word sounds and articulations, scaffold language development (MacNeilage & Davis, 2001; Scott et al., 2009; Werker & Tees, 1999; Westermann & Miranda, 2004).

In line with this research it may be that this deficit in the 2-3Hz range is present from an early age in children with dyslexia. Inefficiency in the brain systems that process these slow frequency components of the speech signal may contribute early in the language learning process. Genetic differences in chromosome regions that are associated with neuronal myelination and migration (see reviews by Galaburda et al., 2006; Paracchini, Scerri, & Monaco, 2007) and differences in the anisotropy of white matter tracts (efficiency along axons) and density of gray matter in children with dyslexia (Steinbrink et al., 2008) may act as a potential cause of the failures of children with reading difficulties to produce efficient networks controlling functions such as sensorimotor and cognitive processing.

10.4. Methodological considerations and limitations

The error correction task applied in Chapter 9 was used to examine whether participants are aware of small errors in their motor timing performance that are similar to the tap-to-tap synchronisation errors that contribute to variability in timing (higher SD of IRIs). An MRI study indicated that this type of phase correction task was associated with the same areas of the timing network that are used for monitoring spontaneous errors that contribute to variability in timing (Bijsterbosch, Lee, Hunter, et al., 2011). However, Repp et al. (2012) recently modelled responses to both types of errors and proposed that they differ in the manner in which they are corrected. They adapted the Wing-Kristofferson model to incorporate an error correction parameter that estimated the degree to which each timekeeper interval was corrected, based on the asynchrony of current responses. The degree of correction immediately following a phase shift was found to be greater than that following asynchronies generated by ongoing intra-individual variability, although soon after phase shifts the degree of correction returned to a level which matched the correction occurring with spontaneous variability.

Repp and colleagues acknowledge that the most likely mechanism for phase shift correction involves the same calculations as for maintaining synchrony using predictions and expectancies implemented

in forward models. However, they also propose that the phase shifts are corrected by a wholly different (as yet unspecified) mechanism. If future research confirms that the correction of phase shifts proceeds via an entirely different mechanism of control to that responsible for monitoring and reducing spontaneous variability, then there could be two different forms of correction response i.e. phase correction ability and general intra-individual monitoring of synchrony, which could separately be related to the literacy abilities measured here. Future studies of this kind should consider measuring error correction during standard synchronisation performance using supplementary statistical modelling techniques (Repp et al., 2012; Vorberg & Schulze, 2002), in addition to measuring responses to errors in a phase correction paradigm.

Some difficulties with the application of time series models to data from clinical populations or unpractised participants have already been discussed (Chapter 7 and 8, and see Section 10.5.3 below). Whilst the use of such methods are important to advance understanding of timing performance characteristics, it is important to ensure that task parameters are appropriately tailored to the participant group. For example, in the Chapter 9 it appeared that very few of the children had achieved a consistent or stable response asynchrony prior to the occurrence of the phase shift, demonstrating a trend of decreasing asynchrony in the three taps before the shift. To enable correction to be measured in the presence of this trend correction was analysed relative to the asynchrony at the time of the shift (as described by Repp, 2011, Experiment 3A). This factor may have caused phase correction responses to be somewhat moderated by the degree to which participants had already achieved a steady response output. Increasing the number of stimuli occurring before the phase shifts will help to overcome this difficulty. This may extend the lengths of trials, which is undesirable when assessing populations of children, but may be overcome by introducing multiple phase shifts within each trial (as implemented by Repp et al., 2012).

In order to examine the relationships between motor timing and symptoms of dyslexia and ADHD correlation based analyses were applied throughout these studies. With motor timing tasks being conducted over a number of stimulus modalities (Chapter 6) or tapping rates (Chapter 8) and a number of variables being used to operationalise timing performance a large number of statistical comparisons were conducted. This gave rise to a high risk of making Type I errors in the analyses. Where possible the number of comparisons was reduced by using hypothesis driven strategies to select comparisons, for example for the regression analyses in Chapter 8. In these exploratory studies it was deemed appropriate to apply post-hoc corrections to clarify the significance of the associations in light of these multiple comparisons. The need to control for the effects of multiple comparisons has not always been recognised in previous studies in this field that have conducted similar numbers of comparisons (Thomson et al., 2006; Thomson & Goswami, 2008). Presently, following correction a number of the predicted associations remained significant and further examination revealed clusters of statistical relevant relationships between motor timing and

particular aspects of literacy and ADHD. Another approach would have been to minimise the number of comparisons by collapsing performance variables across rates of response, but this would not have revealed the apparent importance of the relationships between the 2.5Hz rate and the literacy and attention variables. Future studies may however seek to conduct a more statistically robust replication of these exploratory analyses by focusing on a single response rate and a narrow range of literacy or attention related variables based on the present results.

A final comment should be made about the relatively narrow focus of this thesis on symptoms of dyslexia and ADHD. This was motivated by the overlap in motor timing difficulties highlighted by the literature review presented in the introduction (Chapter 3) and to ensure it was feasible to measure the symptoms of interest in the relatively short periods of time allotted for testing the clinic and school children. However, difficulties with motor timing (such as increased variability in IRIs) are also found in children with other developmental disorders such as autism, asperger's syndrome, developmental coordination disorder (DCD) and stuttering (Debrabant, Gheysen, Caeyenberghs, Van Waelvelde, & Vingerhoets, 2013; Olander, Smith, & Zelaznik, 2010; Price, Edgell, & Kerns, 2012; Whitall, Chang, Horn, Jung-potter, & Mcmenamin, 2008; Wimpory, 2002). None of the children included in the Clinical sample in the study in Chapter 8 had formal diagnoses of any other disorders and the absence of known diagnoses was specified in the exclusionary criteria for all the samples of school children. Despite this there is the potential that symptoms associated with alternative developmental disorders could have explained the difficulties shown by the poor readers in the present studies.

For example, comorbid symptoms of dyspraxia in the Clinical group in Chapter 8 could have been the cause of the higher numbers of excluded trials in that group where children did not produce complete time series. It is likely however that the timing deficit reported in each of these disorders is associated with changes in different components of the timing system (for example the timing difficulties in autism have been liked to implementation or motor skill problems, Price et al. 2012, and those in DCD may be associated with failures in temporal prediction). Therefore statistical analyses or neuroimaging research similar to that recommended below (Section 10.5) would be beneficial for each of these disorders. In light of the current multi-factorial perspective on developmental disorders in which symptoms are likely to overlap, this issue of controlling for comorbidity amongst cases will remain a challenge and so controlling for or isolating the symptom which explain most variance in timing behaviour will continue to be important.

10.5. Future directions and implications for practice

10.5.1. Assessing the location of timekeeping impairments

The experiments and discussions within this thesis support the proposal that the motor timing difficulty in dyslexia may be attributable to the timekeeping component of the temporal processing system. As shown in Section 10.3.1, careful analysis of different aspects of performance can help to pinpoint more accurately the location of these difficulties within the timing system. Another possibility is to apply neuroimaging measures to these questions. As noted in the introduction (Chapter 2) and in this chapter, analysis using MRI has revealed particular brain areas that are involved in particular processes that contribute to motor timing behaviour and has led to the development of models like that shown in Figure 10.1 in which different brain areas are implicated in different processes that contribute to timed motor responses (Wiener et al., 2010). MRI could therefore be implemented with children with dyslexia whilst conducting synchronisation timing tasks to determine where these children show differences in activation of the timing network, if any compared to controls. Furthermore, imaging methods with higher temporal resolution, such as electroencephalography (EEG) and magnetoencephalography (MEG) have been used to measure the time of occurrence and intensity of components of neural oscillatory activity in particular brain areas that are associated with the presentation of repeated isochronous temporal stimuli. For example, the M100 response in the auditory cortex has been shown to be affected by millisecond variability in timing stimuli (Tecchio et al., 2000) which contributes to judgements of the accuracy of synchronisation behaviour. Similarly, mismatch negativities and the subsequent positive peaks in evoked responses to rhythmic stimuli recorded in the temporal cortex adjacent to the primary auditory cortex are implicated in the anticipation and prediction of future temporal stimuli. Analysing such components in children with dyslexia would substantiate claims that the timing difficulties associated with dyslexia are attributed to any of the various processes that contribute to achieving synchronous motor timing behaviour.

10.5.2. Questions of development and implications for remediation programmes

The literature described above assumes that the temporal processing difficulty arises early in development alongside differences in brain structure and function. Indeed evidence is being amassed which links particular genetic risk loci, responsible for processes occurring very early development of the brain (such as neural organisation and myelination), to developmental dyslexia (Galaburda et al., 2006; Meng et al., 2005; Paracchini et al., 2006). In light of the need to understand how temporal processing deficits influence language development early in infancy, to better target educational remediation strategies, further research with infant populations is required. Some studies have already successfully measured aspects of temporal processing in very young children using imaging methods and found, for example, that components of neural oscillatory responses to

language stimuli predict later literacy development (Guttorm et al., 2005; Leppänen et al., 2010; Lyytinen et al., 2005; Plakas et al., 2012; Richardson et al., 2010). Repetitive isochronous stimuli of the type used in motor timing tasks are particularly useful for assessing temporal processing in young children because they automatically generate event-related potentials or oscillatory activity in the brain, even in the absence of movements. When coupled with non-invasive imaging techniques (e.g. MEG) to measure the characteristics of this activity in infant populations we will be able to assess questions of how and when timing difficulties arise and how they contribute to the stages of language development.

Such research needs to be carried out to establish whether aspects of temporal processing scaffold language development. For example whether the development of the ability to anticipate and predict repeated events in time (e.g. in the motor system; Arnal, 2012) occurs in tandem with the development of components of language which rely on temporal cues, such as for decoding speech using prosody. Understanding precisely which aspects of motor timing are associated with language development, or when in development these forms of processing are important, will also help to develop appropriate targets for remediation. At present, some remediation programmes exist which are based on the assumption that temporal processing scaffolds language development (Besson, Chobert, & Marie, 2011; Bhide, Power, & Goswami, 2013; Gaab, Gabrieli, Deutsch, Tallal, & Temple, 2007; Patel, 2011; Thomson et al., 2013). Such interventions train children using rhythmic stimuli and measure outcomes in relation to reading or reading component skills. However, these programmes are applied without full understanding of which components of temporal processing are being affected by the training, if at all. Programmes which have found literacy skills and phonological awareness improve following such interventions may simply be measuring compensatory phenomenon in which children learn additional strategies to support literacy but where any temporal processing difficulty is not altered.

Research suggests that timing based training could be beneficial, with evidence from studies with adults indicating that the perception of synchrony can be altered adaptively over time by presenting quasi-isochronous stimuli in which the IOI lag is manipulated at a millisecond level (Vroomen & Keetels, 2010). Similarly, musical training (as a musician or within experimental settings) can improve the accuracy and reduce the variability of synchronisation and improve error correction (Bailey & Penhune, 2010; J. L. Chen et al., 2008; Zatorre, Chen, & Penhune, 2007). Musical training also leads to functional changes in the brain, in particular, recruiting areas of the timing network more efficiently (Gaab et al., 2005; Jäncke, Shah, & Peters, 2000; Koeneke, Kai, Wustenberg, & Jancke, 2004). However, in applying such training programmes to children, researchers should be cautious to assess whether the programme effects functional changes in temporal processing across musical and non-musical domains or whether such programmes simply work to improve cognitive factors such as working memory, attention or processing speed. Such factors could be improved as

effectively by any one of several non-musical/non-temporal remediation strategies with similar impacts on literacy development. Intervention studies should therefore focus on targeting a single hypothesis driven aspect of temporal processing (e.g. detecting temporal errors) rather targeting the many areas of processing that are often included in these intervention programmes to make claims about the success of "rhythmic" training (e.g. Besson et al., 2011). Such all encompassing remediation programmes may show significant effects on literacy but they present challenges in teasing apart the relative contribution of individual aspects of temporal training that may lead to the creation of more specific targeted remediation strategies in future.

10.5.3. Application of time series models

The research conducted within this thesis has demonstrated the benefits of applying time series analysis models, such as the Wing-Kristofferson model, to questions of motor timing in children with developmental disorders. The use of this type of model allows variability in timing (typically only operationalised simply as the standard deviation of inter-response intervals) to be decomposed into more discrete elements such as timekeeper and implementation variance. As described in Chapter 2, these models have been variously applied in investigations of other clinical populations such as patients with damage to the basal ganglia (Freeman et al., 1996; Harrington et al., 1998), cerebellum (Bolbecker et al., 2011; Harrington et al., 2004; Ivry & Keele, 1989), or more pervasive disruption throughout circuits associated with time based processing (Carroll et al., 2009; Kooistra et al., 1997; Simmons et al., 2009; S. J. Wilson et al., 2002). Application of the models in disorders where brain damage is relatively discrete has allowed neural systems to be identified which appear to be responsible for putative elements of the timekeeping system that are derived from statistical analyses. Similar application here has allowed a greater focus on the elements of the timekeeping system that may be responsible for the motor timing impairments seen in children with reading difficulties.

Models, such as the Wing-Kristofferson model, were initially derived from time series data drawn from well practiced adult participants who were considered to exhibit very stable or at least typical timing behaviour. Applying such models to data from participants from clinical samples where time series do not always mirror the statistical properties of typical behaviour creates some challenges for analysis because such participants may show instances of closed loop timing or non-stationarity which are not predicted by the model. However, monitoring and reporting these inconsistencies can in fact provide additional understanding of the differences in timing behaviour demonstrated by clinical populations as demonstrated in Chapter 8. In addition, such research can help to constrain the models themselves by demonstrating instances of behaviour that need to be better explained by timing models. Indeed, research employing these tools (Beek, Peper, & Daffertshofer, 2000; Kooistra et al., 1997; Madison, 2001; O'Boyle et al., 1996; and the analyses and discussions presented in

Chapter 2 and 7) indicate that providing inconsistencies are reported, data from clinical populations can provide sufficient fit to the models. As such, the application of these models provides substantial advantages in understanding the nature of timing behaviour that their application in this type of timing study continues to be warranted.

Notwithstanding the inability of the model to distinguish components of the timekeeping system beyond the two variance components (as discussed above), the studies here were successful as a first attempt to apply these linear models to data from developmental populations in relation to reading and attention. The identification of basic subcomponents in the timing system that may be responsible for the deficits seen in dyslexia and ADHD allow future investigations to expand the time series analysis further to explore additional processing elements. In future, non-linear, dynamic systems models may eventually be sufficiently well operationalised to allow their successful application to small samples of data from clinical populations. Currently, the many thousands of responses required for model fitting under dynamic systems approaches means that they are impractical to employ. Based on the statistical properties of a large number of biological processes, these dynamic models can provide greater detail about the statistical properties of behaviour. However, there are indications that both linear and dynamic models predict variance structures that are similar enough that linear models can provide a good-enough reflection of the complexities of human timing behaviour for current requirements with clinical populations (Pressing, 1999; Wing & Beek, 2002).

10.6. Conclusion

This is the first study to apply time series models to the analysis of motor timing behaviour deficits in children with reading difficulties. The experiments conducted demonstrate the utility of such statistical tools in going beyond simple measures of performance variability.

Differences were demonstrated between visual and acoustically paced timing tasks, suggesting that visual tasks may not assess the true capacities of timekeeping systems, but rather the ability to use feedback from stimuli under conditions of temporal uncertainty. These findings indicate that future investigations of timing in developmental disorders should implement auditory tasks to ensure that variability is reflective of the timekeeping systems.

Differences were also found in the manner in which children with poor reading and poor attention skills completed timing tasks. This is despite the apparent overlap in motor timing deficits in children with dyslexia and ADHD. The evidence was summarised to suggest that children with reading difficulties have more variability in their timing system, perhaps as a consequence of impairments in the recognition of synchrony, causing them to rely less heavily on the timekeeping systems that normally use anticipation, prediction and judgements of synchrony to complete motor timing tasks.

The final study provided a useful preliminary examination of how phase correction may contribute to motor timing deficits and how this task may be beneficial in examining the functioning of these timekeeping systems in future.

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Appendices

Appendix A: Terminology

A.1. Nomenclature

Term	Definition
β	Drift coefficient
σ	Variance
$E[\hat{\gamma}(j)]$	Expected value of the autocovariance estimate at a lag of j
$f'(k)$	Function of a plot of the median of differences at each lag (Δ_j) against lag number
$\gamma(j)$	Estimate of autocovariance at a lag of j
$\gamma_I(k)$	Auto covariance function
$\gamma_I(1)$	Lag one covariance
$\gamma_I(0)$	Lag zero covariance
I	Inter-response interval
j	Response / interval number
k	Lag
μ	Mean
P	Peripheral implementation delay
$\rho_I(k)$	Autocorrelation
$-0.5 < \rho_I(1) < 0$	The range of valid values of $\rho_I(1)$ for a linear lag one autocorrelation time series model
Σ	Sum of
T	Timekeeper (sometimes referred to as “clock”)
Δ	Change in or difference

A.2. Glossary

Term	Definition
ADHD	Attention Deficit Hyperactivity Disorder
Anisochronous	Intervals occurring with an irregular beat, not occupying equal time
Continuation Task	Following a synchronisation phase (see Synchronisation Task), the stimulus is removed and participants must attempt to produce finger tap responses unpaced but at the same tempo as in the synchronisation phase
CV	Coefficient of variation
Deterministic	Predicts future values exactly (compare to Stochastic)
IOI	Inter-onset interval, the time between the onsets of two stimuli
IRI	Inter response interval, mean IRI calculated as $\mu_I = \sum_{j=1}^{N} I_j \Big/ N$
Isochronous	Intervals occurring with a regular beat or occupying equal time; in reference to beats occurring at equal intervals over time
n.s.	Non-significant
Period Shift	Used in paradigms assessing error correction performance: the alteration of mean IOI for all subsequent stimuli such that a new stimulus rate is established.
Phase Shift	Used in paradigms assessing error correction performance: the alteration of the onset time of a single stimulus such that a single IOI is lengthened and subsequent stimuli occur at the original stimulus rate
SEM	Standard error of the mean
SD	Standard deviation
Stationarity	Where the covariance of a time series depends only on the lag, not on time
Stochastic	Uses a probability distribution to determine future values (compare to Deterministic)
Synchronisation Task	A motor timing task in which participants must attempt to produce finger tap responses in time to the stimulus beat, i.e. a paced task
$-0.5 < \rho_I(1) < 0$	The range of valid values of $\rho_I(1)$ that create a valid trial

A.3. Inter-onset interval reference guide

Historically, studies have historically referred to tapping rates in Hertz (i.e. cycles per second) or beats per minute (bpm) although the current convention is to describe the inter-stimulus intervals (or onsets) in milliseconds. A guide is provided here for some of the interval sizes mentioned in the thesis for ease of conversion between the two terms.

milliseconds	Hz	bpm
1000	1.00	60
833	1.20	72
670	1.49	89
666	1.50	90
650	1.53	92
505	1.98	119
500	2.00	120
400	2.50	150
333	3.00	180
329	3.03	182
285	3.50	210
282	3.54	212
250	4.00	240

A.4. List of equations

Equation Number	Equation
Equation 1	$I_j = T_j - P_{j-1} + P_j$
Equation 2	$\gamma_I(1) = \frac{\sum_{j=2}^{N}(I_j - \mu_I)(I_{j-1} - \mu_I)}{N-1}$
Equation 3	$\gamma_I(1) = -\sigma_P^2$
Equation 4	$\gamma_I(0) = \frac{\sum_{j=1}^{N}(I_j - \mu_I)^2}{N}$
Equation 5	$\gamma_I(0) = \sigma_T^2 + 2\sigma_P^2$
Equation 6	$\sigma_T^2 = \gamma_I(0) + 2\gamma_I(1)$
Equation 7	$\rho_I(k) = \gamma_I(k) \big/ \gamma_I(0)$

Equation Number	Equation
Equation 8	$\rho_I(1) = -\,1/2+\left(\sigma_T^2/\sigma_P^2\right)$
Equation 9	$I_j = T_j - P_{j-1} + P_j + \beta_j$
Equation 10	$\hat{\sigma}_P^{\,2} = \frac{1}{a_{\gamma_I(1)n} b_{\gamma_I(0)n} - b_{\gamma_I(1)n}} \left(-b_{\gamma_I(1)n}\gamma_I(0) + b_{\gamma_I(0)n}\gamma_I(1)\right)$
Equation 11	$\hat{\sigma}_T^{\,2} = \frac{1}{a_{\gamma_I(1)n} b_{\gamma_I(0)n} - b_{\gamma_I(1)n}} \left(a_{\gamma_I(1)n}\gamma_I(0) - \gamma_I(1)\right)$
Equation 12	$E[\hat{\gamma}(j)] = -\left(1/N\right)\gamma(0) - \left(2/N\right)\left\{1 - j/[N(N-1)]\right\}\gamma(1)$
Equation 13	$\Delta I_j = \left\| I_j - I_{j+1} \right\|$

A.5. Additional reference equations

Description	Equation
Total Inter-response time	$\bar{I} = \sum_{j=1}^{n} I_j$
Estimate of drift	$\hat{\beta} = \frac{1}{S_n} \sum_{j=1}^{n} \tilde{j}\, I_j$
Lag zero covariance including drift	$\gamma_I(0) = \frac{1}{n-2} \sum_{j=1}^{n} \left(I_j - \bar{I} - \hat{\beta}\,\tilde{j} \right)^2$
Lag one covariance including drift	$\gamma_I(1) = \frac{1}{n-1} \sum_{j=1}^{n-1} \left(I_j - \overline{I_{(1)}} - \hat{\beta}\,\check{j} \right)\left(I_{j+1} - \overline{I_{(2)}} - \hat{\beta}\,\check{j} \right)$
Corrected interval number	$\tilde{j} = j - \frac{n+1}{2}$
Corrected Kooistra term	$S_n = \sum_{j=1}^{n} \left(\tilde{j} \right)^2 = \frac{n\left(n^2-1\right)}{12}$
Corrected Kooistra term	$\overline{I_{(1)}} = \frac{1}{n-1} \sum_{j=1}^{n-1} I_j$
Corrected Kooistra term	$\overline{I_{(2)}} = \frac{1}{n-1} \sum_{j=2}^{n} I_j$
Corrected Kooistra term	$\check{j} = j - \frac{n}{2}$
Estimated total Inter-response time	$E\left\{\bar{I}\right\} = \mu + \beta \frac{n+1}{2}$
Estimated drift	$E\left\{\hat{\beta}\right\} = \beta$
Estimated Lag zero covariance including drift	$E\{\gamma_I(0)\} = \sigma_T^2 + \sigma_P^2 \left(2 + \frac{3n+5}{n(n+1)} \right)$

Description	Equation
Estimated Lag one covariance including drift	$E\{\gamma_I(1)\} = \frac{1}{n-1}\left(-2+\frac{7n-5}{n^2-1}\right)\sigma_T^2 - \left(1-\frac{3(n+3)}{n(n-1)^2(n+1)}\right)\sigma_P^2$
Kooistra model term	$b_{\gamma_I(0)n} = 2+\frac{3n+5}{n(n+1)}$
Kooistra model term	$a_{\gamma_I(1)n} = \frac{1}{n-1}\left(-2+\frac{7n-5}{n^2-1}\right)$
Kooistra model term	$b_{\gamma_I(1)n} = -1+\frac{3(n+3)}{n(n-1)^2(n+1)}$

Appendix B: Published works

B.1. Published articles

Birkett, E. E. & Talcott, J. B. (2012). Interval Timing in Children: Effects of Auditory and Visual Pacing Stimuli and Relationships with Reading and Attention Variables. *PLoS ONE,* 7, 8, e42820

Talcott, J. & Birkett, E. (2009). Dyslexia from a cognitive neuroscience perspective: making links and moving forward. In: Brunswick N. & Lawson, R. (Eds.). The Dyslexia Handbook 2009/10 (pp. 151-160). Bracknell: BDA.

B.2. Conference abstracts

Aston University Postgraduate Research Day (June, 2010)

Reading Clocks: Investigating the relationship between millisecond movement timing, reading and attention.

E. E. Birkett & J. B. Talcott

This study explores the relationship between timing, literacy ability and attention in unselected groups of adults and children. Deficits in temporal processing have been identified in several developmental disorders including dyslexia and ADHD and may be a feature of the co-morbidity between such disorders. A finger tapping task in which participants synchronised to a beat then continued to tap unsynchronised was employed to assess motor timing. This task was presented at different tapping rates (1.5, 2, 2.5, 3, 3.5 Hz) and allowed timing accuracy to be measured. This was then decomposed into timekeeper and afferent variance using a linear, information processing approach to timing. The participants also completed a full battery of literacy and psychometric measures. Results indicate that different components of timed performance relate separately to literacy and attention measures. The utility of such temporal processing tasks in predicting literacy skills is discussed, together with a summary of ongoing research being conducted with clinical participants. This project is part of a PhD supported by an Open Competition Studentship from the ESRC.

Society for Neurosciences (September 2010)

Stimulus Parameters of Finger Tapping Tasks & Developmental Disorders: effects on performance and relations to cognitive skills.

Emma E. Birkett, Joel B. Talcott, Kim S. H. Rochelle

This study explores the use of finger tapping tasks as indices of temporal processing in dyslexia and Attention Deficit Hyperactivity Disorder (ADHD). Deficits in temporal processing have been identified in several developmental disorders and may be a feature of co-morbidity between such disorders. Previous studies measuring timing in dyslexia and ADHD have employed finger tapping tasks incorporating either auditory or visual stimuli respectively. This study aims to bring together these different task parameters in order to clarify the effects of stimulus modality on performance and examine how performance each task relates separately to the phenotypes of dyslexia and ADHD.

The participants consisted of both clinical and unselected groups of adults and children, with and without developmental disorders, who each completed a full battery of literacy and psychometric measures. A 3Hz synchronisation finger tapping task was presented in three modalities (auditory, visual and bimodal). Inter-response intervals (IRI) and stimulus-response asynchronies were recorded and responses were decomposed into timekeeper and afferent variance using a linear, information processing approach to timing (Wing, A. M. & Kristofferson, A. B. (1973). Perception & Psychophysics, 14(1), 5-12). We found that with visual stimuli there was greater within-individual variation in IRI and asynchrony compared to the other modes and that this variation appears to result from greater variability in the timekeeper mechanism. In addition, different stimulus modalities were each found to be significant predictors of different skills that are affected in developmental disorders: reading, spelling and attention. Together these results indicate that finger tapping to stimuli of different modalities are not directly comparable and that deficits in temporal processing found in dyslexia and ADHD using these tasks do not necessarily relate to the same functional or cognitive construct. Suggestions for appropriate task selection are highlighted.

See the Beat, Hear the Beat: Effects of Stimulus Mode in Synchronisation Timing Tasks

Emma E. Birkett & Joel B. Talcott

Motor timing ability in developmental disorders has been studied using synchronisation tasks with stimuli presented in different sensory modalities (auditory, visual or combined auditory-visual). Here, we examine the nature of motor timing behaviour under different stimulus conditions. We aim to replicate findings of increased variability in the visual task in adults and examine whether the same performance characteristics are evident in children. Twenty-seven adults and twenty-one children completed a motor timing synchronisation task, which required finger tapping to a beat occurring at 330 ms intervals. The beat was presented via the three modes: auditory, visual or combined auditory-visual. Inter-response intervals (IRI) and asynchronies to the beat were recorded over three trials within each mode. Participants also completed measures of cognitive and literacy ability and attention. Children showed greater IRI variability than adults as well as larger asynchronies between their taps and the beat, and greater asynchrony variability. Both groups also demonstrated larger and more variable asynchronies on the visual task compared to the other modes. When an information processing model of timing was applied to the data it showed that in comparison to adults, the children had both greater timekeeper variance and greater motor-implementation variance. Furthermore, both groups showed greater timekeeper variance but reduced motor variance in the visual modality compared to the other modes. The development of timing over the lifespan and its relationship with cognitive and behavioural variables is discussed. A commentary about the value of these tasks in developmental research is also provided.

British Dyslexia Association International Conference Symposium, Understanding comorbidity between dyslexia and other developmental disorders (June, 2011)

The association between reading, attention and timing performance in a time-reproduction paradigm.

Emma E. Birkett

Studies have demonstrated differences in accuracy and variability of motor timing in participants with dyslexia and ADHD. Here a time reproduction, finger-tapping continuation task, was administered alongside measures of attention and literacy to adults and children (with and without dyslexia). Measures of timing were contrasted with measures of literacy and attention to establish whether different components of the timing response were separately related to reading and attention. The data will be discussed with reference to the proposal that deficits in processing stimuli of a temporal nature may form a 'domain-general' deficit underlying the developmental disorders that commonly overlap in individuals.

Aston Research Centre for Healthy Ageing (ARCHA) Showcase Event (September, 2011)

Development of Temporal Control in Synchronised Coordination: A Comparison Between Children and Young Adults

Emma E. Birkett, Kim S. H. Rochelle & Joel B. Talcott

Purpose: Synchronised coordination tasks have been used to establish the effects of ageing on temporal control in adult populations. Some results have indicated that beyond the early twenties there are only minor changes in the mechanisms governing temporal control, even late in life. Here we examined temporal control in children and young adults to provide evidence of how these functions develop across the early years. An information processing model of timing was applied to the data to determine whether any group differences were attributable to timekeeping or implementation systems.

Method: Two groups of children (aged 8-9 and 10-11) and a group of adults (aged 18-24) completed a finger tapping task. They were instructed to tap in time to an auditory beat, and after the beat stopped, to carry on tapping at the same speed. Trials were presented at five stimulus rates (670, 505, 400, 329 and 282 ms) and inter-response intervals (IRIs) in the continuation phase were recorded.

Results: As expected, performance was related to the stimulus rate, with IRIs and variability of responses decreasing at higher tapping speeds. There was also a group effect, with less variability being found in the older children and young adults than the younger children. A pronounced group effect was also found in timekeeper variance indicating that the younger children have more variance in their timekeeping systems which reduces during development.

Discussion: The data provide evidence of how temporal control changes over childhood and validates the use of this method for use with younger populations. The work also provides baseline data with which temporal control data from clinical populations can be compared. Further work to explore a larger range of age groups throughout childhood and adolescence may be useful to complement existing data regarding changes in temporal coordination systems over the lifespan.

Third British Psychological Society Seminar on Language & Music (Communicative interaction, Literacy & Expertise), Middlesex University (September, 2011)

Motor Timing in Adults and Children - Relationships with Reading and Attention.

Emma E. Birkett

Impairments in motor timing have been found in clinical populations including groups with dyslexia and ADHD. Here we examined how motor timing differs in adults and children, and whether it is associated with cognitive/behavioural measures of interest. Thirty-five adults, 49 school children and 18 children from a clinical group completed a finger tapping task and measures of literacy, attention and reasoning. The tapping task comprised trials at five speeds (intervals of 670, 505, 400, 329, and 282ms). Participants tapped to an auditory beat in the synchronisation phase and maintained their responses in the continuation phase (without the beat). Response intervals were recorded and response variance was also decomposed using an information processing model of timing. Timing performance in adults was more accurate and less variable than in children with group differences found in both timekeeper and implementation variance. The clinical group of children did not differ from the non-clinical children on any of the measures at any tapping speed. Correlational analyses indicated that motor timing performance in children was related to spelling, hyperactivity-impulsivity and verbal reasoning, however, only hyperactivity-impulsivity predicted unique variance in timing ability. The utility of this approach to measuring temporal processing in developmental disorders is discussed.

New Bulgarian University, Erasmus Intensive Programme: Language, Cognition and Developmental Disorders (October, 2011)

Motor Timing in Adults and Children: Relationships with Reading and Attention

Emma E. Birkett

Emma Birkett, from Aston University, presented an overview of her work examining the relationships between fine motor timing and literacy in groups of children and adults. The project investigates motor timing as an index of temporal processing in relation to dyslexia and ADHD. The talk included an introduction to the use of synchronisation and continuation finger tapping tasks in measuring motor timing ability and the associated statistical analyses applied. The cross-sectional research replicates previous findings of gradually improving motor timing during childhood, with changes in timekeeping variance between ages 8 and 10. Results also indicated that timing accuracy relates to symptoms of inattention, whereas decomposed timing components are strongly associated with reading ability. The group discussed how timing functions may be related to performance on processing speed tasks and to the development of language skills early in life.

Discerning the timing problem in children with reading difficulties

Emma E. Birkett & Joel B. Talcott

Aims: Evidence suggests that motor timing is one component of temporal processing that is affected in dyslexia. Difficulties with motor timing have been found at different tempos in children with literacy problems or other developmental disorders such as Attention Deficit Hyperactivity Disorder. Here we assessed variability in motor timing performance in children at five different speeds using a synchronise-and-continue finger tapping task.

Methods: A clinical sample of 16 children aged 8-12 with literacy difficulties participated, alongside two control groups matched for reading-age and chronological-age. All participants completed the finger tapping task and measures of literacy, attention and cognitive ability.

Results: The time series data from the children was analysed for the presence of global drift and local variability. The presence of drift in the time series data precluded the use of linear timing models for this analysis. Group-wise comparisons of response variability did not support our prediction of increased variability in the clinical group. Correlation analyses, collapsed across groups, showed a moderate association between local variability at the 2.5Hz tempo and spelling performance in children.

Conclusions: This evidence provides support for motor timing as a continuous population variable that is associated with more global literacy skills such as spelling.

Motor timing error correction in good and poor readers

Emma E. Birkett, Joel B. Talcott

Performance variability on motor timing tasks tends to be higher in children with reading difficulties and also predicts variance in literacy skills across the population. This experiment investigates whether variability in motor timing is associated with failures in error correction. If error correction is related to performance variability, differences are expected in the error compensation function in good compared to poor readers. School children aged between nine and ten were assessed with measures of reading efficiency, phonological processing and orthographic processing. The participants were then divided into groups of good and poor readers. A synchronise-and-continue finger tapping task provided measures of performance variability across the groups with the aim of replicating greater variability in the poor-reader group. Participants also completed the error correction paradigm comprising an isochronous finger tapping task with inter-stimulus intervals of 500ms. Each trial contained a single 12-ms phase shift in the stimulus sequence which was below the level of conscious detection but produced a compensation response. This is the first experiment to use this method of error correction with children and the task provides another level of analysis of the timing difficulties shown in poor readers. The results allow comparison of error correction compensation functions across the groups of children. The results influence the conclusions that can be drawn from previous studies that demonstrated increased motor timing variability in poor readers.