PROJECT REPORT

MORPHOSYNTACTIC PROCESSING IN DYSLEXIA: APPLICATION OF AN ERP MEASURE

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CHAPTER 1: INTRODUCTION

Dyslexia is a developmental disorder of reading that occurs in persons with otherwise normal intelligence, sensory acuity and general motivation (World Health Organization, 1993). Approximately 5–18% of the population are affected by dyslexia (Shaywitz, 1998; Snowling, 2000). Dyslexia can be defined as an unexpected difficulty in learning to read and the other associated problems include difficulties with writing, spelling, motor co-ordination and attentional abilities, which vary across individuals. The National Institute of Neurological Disorders and Stroke gives the following definition for dyslexia: "Dyslexia is a brain-based type of learning disability that specifically impairs a person's ability to read. These individuals typically read at levels significantly lower than expected despite having normal intelligence.

Although the disorder varies from person to person, common characteristics among people with dyslexia are difficulty with spelling, phonological processing (the manipulation of sounds), and/or rapid visual-verbal responding". There are a number of theories on the potential causes of dyslexia. Literature review supports the fact that some children with developmental dyslexia (DD) show problems in linguistic domain along with reading difficulties. Among the linguistic sphere, phonology has been largely studied with many supporting the role of phonological deficits in reading impairments (Ramus et al., 2003; Snowling, 2000). The other aspects in the linguistic sphere have received relatively lesser attention.

Few studies that have focused on linguistic difficulties have revealed that difficulties in children with dyslexia are not restricted to only written language but also to spoken language. These difficulties have been reported in comprehension and/or production of complex sentence constructions such as relative clauses or passive

sentences, in both children and adults with dyslexia (e.g., Barshalom, Crain, & Shankweiler, 1993; Robertson & Joanisse, 2010; Waltzman& Cairns, 2000; Wiseheart, Altmann, Park, & Lombardino, 2009).Other studies revealed lack of sensitivity to subject-verb agreement morphology (Jiménez et al., 2004; Rispens, Roeleven, &Koster, 2004), impaired inflectional morphology (Altmann, Lombardino, &Puranik, 2008; Joanisse, Manis, Keating, & Seidenberg, 2000) as well as weakness in morphological awareness tasks (Leikin & Hagit, 2006).

In addition, (morpho) syntactic skills have been investigated also in children at risk for DD, generally showing linguistic delays (Bar-Shalom, Crain & Shankweiler, 1993; Sevcenco, Avram, & Stoicescu, 2014; Arosio, Panzeri, Molteni, Magazù, & Guasti, 2017; Cardinaletti & Volpato, 2015). These studies, mostly conducted on preschool children who haven't started formal acquisition of literacy are particularly important, since they suggest that it is not the lack of exposure to printed text that hampers language development. Moreover, the fact that DD is often associated with Specific Language Impairment (SLI) further strengthens the idea that the linguistic impairment in DD might go beyond decoding written language. In the light of the broad literature on the comorbidity and overlap between the two disorders (Bishop & Snowling, 2004), the investigation of the morphosyntactic and syntactic domains in DD is particularly important, given that some morphosyntactic properties are generally impaired in children with SLI.

Research in the area of dyslexia has revealed enough evidence that a sub-group of children with DD or learning disability show some form of underlying linguistic deficits leading to reading difficulties (Adams, 1990; Snowling, 1987; Stanovich, 1986a; Vellutino, 1979; Wagner & Torgesen, 1987). These linguistic deficits may be at the phonological/ morphological/ morphosyntactic levels of processing. However, the focus

on understanding whether these deficits are at a perceptual level (Mann, 1984; Mann & Ditunno, 1990; Stuart & Coltheart, 1988; Lefly & Pennington, 1996), awareness level (Tunmer et al., 1987) or cognitive level has been attempted through offline behavioral tasks. Behavioral tasks only reveal the end performance of the child and these tasks may not help in understanding various processes. This can be accomplished through real time online tasks such as event related potentials (ERPs). The ERP components indicate the point in time at which some variable exerts its effect on information processing. It has been found that areas of brain specialization can be identified by observation of variations on amplitude and latency of ERP components across different scalp locations (Halgren, 1990).Also, a vast amount of information can be collected in one experimental session, giving insight into the sensitivity to incoming linguistic information (for instance, recognition of a grammatical violation).

According to a few neurocognitive models (Friederici, 2002), a biphasic electrophysiological pattern (LAN/P600) is normally expected in response to syntactic violations. Differences in these electrophysiological components have not been reported frequently in dyslexia (Leikin, 2002; Rispens, Been, &Zwarts, 2006, Sabisch, Hahne, Glass, von Suchodoletz, & Friederici, 2006; Russeler, Becker, Johannes, & Münte, 2007). Sabisch et al. (2006) reported that for the syntactic violation, control children demonstrated a combined pattern, consisting of an early starting bilaterally distributed anterior negativity and late centro-parietal positivity (P600). Children with dyslexia showed a different pattern that is characterized by a delayed left lateralized anterior negativity (ELAN) followed by a P600. Rispens et al. (2006) investigated the presence and latency of the P600 component in response to subject-verb agreement violations in Dutch-speaking adults with DD. Despite the absence of differences between adults with dyslexia and control participants in judging the grammaticality of the sentences, their

ERP data revealed subtle differences between groups, particularly related to latency (the P600 tended to peak later in the dyslexia group compared to the control group).

Need for the study

According to various studies conducted across the world, differences in the electrophysiological components such as P600 and ELAN have been infrequently reported in persons with dyslexia (Leikin, 2002; Rispens, Been, &Zwarts, 2006, Sabisch, Hahne, Glass, von Suchodoletz, & Friederici, 2006; Russeler, Becker, Johannes, & Münte, 2007). Attempts have also been made compare language comprehension on the behavioral tests for linguistic skills versus the electrophysiological tests. In this direction, Rispens et al. (2006) investigated the presence and latency of the P600 component in response to subject-verb agreement violations in Dutch-speaking adults with DD. They reported that the ERP data revealed subtle differences between the groups, particularly the P600 latency, while there was no difference reported in grammaticality judgment of the sentences. A study by Sabisch et al. (2006) indicated a similar P600 in controls, children with dyslexia and SLI groups. However, instead of the early starting LAN shown by control children in response to syntactic violations, children with dyslexia were reported to show a delayed LAN (300-600 ms) that was even more delayed in the SLI group (700-1000 ms).

There are no studies conducted in the Indian context with respect to ERPs at the level of sentence processing in children with developmental dyslexia. Though there is ample evidence in terms of behavioral research in the area of language comprehension in children with dyslexia, little is known about the processing issues through ERPs.

The previous investigations focused on investigating the sentence processing deficit in children with DD in languages such as English, Dutch and German. These languages involve a different sentence structure and semantic-syntactic processing may

vary the language that has been processed. The children in the Indian context are often exposed to more than two or more languages in schools as part of their academic curriculum. Understanding the influence of sentence processing in a different language such as Kannada will help us explore the language processing and its effect on reading and writing skills. Therefore, it is important to understand the level of syntactic processing in children with DD in a different language such as Kannada which follows a different word order in sentences when compared to other languages such as English. In Kannada the word order follows Subject-Object-Verb (SOV) whereas in English it is predominantly Subject-Verb-Object (SVO). This can be facilitated better through objective measures such as ERPs- ELAN, and P600 measures.

Aim of the Study

The aim of the present study was to compare the behavioral correlates with event related potential (ERP) correlates of implicit morpho-syntactic processing in children with DD(DD) in comparison to typically developing children (TDC).

Specific Objectives

- To study the behavioral correlates of implicit morpho- syntactic processing in Kannada in children with DD and typically developing children.
- To study the event-related potential (ERP) correlates of implicit morpho- syntactic processing in Kannada in children with DD and typically developing children.
- To compare the behavioral correlates and the event-related potential (ERP) correlates of implicit morpho-syntactic processing in Kannada in children with DD and typically developing children.

CHAPTER 2: REVIEW OF LITERATURE

Developmental Dyslexia is a "learning disability which initially shows itself by difficulty in learning to read and later by erratic spelling and lack of facility in manipulating written as opposed to spoken words. The condition is cognitive in essence and usually genetically determined. It is not due to intellectual adequacy, or to lack of socio-cultural opportunity, or to emotional factors, or to any known structural brain defect. It probably represents a specific maturational defect, which tends to lessen as child gets older and is capable of considerable improvement, especially when appropriate remedial help is afforded at the earliest opportunity" as defined by the World Federation of Neurology (cited in Critchley, 1978).

Learning disability/ Dyslexia and SLI

Researchers suggest that dyslexia and SLI exist on a continuum of language disorder (Goulandris, Snowling & Walker, 2000; Catts, 1995; Stackhouse & Wells, 2001). Within such a continuum, dyslexia is regarded as a form of language impairment affecting primarily the phonological system. Additionally, dyslexia is seen as the lasting result of the disorder provided oral language difficulties disappear. The two syndromes are accordingly treated as different manifestations of the same underlying problem, differing only in severity and in the stage when the disorder manifests itself (i.e. SLI is considered the more severe manifestation of the disorder, appearing in early childhood. Dyslexia is considered to be the less severe manifestation of the disorder, appearing later in childhood).

Past literature on relationship between dyslexia and SLI, Bishop and Snowling (2004) pointed out that single dimension of severity is not sufficient to capture the range of clinical variation that exists in these disorders. The authors admit that there are

commonalities between dyslexia and SLI in the type of phonological deficits experienced, but believe that children with SLI generally have additional syntactic and semantic difficulties affecting their language. Furthermore, a subgroup of LD called *language learning disability also have found to exhibit deficits at phonological, syntactic and semantic levels.* These deficits play an important role in attaining fluent reading. Bishop and Snowling (2004) believe that there could be a distinction between dyslexia and SLI. Children with SLI usually have the same phonological deficits that are traditionally regarded as a core feature (and a causal factor) of dyslexia. However, children with SLI also experience pronounced deficits in syntax and semantics, which have an additional affect on their literacy development. Bishop and Snowling (2004) concluded that although dyslexia and SLI present enticing similarities, it might prove essential to rethink the relationship between these disorders.

2.1. Theories on DD

Numerous theoretical approaches have identified different potential causes for dyslexia. Dyslexia is conceptualized as either a phonological, attentional, auditory, magnocellular or automatisation deficit by different theories.

2.1.1. The Phonological theory

The role of phonology and awareness to the phonological structure of a lexical representation is important in learning to read. Failure to acquire that skill is very well represented in the literature on dyslexia. Children with DD have difficulty with tasks tapping phonological awareness, indicating that dyslexia is related to the phonological component of language (Bryant, 1995; Elbro, 1996; Rack, 1994). Phonological awareness and decoding can provide a self teaching mechanism that can lead to and support accurate recognition of printed words. The mechanism is particularly important when children

encounter unfamiliar words in independent reading (Jorm & Share, 1983; Share, 1995). The phonological theory (Liberman, 1973; Snowling, 2000) is the most influential account for reading problems in children with dyslexia.

According to this theory, children with dyslexia have a specific impairment in the representation, storage and/or retrieval of speech sounds. It relates dyslexia to a deficit in phonological awareness, i.e. the ability to segregate and manipulate the speech sounds which form a word (e.g., deleting the first sound from the word "pant" gives "ant"). It explains reading difficulties showed by the children with dyslexia as a break down in developing the correspondence between letters and constituent sounds of speech i.e. grapheme-phoneme correspondence. If the sounds are poorly represented, stored or retrieved, the learning of grapheme-phoneme correspondences will be affected which will affect the learning of alphabet system (Bradley & Bryant, 1978; Brady & Shankweiler, 1991; Snowling, 1981; Vellutino, 1979).

Poor verbal short term memory and slow automatic naming also points to a basic phonological deficit (Snowling, 2001). Phonological Short-term Memory (PSM) is assumed to form sound-based representations of written symbols being stored transiently in the left posterior parietal cortex of brain. Efficient phonetic recoding in Broca's area of brain appears to be an important tool for the early reader. At the neurological level, functional brain imaging studies (Pugh et al., 2000; Shaywitz et al., 2002) and anatomical work (Galaburda, Sherman, Rosen, Aboitiz & Geschwind, 1985) suggested that a congenital dysfunction of the left perisylvian brain areas forms the basis of the phonological deficit.

Converging lines of evidence suggests that children with dyslexia can be characterized by one of several phenotypic manifestations of a phonological deficit (e.g.,

phonological awareness, PSM, phonological re/de-coding [i.e., Rapid Automatized Naming (RAN)]) (Brady & Shankweiler, 1991; Rack, Snowling & Olson, 1992). The researchers agree that these deficits in dyslexia are part of a more general double deficit theory (Wolf & Bowers, 1999; Wolf, Bowers & Biddle, 2000).

2.1.2. The double deficit hypothesis

Processes which underlie naming speed are largely independent of phonological processes and they represent a second core deficit in dyslexia. Wolf et al. (2000) had suggested that deficits in phonological awareness and RAN reflected a general impairment in automatizing low-level sub processes which is involved in reading. This suggests new sub-types that can be characterized by the presence, absence, or combination of the two core deficits in phonology and naming speed: the children with dyslexia having phonological-deficit, impaired word-identification accuracy (poor phonological awareness); the individuals with rate-deficit, exhibiting slowly word decoding profile and the double deficit reader, showing a general dysfunction on all decoding measures (Wolf et al., 1999). They also suggested that the presence of deficits in both phonological processing and RAN had an additive negative influence on reading performance above and beyond that of a single deficit.

Wolf et al. (2000) reported that the relationship of speeded naming to reading is dependent upon the subject's age and stimulus type. Semrud-Clikeman, Guy, Griffin and Hynd (2000) found that children with reading disability (RD) were found to be slower on letter- and number-naming tasks and made more errors on all tasks than controls. There was an age effect for the RAN/RAS tasks. Younger children with RD performed more poorly on all tasks, while the older children with RD showed poorer performance only on the letter- and number-naming tasks.

2.1.3. The Auditory processing deficit theory

This theory assumes that children with dyslexia have a deficit in rapid auditory processing. Auditory sensory deficits are proposed to cause impaired speech perception. There are evidences that children with dyslexia categorize speech stimuli less well than normal readers (Godfrey, Syrdal-Lasky, Millay & Knox, 1981; Steffens, Eilers, Gross-Glenn & Jallad,1992; Werker & Teas, 1987) and also their physiological responses to speech stimuli are different compared to those of control listeners (Schulte-Ko¨rne, Deimel, Bartling & Remschmidt,1998). Such speech perception deficits may lead to deficits in the ability to manipulate and process speech sounds or phonemic awarenessdeficits which can lead to difficulties in learning letter–sound correspondences during the process of reading development (Bradley & Bryant, 1983; Liberman & Shankweiler, 1985).

It is argued that no adequate phonological representations can be built due to this basic deficit, resulting in additional phonological impairments. Thus, according to this theory, phonological problems are only secondary to the auditory deficits i.e. the deficit lies in the perception of short or rapidly varying acoustic sounds (Tallal, 1980; Tallal, Miller, & Fitch, 1993).

2.1.4. The visual theory

It is assumed that dyslexia was not related to any deficiencies in visual functioning, but a problem with phonological deficit. In spite of this widespread belief, some researchers conceptualize dyslexia as a visual processing deficit (Livingstone, Rosen, Drislane & Galaburda, 1991; Lovegrove, Bowling, Badcock & Blackwood, 1980; Stein & Walsh, 1997) arising from the impairment of the visual magnocellular system in the brain. These studies demonstrated the presence of a variety of visual deficits among

children with dyslexia giving rise to difficulties with the processing of printed text. This can take many forms such as unstable binocular fixations, poor vergence (Cornelissen, Munro, Fowler, & Stein, 1993; Stein & Fowler, 1993; Eden, Stein, Wood & Wood, 1994), or increased visual crowding (Spinelli, De Luca, Judica & Zoccolotti, 2002). This led to the development of training programs for visual-perceptual and/or visual-motor disabilities.

The visual theory does not exclude a phonological deficit, but emphasizes a visual contribution to reading problems, at least in some children with dyslexia. The basis of magnocellular-deficit theory is the observation that the visual pathway leading from the eyes to the visual cortex consists of two parallel systems: the magnocellular and the parvocellular systems. The prevailing theory of dyslexia is that the magnocellular system of dyslexic readers has reduced sensitivity.

The magnocellular-deficit theory assumes that the inhibition of the parvocellular system by the magnocellular system is selectively disrupted in certain individuals with dyslexia, leading to deficiencies in visual processing, and, via the posterior parietal cortex, to abnormal binocular control and visuospatial attention (Hari, Renvall & Tanskanen, 2001; Stein et al., 1997). Evidence for magnocellular dysfunction comes from anatomical studies, psychophysical studies and brain imaging studies (Eden et al., 1996). There are anatomical studies showing abnormalities of the magnocellular layers of the lateral geniculate nucleus (Livingstone et al., 1991) and also psychophysical studies showing decreased sensitivity in the magnocellular range in children with dyslexia (Cornelissen et al., 1995; Lovegrove et al., 1980).

2.1.5 The automaticity or cerebellar theory

The Cerebellar Theory states that a mildly dysfunctional cerebellum can cause dyslexia. (Nicolson & Fawcett, 1990; Nicolson, Fawcett & Dean, 2001) The cerebellum contributes to motor control during the articulation of speech. The Cerebellar theory proposes that articulation problems can lead to deficient phonological representations that can cause dyslexia. The cerebellum also contributes to the automatization of learnt behaviors, which includes tasks such as driving, typing and reading. A weak capacity to automatize would affect many things including the learning of grapheme-phoneme correspondences. Evidences for poor performance of individuals with dyslexia in a large number of motor tasks support the cerebellar theory (Fawcett & Nicolson, 1996). Dual tasks which demonstrate impaired automatization of balance (Nicolson et al, 1990), and a non-motor cerebellar task, in time estimation (Nicolson et al., 1994) are a few supporting the same. Brain imaging studies have also shown metabolic, anatomical and activation differences in the cerebellum of children with dyslexia (Brown, Eliez, Menon, Rumsey, White & Reiss, 2001; Leonard, Eckert, Lombardino, Oakland, Kranzler & Mohr, 2001; Nicolson, Fawcett, Berry, Jenkins, Dean & Brooks, 1999; Rae, Lee, Dixon, Blamire, Thompson & Styles, 1998)

Different theories conceptualize dyslexia as phonological, auditory, visual or cerebellar deficit. But not all children with dyslexia suffer from deficits in all these domains. Such heterogeneity suggests the possibility of existence of distinguishable subtypes of dyslexia

2.3 Grammaticality deficits in children with DD

Various studies conducted on preschool children at genetic risk for DD have reported language delays, specifically concerning the perception and production of

grammatical morphology (Lyytinen, Poikkeus, Laakso, Eklund, & Lyytinen, 2001; Rispens, 2004; Scarborough, 1990; van Alphen et al., 2004; Wilsenach, 2006).

In young children as old as 5 year and 8 year old children Theakston (2004) found overgeneralization errors while producing a sentence. Prema (1979) attempted to investigate syntactic abilities of 5-6 year old children. Four children in the aforementioned age range with Kannada as their mother tongue were chosen for the study and their spontaneous speech and story narration was studied. It was reported that transformational rules to derive negative sentences were still in the process of acquisition. Speech samples from these children were collected for three days using spontaneous speech and story narration task.

Scarborough (1990) conducted a study with children at-risk of developing dyslexia. She found that 65% of the children in her sample of at-risk children could be classified as dyslexia by the age of 8 years. A retrospective analysis of the early language skills of these children revealed that they experienced more language difficulties as kindergartners than a group of normal controls. These language difficulties had a changing pattern over time. At age 30 months, the children with dyslexia had a similar range of vocabulary items as the comparison group, but they demonstrated a more restricted range of syntactic devices and made more speech production errors. However, at the ages of 36 and 42 months, the vocabulary skills of children with dyslexia were less well developed than those of the controls and their syntactic difficulties persisted (Scarborough &Dobrich, 1991). At age 60 months,children with dyslexia displayed deficiencies in phonological awareness and letter knowledge, but their syntactic difficulties were no longer visible. According to Scarborough, the most important finding of her study was that phonological skills did not account for significant variance in

outcome, but that syntactic skills was a unique predictor of reading disability. Thus, she argued, the phonological deficit hypothesis cannot fully explain the occurrence of dyslexia.

In an attempt to evaluate the phonological deficit hypothesis and extend the available evidence concerning the precursors of dyslexia, Gallagher, Frith, & Snowling (2000) studied 63 at-risk children with an average age of 45, 68 months. They found that almost half of the at-risk children were late in taking their first steps into literacy development. Retrospective analysis of their language development suggested that these children were subject to mild delays in all aspects of their spoken language. Consistent with Scarborough's finding that preschool syntactic ability was a significant predictor of reading at 8 years, the language factor (sentence length as a measure of syntactic proficiency) in Gallagher et al.'s study accounted for unique variance in literacy development. Furthermore, consistent with the other above-mentioned studies, Gallagher et al. found that the at-risk children recognized fewer letters than the control children at age 45 months.

Thus, it is still not clear whether a specific aspect of language ability (e.g. phonological processing) or a more general language delay is most directly responsible for reading failure. More evidence of delayed morphosyntactic development in young at-risk children comes from studies by Lyytinnen et al. (2001) and Rispens (2004). In Lyytinnen et al's study, at-risk children produced significantly shorter sentences (measured in MLU) than their normally developing peers at the age of 2; 0. Rispens (2004) found evidence that kindergarten children at-risk for dyslexia were less sensitive to violations of subject-verb agreement than normally developing children. Rispens revisited the at-risk children in her sample after they had received one year of reading instruction and found that those children who did not show normal reading progress

differed significantly in their sensitivity to subject verb agreement from children who show normal reading progress.

2.3.1 Extended Optional Infinitive Stage, the Agreement and Tense Omission Model

Rice & Wexler (1996) and Wexler, Schutze and Rice (1998) proposed the Extended Optional Infinitive Stage (EOIS) as an explanation of SLI. This explanation suggests that the *Optional Infinitive Stage (OI)* (Wexler, 1994), a developmental stage that normally developing children go through between the ages of approximately 1; 10 and 3; 6, persists in children with SLI. During this developmental stage, children use both bare infinitives and finite verbs in finite contexts. Finiteness is not obligatory yet and as a result, the tense and agreement features of the verb are not always marked. So, for example, in a past tense context a child in the OI stage might say either *I fell on my knee* or *I fall on my knee*, alternating between the finite verb form *fell* and the bare infinitive form *fall*. Wexler also observed that during the OI stage, children tend to omit auxiliaries and copula BE in finite contexts, saying for instance *Mommy eating* instead of *Mommy is eating* or *Daddy gone* rather than *Daddy has gone.* The generalization is that during the OI stage, children alternate between producing finite and non-finite clauses in finite contexts. Wexler and Rice suggest that children with SLI go through an Extended Optional Infinitive stage, which typically lasts until they are 7 or 8 years old.

Initially, Wexler (1994) argued that children with SLI have a Tense Deficit in the sense that they sometimes mark tense but sometimes leave verbs underspecified for tense. The use of bare infinitive forms in contexts that require finite forms represents a tense-omission error under Wexler's assumptions. In later collaborative work between Wexler and Schütze (Schütze & Wexler 1996), it was argued that optional infinitives could arise as a result of either tense or agreement features (or both) being underspecified (i.e.

omitted). In their 1998 paper Wexler, Schütze and Rice argue that SLI involves a syntactic feature deficit, which leads affected children to sometimes omit tense and agreement features in obligatory contexts. Wexler et al. refer to this model as ATOM (Agreement & Tense Omission Model). Support for this model comes from erroneous case marking of subject NP's by children with SLI and (younger) normally developing children using root infinitives. In adult English, the subject must be marked with nominative case. However, in SLI and in early (normal) language development, children use root infinitives with accusative subjects as in *him fall down* and *her have a big mouth* (examples from Schütze, 1997). Tense and Agreement both have a relation with the subject. Tense licenses overt participants, while Agreement assigns the subject's case. With regard to case marking, the Agreement & Tense Omission Model predicts that:

- Structures containing an inflected verb (both tense and agreement are marked) will have nominative subjects.
- Modal/past tense structures in which tense is marked, but agreement may or may not be marked will have either nominative or default participants (i.e. *He/Him went out*).

A bare infinitive is an infinitival verb-form used without the infinitive particle *to*. The infinitival status of the verb-form is clearer in languages in which infinitives carry an overt inflection, such as German or Dutch. A finite context is a context where an adult would use a (auxiliary or main) verb marked for tense and agreement. Bare verb/missing auxiliary structures are ambiguous with respect to whether they are (i) specified for agreement but not for tense, (ii) specified for tense but not agreement, or (iii) specified for neither tense nor agreement. Such structures will have nominative subjects (i.e. *He snore, He snoring*) in the case of (i) and accusative subjects (e.g. Him snore, Him snoring) in the case of (ii) / (iii).

Both the EOI and ATOM accounts presume a delay in the language development of children with SLI. Thus, the grammar of a child with SLI is not qualitatively different from the grammar of a normally developing child; it merely develops at a slower pace.

2.3.2 The Missing feature hypothesis / Implicit Rule Deficit Hypothesis

In this account, the grammatical problems of children with SLI are believed to stem from an underspecification of the morphosyntactic rules that mark features such as tense, number and person. Gopnik (1990a, 1990b) originally characterized the problem as *feature blindness* (the features of tense, number and person are missing from the underlying grammars of children with SLI) and based this assumption on a case study of a language-impaired boy. Morphophonological rules and rules that match features in the syntax were absent in the language production of this boy. Feature blindness entails that the grammatical morphemes that encode these features will be produced in a haphazard way. Gopnik presented further evidence for her account with data from a British family (the KE-family).

Gopnik and Crago (1991) reconstructed the missing feature hypothesis by proposing an absence of rules rather than features in the grammars of children with SLI. They proposed the Implicit Rule Deficit account as an explanation of SLI, using as backdrop the dual model of acquisition of morphology (Pinker & Prince, 1988). According to the dual model, regular and irregular inflections are acquired differently. Irregular forms are stored in memory, whilst the acquisition of regular morphology necessitates an abstract rule that affixes a morpheme to a verb stem. The suggestion of the implicit rule deficit hypothesis is that such abstract rules are not available to children with SLI and that these children can use only memory. Thus, learning regular forms will be approached in the same way by children with SLI than learning irregular forms (i.e. they will also be rote-learned).

However, the implicit rule deficit hypothesis cannot account for the fact that children with SLI sometimes produce overgeneralizations of irregular verb forms. Such productions are an indication that a rule is 'at work' and cannot be rote-learned, as they do not appear in the input. Also, the authors of this account have struggled to deal with the problem of grammatical utterances in an 'ungrammatical child'. Gopnik (1991) assumes that correct manifestations of grammatical morphology in children with SLI are also to be interpreted as the outcome of a deficient system. Thus, examples that do not fit the theory are not seen as counterevidence, but as the chance output of a deficient grammar. As Bol and De Jong (1999) point out, falsifying such a theory is hard or even impossible, making the theory itself weak.

2.3.3 The Missing agreement hypothesis

The Missing Agreement hypothesis was proposed by Clahsen (1989). Working with data from German-speaking children with SLI, Clahsen proposes that the children's problem lie in establishing the structural relationships of grammatical agreement. Clahsen claims that children with SLI lack the knowledge of asymmetrical relations between categories, where one category controls the other. Support for this claim came from his observations that children with SLI have trouble with gender and number markings on determiners and articles. Furthermore, the children in this initial study frequently made agreement errors on verbs and produced sentences with the verb in the final position rather than in the appropriate second or first position. According to Clahsen, these children's ungrammatical verb-final productions are not the result of a deficit in 'movement' itself, but are related to their inability to generate the morphology (i.e., finite forms) required for the verb-second position.

Grammatical morphemes are missing from the output as children lack the agreement relations that permit their use and not because the children cannot produce the

forms themselves. Following Generalized Phrase Structure Grammar (particularly the Control Agreement Principle (see Gazdar, Klein, Pullum, & Sag, 1985), Clahsen (1992) anticipates that a deficit in Agreement will cause problems in several domains that depend on agreement relations. The Missing Agreement hypothesis predicts that subject-verb agreement, finite forms of auxiliaries, overt structural case marking and gender marking on determiners and adjectives will all be deficient in SLI. Furthermore, the Missing Agreement hypothesis does not predict significant difficulties with Tense. Clahsen acknowledges that children with SLI have problems with Tense, but he sees such problems as marginal in comparison to the problems experienced with subject-verb agreement.

2.4.6 The surface hypothesis

The surface hypothesis (Leonard, 1989, 1998) is perhaps the best known of the non-modular accounts of SLI. The physical properties of grammatical morphemes in English are accentuated in this account, hence the term "surface" hypothesis. Acoustic features such as low phonetic substance and short duration render the acquisition of English grammatical morphology difficult even for normally developing children. The surface hypothesis supposes "a general processing capacity limitation in children with SLI but assumes also that this limitation will have an especially profound effect on the joint operations of perceiving grammatical morphemes and hypothesizing their grammatical function" (Leonard, 1998). This limited processing capacity is maybe best described as a *reduced speed* of processing. The main idea is that children with SLI can perceive word-final consonants and weak syllables, but that these children's processing capacity is severely strained when such taxing forms play a morphological role. When this is the case, children do no not only have to perceive the short duration consonant or weak syllable, they also have to perform additional operations to discover that it functions

as a separate grammatical morpheme and fills a specific cell of the morphological paradigm. These additional operations are in effect those discussed in Pinker's (1984) learnability model, in which Pinker offers an explanation for the way in which children build morphological paradigms. According to this model, language acquisition starts off with children forming word-specific paradigms. In the course of language development these paradigms become general and children become aware of the fact that specific affixes represent specific syntactic features. When this happens, children "know" that different affixes represent different dimensions and they can apply them to new words. Thus, on hearing the Italian verb *corre* (he/she runs) a child who has moved from word-specific paradigms to general paradigms can produce *corro* (I run) without having heard it before. Affixes are not all acquired at the same time. Rather, Pinker (1984) argues for a ranking order determined by several characteristics of the affixes themselves. Affixes that are perceptually salient and semantically transparent (e.g. *–ing* and plural *–s* in English) are introduced earlier than affixes that are non-salient or abstract in nature (e.g. 3rd singular *–s* in English).

Ultimately, the surface hypothesis assumes the underlying grammars of children with SLI to be in tact; the morphological paradigms that these children form are essentially the same as those of normally developing children. However, due to their reduced processing speed, the input of children with SLI is distorted. In particular, grammatical morphemes that are perceptually unsalient are at risk of not being perceived or processed. As the processing speed limitation is believed to be general rather than specific, its effect may be different from one language to another. In English, grammatical morphology is affected because of the fact that morphology is quite fragile. In a language with a typology that differs markedly from English (e.g. Italian), the effects

of the same processing limitation can lead to a different kind of linguistic profile in children with SLI

2.4.7 The limited processing account

Another non-modular explanation of SLI is that children with SLI have a limited resource capacity for the processing and storage of information. The notion of a limited capacity system has been incorporated in various models of language processing (Baddeley, 1986, 1996; Bloom, 1993; Bock & Levelt, 1994; Just & Carpenter, 1992). These models all share the same underlying idea that our cognitive resources (which are assigned to different tasks) are limited. In situations where task demands exceed available resources, the processing and/or storage of information are affected negatively. In other words, when processing one aspect of a cognitive task is unusually difficult and demands a lot of the available resources, fewer resources are left for processing other aspects. Thus, breakdowns in performance can occur when processing demands exceed resource capacity. Although task accuracy also is used to measure processing capacity, the speed with which mental operations are carried out is viewed as a fundamental processing resource.

Capacity limitations are typically revealed in linguistic interactions and trade-offs across language domains. Individual differences in cognitive capacity are thought to hamper language processing more in some individuals than in others (Just and Carpenter's, 1992).

Several researchers have suggested that children with SLI, even more than normally developing children, have limitations in their capacity to process and store information (Bishop, 1992b; Ellis Weismer & Hesketh, 1996; Gathercole & Baddeley, 1990, 1993; Johnston, 1994; Lahey & Bloom, 1994; Montgomery, 1995a, 1995b). Limitations are

proposed to be either specific to a particular resource capacity (e.g. Gathercole & Baddeley, 1990 proposed a limitation in phonological working memory) or more general.

General resource capacity limitations include limitations in working memory size, computational energy, processing rate, or all three (Kail & Salthouse, 1994)

2.4 Behavioral studies in Dyslexia

Scarborough (1990; 1991) studied (morpho) syntax in pre-school children at genetic risk for DD in the context of the aforementioned prospective study. Assessments started at the age of 30 months and there were follow-ups at 36, 42, 48 and 60 months. Expressive syntax was assessed by calculating the mean length of utterances (MLU scores) and by the Index of Productive Syntax (measuring morpho-syntactic complexity). Receptive syntax was evaluated with the Northwestern Syntax Screening Test (Lee, 1971). The results of the three measures showed that from 30 months of age until 48 months the group of children who later turned out to be dyslexic differed significantly from the control group. However, performances at 60 months of age proved not to be significantly different. Thus, between 2 and 4 years of age, children with dyslexia showed problems both in comprehension and production of syntactic structures and morphemes marking morpho-syntactic information. The observation that the discrepancy between the two groups dissolved at the age of 5 may indicate that development of morpho-syntax is delayed in dyslexia, but that children catch up around the age of 5.

According to Scarborough (1990; 1991), this interpretation may not be correct since studies that included older participants with dyslexia have demonstrated syntactic deficits. It could well be that the results of the syntactic measures at the age of 60 months may have been constrained by the way syntax was assessed. Neither the Index of Productive Syntax nor MLU have proven to be valid measures of syntactic proficiency for that age group. Another account of the findings may be that both groups of children

reached a temporary plateau in their track of language development. Therefore, even though no group differences were found at that moment, these may re-emerge later.

More evidence of delayed morpho-syntactic development in pre-school children at genetic risk of dyslexia comes from two longitudinal studies that are currently undertaken. Lyytinen et al. (2001) found that a group of at-risk children produced significantly shorter sentences as measured by MLU in morphemes at 24 months old. Wilsenach & Wijnen (2003) demonstrated that Dutch at-risk children from 18–23 months do not distinguish sentences containing the correct temporal auxiliary combined with a past participle (*heft geslapen*, has slept) from an ungrammatical combination of a modal with the past participle (*kan geslapen,* can slept). This is in contrast with the control children, who showed a significant preference for the natural constructions.

Syntactic abilities have also been assessed in older children with dyslexia. Children with dyslexia were more impaired in the ability to understand and repeat relative clauses (Mann et al., 1984; Stein et al., 1984), and also produced fewer relative clauses with object movement (e.g. *the cat that the monkey scratched climbed up the tree*) compared to non-dyslexic children (Bar-Shalom et al., 1993). Stein et al. (1984) found that children with dyslexia made more errors with the interpretation of passive sentences relative to controls, but that overall performance was good, indicating attainment of the passive construction. Waltzman & Cairns (2000) demonstrated that third grade poor readers 2 had more trouble with the interpretation of pronouns in some sentence contexts compared to normally reading children. Joanisse et al. (2000) found that children with dyslexia of around 8 years old made more errors in inflecting verbs for the past tense than control participants.

Rispens and Been (2007) attempted to investigate whether the phonological processing deficits and morphosyntactic problems in children with dyslexia are causally

related. They found that children with dyslexia performed poorer than age matched controls on subject-verb agreement, phonological awareness and non word repetition task. The authors also found associations between non word repetition and sensitivity to subject verb agreement thus indicating that problems with phonological processing have an impact on the morphosyntactic deficits.

As a group, children with learning disabilities are markedly deficient in their ability to hold together grammatical frames and abstract meanings from larger contexts (Wallach & Goldsmith, 1977). During auditory language comprehension these children often process single words and fail to process clauses together. An accumulating body of evidence indicates that poor readers do not comprehend sentences as well as good readers do (Mann, Cowin & Schoenheimer, 1989). It has been shown that good and poor readers differ in the ability both to repeat and to comprehend spoken sentences that contain relative clauses (Mann, Shankweiler & Smith, 1985). Some investigators attribute this to deficient syntactic abilities in children with learning disabilities while some opine that the comprehension problems were mainly due to their short term memory deficits.

Rispens and Been (2007) examined sentence comprehension in children with dyslexia and found that children with dyslexia were poorer than control children. Botting, Simkin, and Conti-Ramsden (2006) found that in a group of 11-year-old poor readers, the strongest predictor of word recognition and reading comprehension was sentence comprehension at age 7 years.

In conclusion, there is evidence that development of syntactic structures and inflectional morphology in children with dyslexia (pre-schoolers and older) is at least delayed compared to their peers as also observed in children with SLI.

2.5 Event Related Potentials and Language

A strong element in the ERP research regarding the linguistics emerged by the discovery of the first "language" component, the "semantic" N400 (Kutas & Hillyard, 1980). Additional ERP components associated with phonological, acoustic-phonetic, syntactic, orthographic and prosodic processes have been discovered in the intervening years. The ERP components that are early (100-200 ms), fast, and automatic tend to be concerned with basic operations such as phoneme discrimination or word segmentation. Other components which have larger latencies (up to 1s) reflect integration or revision processes.

There have been five ERP components identified that are involved in language processing (Brown, Hagoort & Kutas, 2000).

a) ***Phonological mismatch negativity (PMN):*** PMN is the earliest component which is elicited by contextually unexpected phonemes in language tasks (Connolly, Phillips & Forbes, 1995). This ERP component peaks in the late 200 ms range (270-310 ms) and is fronto-centrally distributed. It is earlier than and distinct from the N400 reflecting pure semantic anomalies. The PMN precedes the N400 in combined violations of phonological and semantic expectations.

   PMN is found to occur in response to all sentence-ending words but is larger to those that violate phonological expectations. The PMN not related to the mismatch negativity (MMN) as the label phonological mismatch negativity (PMN) (Conolly & Philips, 1994) might appear misleading to the description of its behavior. Thus, the phrase that better describes its behavior is phonological mapping negativity as it referred now. As the PMN is equally responsive to words and non-words, it appears prelexical and also modality specific (auditory). It also appears to be related to phonological awareness, is insensitive to phonologically

correct pattern masking and responds to single phoneme violations of localized expectations (Conolly et al., 1992). Preliminary data indicates that PMN is absent in many poor and dyslexic readers. Newman et al. (2004) has isolated the PMN from frequently occurring larger negativities using a phoneme deletion paradigm. These findings have confirmed that the PMN is a prelexical response, which reflects a compulsory stage of word processing that is sensitive to top-down phonological expectations. Currently, it has been suggested that PMN reflects phoneme awareness and the consequent phonological processing activity. Once a violation of expectations is perceived, although PMN is influenced by top-down phonological expectations, it does not appear to be sensitive to gradations of phonological relatedness. But rather shows an "all-or-none" response that is equally large for all violations. The PMN may reflect a phonological stage of word processing that operates at the level of transforming acoustic input into phonological code assisting in the establishment of a lexical cohort. It is also compatible with the data to suggest that the PMN may reflect the earliest point at which top-down contextual information influences bottom-up processes at or just prior to the isolation point within, for example, a version of the Cohort Model (Connolly & Phillips, 1994).

b) ***Left-anterior negativity (LAN):*** This component is elicited about 200–500 ms after word onset and it seems to be involved in the processes of working memory. Also, LAN is involved in the activation and processing of syntactic word-category information that is in a sentence whether the word is acting as a subject, a verb or an adjective, etc. (Friederici, 1995; Friederici, Hahne & Mecklinger, 1996; Kluender & Kutas, 1993). Early LAN (ELAN), between 100 and 300 ms, have been found particularly with the word category violations. ELAN appears to be

more reliable in auditory than visual studies. ELAN has been elicited from the neural generators in Broca's area and the anterior temporal lobe. The later LANs elicited between 300 and 500 ms represent other morpho-syntactic operations (and respective violations) which are processed in parallel to semantic information, affecting agreement features or verb arguments which already depend on a phrase marker. ELANs are not influenced by the relative proportion of violations in an experiment, suggesting their "autonomous" status independent of processing strategies (Hahne & Friederici, 1999). ELANs are seen in robust to greater extent in auditory experiments. Similar early anterior negativity over the right hemisphere [ERAN (early right-anterior negativity)] have been reported in studies for certain musical violations (Patel et al., 1988). Hagoort et al. (2003) replicated ELAN effects in a Dutch reading study that avoided word initial markings of the word category. They observed an anterior negativity only between 300 and 500 ms which, moreover, was bilaterally distributed rather than left lateralized. Lau et al. (2006) found that clear LAN-like effects occurred only if local phrase structure imposed high constraints on the target word, whereas less predictable structures resulted in attenuated LAN effects. Predictability and expectations may be crucial to our understanding of LAN-like effects in morpho-syntactic processing more generally.

c) ***N400 component***: It is a negative waveform that is elicited between 300 and 500 ms after word onset. N400 is related to the semantic processing or, processing of the meaning of the stimulus in its context (Kutas & Federmeier, 2000). This component includes a centro-parietal distribution and, in several tasks, an additional frontally distributed component with a slightly different functional

significance (Kounios & Holcomb, 1994). This peak is of importance in the present study and will be discussed in detail in the following sections.

d) ***P600 component***: A positive waveform that is seen about 500–700 ms after word onset and belongs to the P3b family of components (Kok, 2001). P3b components are large, positive peaks elicited in response to a wide variety of stimuli. P600 are found to be elicited for syntactical violations in a sentence. For simple stimuli such as tones or coloured dots, they can peak as early as 275 ms post onset. Whereas for complex stimuli such as words, they peak at 600 ms. Depending on the difficulty of the task the peak can be delayed up to 2000 ms. Eventhough the latency can be so long, they still belong to the same family of P3b components because the distribution at the peak maximum is over parietal scalp sites. Also, because their amplitude can be modulated by the probability of the occurrence of a particular stimulus (Kutas, McCarthy & Donchin, 1977). It has been speculated that ending of the stimulus evaluation process has been reflected in P3b components. The amplitude increases as the amount of information that has been consciously extracted from the presentation of the stimulus increases (Debruille, 1998; Curran, 1999; Coulson, King & Kutas, 1998; Donchin & Coles, 1988). A large, positive and parietal component peaking at 600 ms post onset has been elicited for unexpected syntactic anomalies in several studies (Osterhout & Holcomb, 1992; Hagoort, Brown & Groothusen, 1993; Munte, Matzke & Johannes, 1997). The authors have thus claimed that P600 would not be a component of the P3b family and is elicited by words in normal discourse reflects a type of syntactic processing, such as "second pass parsing," (Friederici, Hahne & von Cramon, 1996). However, it has been contradicted by many authors that it does not represent syntactic processing (Curran, 1999; Gunter, Stowe & Mulder,

1997; Munte, Heinze, Matzke, Wieringer & Johannes, 1998). In terms of contradiction, Kaan et al. (2000) demonstrated that P600 can be evoked by structurally more complex sentences even in the absence of any violation or ambiguity. Thus, these findings suggest that the P600 is a rather general marker for structural processing.

e) ***Slow Positive Shift:*** The slow positive shift is a broad component that develops throughout the span of a sentence. This slow positive shift has been recently identified which is related to the construction of a representation of the overall meaning of the sentence (Brown, Hagoort & Kutas, 2000).

Three language-related ERPs are often used in the analysis of language processing in children with language impairment (LI). The N400, P600, and early left anterior negativity (ELAN) have been shown to be sensitive to semantic and syntactic processing (Friederici, 2004). There is evidence suggesting that children with LI experience deficits in a variety of linguistic domains including learning semantic and syntactic rules (Cummings & Ceponiene, 2010; Friederici, 2006; Friedrich & Friederici, 2006; Sabisch, Hahne, Glass, Von Suchodoletz, & Friederici, 2006b; Weber-Fox, Leonard, Wray, & Tomblin, 2010). The N400 is typically associated with semantic processes, while the P600 and ELAN are highly correlated with syntactic processes (Friederici, 2004). Research using these three components has been directed more toward the adult population as well as typically developing children. However, research using children with LI may be beneficial in understanding their neurophysiological development of language.

N400 as an indicator of semantic processing. As a reflection of linguistic processing, the N400 is a negative waveform that peaks at around 400 ms after the onset of the

critical stimulus and is correlated with lexical-semantic processes (Friederici, 2004). The N400 is sensitive to the appropriateness or the semantic relationship of a word within a given context.

Generally, larger N400 amplitude is elicited for words that are semantically incongruous to a given context, rather than words that are congruous. If a larger N400 amplitude is seen in the presence of semantically incongruent stimuli, it is known as the "semantic N400 effect" (Friederici, 2006). When the semantic N400 effect is not present with semantically incongruent stimuli, this frequently indicates a deficit in semantic processing (Friederici, 2006).

In a longitudinal study by Friedrich and Friederici (2006), children that were at risk of developing LI, indicated by a family history of LI, were investigated. Participants of this study were first tested at 19 months of age, and again at 30 months. The results of the study showed that children who experienced poor expressive language skills at 30 months, and were also at risk for LI, failed to show a semantic N400 effect at the age of 19 months when presented with semantically incongruous stimuli (Friedrich & Friederici, 2006).

Studies by Kutas and Hillyard (1980a, 1980b, and 1983) also showed that the N400 is sensitive to semantic stimuli. These researchers examined the electrophysiological results of manipulating a word within a sentence and presenting the sentences to a group of young adults. Kutas and Hillyard found that when they replaced a word with a semantically inappropriate word, an increased negative peak was seen at approximately 400 ms. These studies also showed that the amplitude of the N400 was greater when the participant was not expecting the target word (Kutas & Hillyard, 1980a, 1980b, 1983).

Another study conducted by Polich (1985) resulted in similar findings when manipulating the semantic component of a sentence. The study consisted of two experiments. In the first, participants were required to read the sentences that were presented to them. In the second, the participants were asked to indicate whether the sentence presented to them did or did not end in a semantically appropriate word. In the second experiment, the N400 component was present when the sentences ended in a semantically inappropriate word (Polich, 1985).

The N400 has been observed in both visual and auditory modalities. However, the N400 demonstrates topographical differences as a function of presentation modality. When elicited visually, the N400's distribution is greater over the right hemisphere. When elicited auditorily, the N400's distribution is typically lateralized to the left hemisphere. It has also been shown that the N400 latency is shorter for auditory stimuli than for visual stimuli. There may be overlap in the processes that occur in both visual and auditory modalities, but these processes are not identical (Holcomb & Neville, 1990).

ELAN as an indicator of syntactic processing. A second component of ERPs that will be used in the current study is the ELAN. This ERP component typically occurs between 100 and 200 ms and displays an anterior distribution. The ELAN has been reported for phrase structure violations in the auditory domain (Friederici, 2004). In a study by Hahne, Eckstein, and Friederici (2004), participants were auditorily presented with correct, semantically incorrect, and syntactically incorrect sentences. Participants included in the study consisted of children ages 6 to 13. The participants were asked to indicate if the sentences were correct or incorrect. Results showed an ELAN for syntactic violations, but it was not observed in children between the ages of 6 and 10 years. Hahne et al. (2004) indicated that higher automatic structure building processes reflected in the ELAN gradually develop toward adult-like processing during the later years of childhood,

reaching adult-like maturity at about 13 years. Canseco-Gonzalez (2000) stated that the ELAN is observed with outright syntactic violations or word category violations. Canseco-Gonzalez (2000) also stated that it is distinct from the N400 in that it has a more frontal distribution and smaller amplitude than the N400.

P600 as an indicator of syntactic processing. The P600 is a late ERP component that is related to syntactic processing. The P600 is a positive wave elicited at about 600 ms poststimulus and has been observed with violations of structural preferences, outright syntactic violations, and difficulty of syntactic processing (Friederici, 2004). In a study by Hahne et al. (2004), the P600 was analyzed after participants were presented with correct, semantically incorrect, and syntactically incorrect sentences. In this study, the P600 was elicited in response to syntactically incorrect sentences and was present in all groups of children from 7 to 13 years of age. However, the amplitude was smaller and the latency decreased as age increased. Because the amplitude of an ERP indicates the effort a participant uses to process a given stimulus, the results of this study may indicate that as a child ages, syntactic analysis becomes less difficult. Hahne et al. (2004) concluded that "As the P600 can be interpreted as a component related to the difficulty of syntactic integration, the present finding may point to high syntactic processing expenses, even for syntactically correct sentences" (Hahne et al., 2004, p. 1314).

Additional information about the elicitation of the P600 was obtained from Osterhout and Holcomb (1992). In this study, participants were presented with sentences that bring about the "garden-path effect" (Osterhout, 1992, p. 788). Osterhout and Holcomb (1992) explained the garden-path effect by stating that readers will typically construct a preferred syntactic representation in the initial stages of reading. When this preferred syntactic representation proves to be inappropriate, the reader will backtrack or reanalyze the sentence. This is what Osterhout and Holcomb (1992) refer to as the

"garden-path effect". For example, in the sentences *Thebroker persuaded the man to sell the stock* and *The broker persuaded to sell the stock was sent tojail,* the first sentence is the syntactically preferred sentence. However, the second sentence shows an ambiguous interpretation and is not syntactically preferred. In this case, the brain would then backtrack and reanalyze the sentence (Osterhout, 1992).

2.4.1 ERP studies in dyslexia

Sabisch et al. (2006) hypothesized that the syntactic deficits in children Learning Disability may be linked to the inability of these children to use prosodic information for syntactic processing. The authors used ERPs to compare children with typical development and age, gender and non verbal intelligence- matched children with learning disability on the domain of sentence comprehension. In the study, event-related brain potentials (ERPs) were used to compare auditory sentence comprehension in 16 children with DD (age 9–12 years) and unimpaired controls matched on age, sex, and nonverbal intelligence. Passive sentences were presented, which were either correct or contained a syntactic violation (phrase structure) or a semantic violation (selectional restriction). In an overall sentence correctness judgment task, both control and children with dyslexia performed well. In the ERPs, control children and children with dyslexia demonstrated a similar N400 component for the semantic violation. For the syntactic violation, it was found that the children with dyslexia showed a different pattern characterized by delayed negativity compared to control children.

Benton (2013) did an ERP study on seven children which included five typically developing children and two children diagnosed with language impairment. All children were administered on CASL (Comprehensive Assessment of Spoken Language), CELF-4 (Clinical Evaluation of Language Fundamentals) and WISC (Wechsler Intelligence Scale

for children IV). Three types of sentences were presented which included semantic, syntactic and canonical sentences. Syntactic errors included one of the following: (a) a plural noun error, (b) a past tense *–ed* verb error, (c) a past tense irregular verb error, or (d) a third person verb error. The errors were consistent with the participants' regional dialect. All syntactic and semantic errors occurred in the final word of the sentence. The correct and incorrect versions of the same sentence were randomized and never occurred consecutively.

The auditory evoked potential waveforms obtained for each participant were averaged for both linguistically correct and deviant conditions (syntactically and semantically incorrect). The latency of the N400 was defined as the prominent negative peak within the latency range of 300- 600 ms at the central midline (Cz) recording site or at recording sites adjacent to the Cz recording site. The magnitude of the N400 was obtained by measuring the amplitude of the waveform from the baseline to the peak amplitude of the N400. The latency of the ELAN was defined as a prominent negative peak within the latency range of 150 to 300 ms at the Cz recording site or at adjacent recording sites. The magnitude of the ELAN was obtained by measuring the amplitude of the waveform from the baseline to the peak amplitude of the ELAN. The latency of the P600 was defined as the prominent positive peak within the latency range of 500 to 800 ms at the Cz recording site or at recording sites adjacent to Cz. The magnitude of the P600 was obtained by measuring the amplitude of the waveform from the baseline to the peak amplitude of the P600.

The results showed that N400 for LI children, the amplitude decreases and latency increases going from linguistically correct sentences, to syntactically incorrect sentences, and finally to semantically incorrect sentences. The N400 is typically elicited by semantically incongruent stimuli and precedes the P600 in latency, while the P600 is

elicited by syntactical errors (Friederici, 2004; McPherson & Salamat, 2004). This suggests that semantic stimuli are processed before syntactic stimuli. The results are consistent with this notion. In LI-2, the N400 was only seen for the syntactically incorrect sentences.

The P600 is present for all three conditions in LI-1; however, it is only seen in the syntactically incorrect sentences for LI-2. The ELAN is only present in syntactically incorrect and semantically incorrect sentences for LI-1.

When participants were presented with semantically incongruent stimuli, results showed enhanced activity over the posterior temporal and temporal parietal lobes. Thus, the temporal lobe is known to be involved in semantic processes (Newman et al., 2001). The N400 is elicited from semantically incongruent stimuli and, when presented auditorily, will typically elicit an N400 lateralized to the left hemisphere. The amplitude of the P600 for the CG was higher when the CG was presented with syntactically incorrect sentences than when presented with linguistically correct or semantically incorrect sentences. The P600 is a wave observed in the presence of outright syntactic violations (Friederici, 2004). This suggests that the CG had no difficulty in detecting this syntactic error. When analyzing the results from LI-2, P600 is only present when LI-2 was presented with syntactically incorrect sentences. This suggested that LI-2 is also detecting syntactical errors. When analyzing brain activity and its distribution from the current study, the conclusion that both of the participants with LI are detecting the syntactical error may be supported. LI-1 and LI-2 showed more differences in activity from the CG when presented with semantically incorrect sentences than with syntactically incorrect sentences. This suggested greater difficulty in processing semantic errors than syntactic errors for the participants with LI.

The difference between the CG and LI-1 is that LI-1 has significantly larger ELAN amplitude than the CG. Larger amplitude suggested that LI-1 required a greater amount of effort to process the syntactic and semantic errors than the CG (McPherson & Salamat, 2004). This result suggested that, at least by eight years of age, typically developing children may process linguistic information similar to adults in regards to the areas of the brain that are activated during the processing of linguistic stimuli (Hickok & Peoppel, 2007; Newman et al., 2001). This observation is consistent with Hahne et al. (2004) who reported that the neurophysiological basis for semantic processes during auditory sentence comprehension does not change dramatically between early childhood and adulthood.

During the last decade, research on DD has accumulated, and yet the theories about mechanisms underlying this deficit are still controversial (Ramus et al., 2003; Witruk, Friederici, & Lachmann, 2002; Habib, 2000; von Suchodoletz, 1999). The question in the present study is, ‘‘Do children with DD with reading and writing, have impairment in syntactic processing?”

The present study was conducted to investigate whether our assumption holds that children with DD differ from control children in syntactic processing. In the current study, subject-verb agreement sentences were studied. The typical electrophysiological pattern associated with subject-verb agreement violations consists of a LAN (mainly shown at F3 and F7 electrodes) followed by the P600 component (with maximal amplitude at posterior electrodes). This bi-phasic pattern has been found in several electrophysiological investigations (e.g. De Vincenzi et al., 2003; Osterhout & Mobley, 1995; Vos et al., 2001) but has not been replicated in others (e.g. Atchley et al., 2006; Balconi & Pozzoli, 2005; Hagoort et al., 1993; Münte, Matzke, & Johannes, 1997; Osterhout et al., 1996, see Table 2.1). Differences in agreement morphology features

across languages might be the cause of inconsistencies found in the ERP pattern. Adult-like electrophysiological correlates of syntactic processing have been found in 32-montholds who passively listened to correct sentences (Der Löwe brüllt; the lion roars) and sentences with a local phrase structure violation (Der Löwe im __ brüllt; the lion in-the __ roars; Oberecker, Friedrich, & Friederici, 2005). Specifically, a bi-phasic ERP-pattern consisting in a child-like ELAN (300-500 ms) and a late P600 (1100-1500 ms) was observed. Interestingly, by using the same paradigm in 24-month-olds, only a P600 (1100-1700 ms) was observed. The results support the idea that syntactic processes of phrase structure building are in place relative early during language development, but they are less automatic than those shown by adults. To address the question of age-related changes, Hahne, Eckstein, and Friederici (2004) presented sentences involving the same phrase structure violation embedded in more complex syntactic structures (*Das Eis wurde im __ gegessen [The ice cream was in-the __ eaten) to children between 6 and 13 years. An adult-like ELAN (100-300 ms) was only observed in the 13-year-old children, whereas younger children showed a delayed bilaterally-distributed anterior negativity (400-600 ms). The bilateral distribution was hypothesised to reflect the involvement of prosodic processing (generally expressed in an early negativity in the right hemisphere, ERAN), which is thought to support syntactic processing, especially during language development. With respect to the P600 component, this was present in children of all age groups, although it had a delayed onset in 6-year-old children (1250-1500), that was observed to decrease with age.

Rispens et al. (2006) have investigated the presence and latency of the P600 component in response to subject-verb agreement violations in Dutch-speaking adults with DD (same stimuli of the behavioural experiment described in Section 1.2.2.2.2). Despite the absence of differences between adults with dyslexia and controls in judging

the grammaticality of the sentences, the ERP data revealed subtle differences between groups, particularly related to latency (the P600 tended to peak later in the dyslexic group compared to the control group) and lateralization (for participants with dyslexia the P600 was less strong in the left hemisphere compared to the midline and right posterior region, while there were no differences between the presence of the P600 in the three areas in the controls).

CHAPTER 3: METHODS

The aim of the present study was to compare the behavioral correlates and the event-related potential (ERP) correlates of implicit morpho- syntactic processing in Kannada language in children with DD and typically developing children.

3.1 Participants

The study included two groups- Group 1 with the typically developing children (TDC) in the age range of 8-10 years, group 2 included children with dyslexia in the age range of 8-10 years. Thirty TDC and 15 children with dyslexia were selected for the study. Children with dyslexia were selected based on the diagnosis given by Speech Language Pathologists and Clinical Psychologists. The following inclusionary criteria were followed for selecting the children for the present study:

- All the children were screened using the WHO ten disability checklist (cited in Singhi, Kumar, Prabhjot& Kumar, 2007) and Developmental screening test (Bharath Raj, 1972) to rule out any sensory, motor, behavioral, intellectual deficits or obvious neurological deficits
- Native language of all the participants was Kannada with English as the medium of instruction in school.
- All the children underwentaudiological screening including pure tone audiometry and Immittance audiometry (GSI Tympstar) to confirm normal hearing sensitivity.
- There were no symptoms of otological, psychological and neurological disorders.

3.2 Instrumentation

The following instruments were used to carry out the study:

- A calibrated two-channel Madsen Orbiter-922 clinical audiometer (version 2) with TDH-39 headphones and Radio ear B-71 bone vibrator to establish air conduction and bone conduction pure tone thresholds respectively.
- Compumedics Neuroscan instrument with Scan^{TM}4.5module along with Quick Cap, Model C190 for recording of cortical evoked event related potentials (ELAN& P600) and Stimuli preparation and presentation was done through E-Prime software 2.0 (SP2) version (2.0.10.356) for both behavioral and ERP measures.

3.3 Preparation of stimuli

A list of 120 sentences was prepared which included 60 canonical and 60 violated sentences. The sentence length was kept constant for all the sentences at 3 words in a sentence. The violated sentences had a grammatical error in terms of subject-verb disagreement at the last word. The erroneous words used in the violated sentences accounted to the frequency spectrum of the vocabulary (occurrence of words in the vocabulary i.e., high frequent words were chosen from the vocabulary which means words which frequently occurred in their vocabulary)of 8-10 year old children. This stimuli list was given to three experienced judges (Speech-Language Pathologists-SLPs) for linguistic familiarity rating on a three- point scale of 'highly familiar', 'familiar', and 'unfamiliar'. Out of the 120 sentences 60 sentences which were rated as 'highly familiar' or 'familiar' by at least two out of the three judges were selected. Of the 60 sentences chosen, 30 were violated sentences and 30 were canonical sentences (details in Appendix-1). The selected 60 sentences were spoken by three male speakers which were audio recorded. The audio samples were given for goodness rating to three SLPs. The audio samples were rated on a 0-5 rating scale, '5'indicating the higher quality and '0'indicating the poorest. The ratings were done by considering the parameters:

intelligibility, clarity, loudness, naturalness and the overall quality of the audio sample. The audio sample from the three speakers which got the highest rating was selected as the final stimuli.

3.4 Stimuli presentation

Two tasks were considered for the study.

- » Behavioral task
- » ERP measure

For the behavioral task, the 60 recorded sentences were presented through E-Prime software for measuring the response times (RTs) and accuracy of responses. A practice session with 10 trial stimuli (5 violated& 5 canonical sentences) which were not included as final test stimuli, were given to familiarize the participants with the instructions and task. Stimuli sentences in each list were randomized and presented once.

For the ERP recording, the stimuli were presented using E-Prime software. The list consisted of 60sentences. All the violated and canonical sentences were presented in random order during the ERP recording. The inter stimulus interval between any two sentences in a list was 3000 ms. Trigger values were specified at the target word of every sentence. The stimuli were presented once binaurally at 65 dB SPL using ER-3A insert earphones.

3.5 Procedure

Test environment

The study was conducted in a quiet environment, in an air conditioned sound treated room. The noise levels were within permissible limits as per ANSI S3.1-1999, R2013.

The following procedure was carried out to record the responses during behavioral and ERP task in both TDC and children with Dyslexia.

Behavioral task

All the participants were tested individually in a quiet room. The recorded stimuli which was prepared for ERP measure was also used for the behavioral task. The stimuli (Appendix 1) were presented using insert earphones ER-3A. The participants were instructed on a grammaticality judgment task (to judge whether a sentence is grammatically correct or incorrect) as follows: "You will hear sentences. It may be grammatically correct (canonical) or grammatically incorrect (violated) sentences. You have to press '1' for a grammatically correct sentence and '0' for grammatically incorrect sentence in the keyboard as soon as you hear the stimuli". Reaction times (time taken to respond after presentation of stimuli) in milliseconds were recorded and stored in the computer and error rates were calculated.

A '+' sign appeared on the screen for 300 ms before the stimuli was presented. This would help the participant to be vigilant for the upcoming stimuli. The target sentence was then presented while the screen remained blank and remained so for the next 4000ms or till the participant responded, whichever occurred first. If the participant failed to respond to a target by 4000ms, that item was recorded as no response. The stimuli were also presented in a random order to avoid order effect.

Response analysis of measures on behavioral task

The responses of the participants were divided into 'hits' ('yes' to a grammatical canonical sentence) and 'correct rejections' ('no' to a violated). The percentages correct on each condition for each group were calculated. In behavioral task, reaction time was measured in milliseconds (ms). All wrong responses and those responses which exceeded

the 4000ms frame duration were eliminated from the data analysis for reaction time measure. Accuracy was calculated for both canonical and violated sentences. A score of '1'was provided for each correct response and '0' for wrong/ absent response. The responses of the participants were divided into 'hits' ('yes' to a grammatical canonical sentence) and 'correct rejections' ('no' to a violated). The behavioral data was coded and tabulated and then subjected to statistical analysis**.**

ERP measure

The cortical event related potentials were recorded using Compumedics Neuroscan system (Scan 4.5). The participants were seated comfortably in a reclining chair. The Quick Cap consisting of 64 sintered silver chloride electrodes was used for recording evoked potentials. The event related potential was recorded from 24 electrode sites of 10-20 system: F3, Fz, F4, P3, Pz, P4, Oz, Cz, FT7, FT8, Fz, FPz, FP1, FP2, FC3, FCz, FC4, TP7, TP8, CP3, CPz, CP4, M1 and M2.Linked mastoid was used as a reference/ active electrode. Linked mastoid was used as a reference/ active electrode. An electrode site between FPz and Fz was used as ground electrode. The scalp electrode distribution was considered based on a review of previous research on functional organization and temporal dynamics for language processing (cited in Royle et al., 2014). The electrode impedance was lesser than 5kΩ. The participants were shown a cartoon video while placing the electrodes to distract their attention and facilitate electrode placement. A blunt needle was used to clean the electrode site. Quick Gel™ filled up in the syringe was used as conduction gel to bridge the scalp with the electrode surface.

A continuous EEG data was recorded and digitized at a sampling rate of 1000 Hz. The data was low pass filtered at 100 Hz, and high passing 1 Hz. The time window of 1800ms with a pre stimulus interval of -200ms was considered for online averaging (was kept as a window for observation and did not affect our recording). Notch filter was set to

off. The corresponding trigger values as given in E-Prime software was entered such that the responses recorded will be time locked with the stimulus given. To maintain the attention of the participants, they were instructed to press the button no.1 on keyboard if they hear correct sentence and to press the number '0' on a keyboard if they hear incorrect sentence. Two recordings were obtained to check for the replicability of the waveforms. The total duration of the testing was approximately one hour per participant.

Offline analysis of ERP waveforms

The electrophysiological signals were digitalized at the rate of 1000 Hz and offline bandpass zero-phase filtered (1-30 Hz). Automatic rejection criterion was applied to all the electrodes and sections exceeding +200μV were excluded. All ERPs were time-locked to the onset of the target word of the sentence. Continuous filtered EEG waveform was epoched from -200 to 1800 ms and was baseline corrected. Finally, the epoched files were averaged to obtain different waveforms for canonical and violated words of the sentences.

Response analysis of ERP waveforms

The waveforms were marked by two experienced Audiologists (minimum of ten years of experience) apart from the research officer. The negativity between 100 msto 300ms was marked as the ELAN peak. The peak amplitude and latency of ELAN for 24 channels was calculated and tabulated for further statistical analysis. Similarly, the positivity between 600 msto 1000ms was marked as the P600peak. The peak amplitude and latency of P600 for 24 channels was calculated and tabulated for further statistical analysis.

Statistical analyses

Appropriate statistical analysis was done using Statistical Package for the Social Sciences (SPSS) version 17.0 software. The following statistical analyses were used to analyze the data,

- Descriptive statistics (Mean, Standard deviation, and Median) were obtained for all the quantitative data – Behavioural and ERP data.
- The data was subjected to tests of normality.
- Shapiro-Wilk's test was done to check for normality. The results indicated that the data did not follow a normal distribution ($p<0.05$). Hence, non-parametric tests such as Mann-Whitney U test and Wilcoxon's signed rank tests were used to check for significant difference between TDC and DD across various parameters.

CHAPTER 4: Results

The aim of the present study was to compare the behavioral correlates with event related potential (ERP) correlates of implicit morpho-syntactic processing in children with DD in comparison to typically developing children (TDC). The participants included were 30 typically developing children divided into two groups: 8-10 years old and 15 children with dyslexia in the age range of 8-10 years. Both behavioral and ERP data were analyzed. The behavioral data included reaction time (RT) in milliseconds and accuracy for canonical and violated sentences. The ERP data included latency (in milliseconds) and amplitude (in micro volts) of the ELAN at 24 different channels.

The quantitative and qualitative data obtained were subjected to tests of normality and depending upon results of normality parametric and non-parametric statistical analyses were employed using Statistical Package for Social Sciences (SPSS) version 17. Descriptive statistics were obtained for all the quantitative data. To check the normality, obtained quantitative data was subjected to normality by using the Shapiro-Wilk's test. The data did not follow a normal distribution ($p<0.05$) in Shapiro-Wilks test. Hence, these parameters were subjected to a non-parametric test using Mann-Whitney U test and Wilcoxon's signed rank test to find out the significant difference. The results of the present study are discussed under the following sections:

4.1 Comparison of performance of typically developing children (TDC) and children with dyslexia on behavioral measures

4.2 Comparison of performance of typically developing children (TDC) and children with dyslexia on ERP (ELAN and P600) measures

4.3 Correlation of behavioural measures and ERP (ELAN and P600) measures in typically developing children (TDC) and children with dyslexia.

4.4 Sub typing of dyslexia using ERP (ELAN and P600) measures

4.1. Comparison of performance of typically developing children (TDC) and children with dyslexia on behavioral measures

The results of performance of TDC and children with dyslexia for canonical and violated sentences were analyzed for reaction time (RT) and accuracy measures. On behavioral measure, all inaccurate responses and those responses, which exceeded the 4000 ms, duration were eliminated from the data analysis for reaction time measures, however these responses were considered for accuracy measure. The reaction time was tabulated in milliseconds (ms).

The reaction time (in ms) was analyzed for canonical and violated sentences in the two groups i.e., Group 1 (8-10 year) of typically developing children (TDC) and Group 2 of children with DD (DD). The mean and standard deviation (SD) for performances of two groups of TDC and children with DD on canonical and violated sentences for reaction time and accuracy were analyzed using descriptive statistics and it is shown in Table 4.1 and Figures 4.1 a and 4.1b.

Table 4.1

Mean and SD for reaction time and accuracy measure on grammaticality judgment task in TDC and DD

Sentences	Reaction time (in ms)				Accuracy			
	TDC		DD		TDC		DD	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Canonical	736.92	224.64	1135.77	368.33	71.52	25.18	85.30	19.59
Violated	802.18	245.13	1065.71	509.87	70.20	18.95	71.74	33.48

Analysis of results as indicated in table 4.1 indicated that the mean reaction time was longer for both canonical and violated sentences in children with DD when compared to TDC [canonical and violated sentences]. Wilcoxon signed ranks test resultsindicated that there was a significant difference on reaction time measure between TDC and DD for both canonical ($|z|$=-4.75, $p<0.001$) and violated ($|z|$=-1.77, $p<0.05$) sentences. The findings indicated that on reaction time measure, children with DD took significantly longer time when compared to TDC on both canonical and violated sentences. On similar lines, data on accuracy measures were analyzed and the results indicated that children with DD showed poorer performance when compared to children with TDC on violated sentences. The typically developing children showed greater accurate scores [canonical and violated sentences] when compared to children with dyslexia [canonical and violated sentences] (see table 4.1) for both canonical and violated sentences. Further, Wilcoxon Signed Rankstestresults indicated that there was no significant difference on accuracy measure between TDC and DD for both canonical ($|z|$=-1.44, $p<0.05$) and violated ($|z|$=-0.85, $p<0.05$) sentences

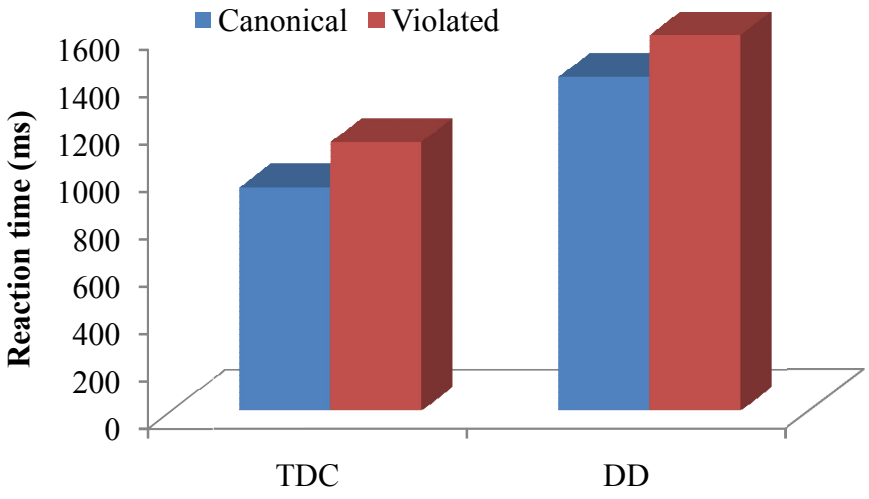

*Figure 4.1a.*Means of reaction time on the grammaticality judgment task in TDC and DD

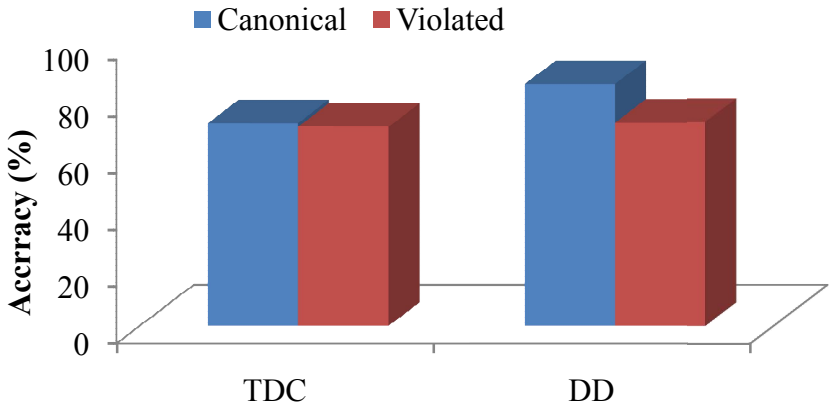

Figure 4.1b Means of percentage correct (and standard deviations) in the grammaticality judgment task in TDC and DD

To summarize, the behavioral data results indicated that there was significant difference between DD and LD for reaction time measure for both canonical and violated sentences. However, there was no significant difference between TDC and children with DD on accuracy measures for both canonical sentences and violated sentences. Figure 4.1a shows the results of reaction time on the grammaticality judgment task in TDC and DD. Figure 4.1b shows the results of reaction time on the grammaticality judgment task in TDC and DD. The results thus suggest that children with LD took longer time to respond to grammaticality judgment when compared to TDC as indicated by the results of reaction time measure. However, the children with DD were able to accurately respond to grammaticality judgment task in similar to TDC.

4.2. Performance of typically developing children (TDC) and children with dyslexia on ERP (ELAN & P600) measures

The ERP measures included the peak latency (in milliseconds) and peak amplitude (in microvolts) of ELAN and P600 potentials. The peak latency and amplitude were measured across 24 different channels in 30 typically developing children and 15

children with dyslexia. The peak latency and amplitude for TDC and DD was compared across different channels separately.

4.2.1. Performance of typically developing children (TDC) on ERP (ELAN & P600) amplitude measures

The peak latency and amplitude of ELAN and P600 was analyzed in thirty TDC of 8-10 year old. Table 4.2 shows amplitude measure of ELAN and P600 for TDC. Figure 4.2 shows grand averaged ERP waveforms of TDC at different channels respectively.

Table 4.2

Median and IQR for ELAN and P600 amplitude in TDC

Electrode sites	ELAN Amplitude (in μV) TDC		P600 Amplitude (in μV) TDC	
	Median	*IQR*	*Median*	*IQR*
CP3	-3.08	4.88	1.85	3.86
CP4	-3.90	5.75	3.75	3.99
CPz	-3.86	3.42	2.08	3.81
Cz	-5.53	6.54	7.28	8.74
F3	-4.90	7.84	6.69	15.36
F4	-5.80	4.63	5.88	10.96
F7	-5.07	8.66	10.35	25.76
F8	-4.42	19.63	9.63	6.11
FC3	-4.86	5.72	6.07	9.72
FC4	-3.29	8.73	4.65	9.09
FCz	-6.30	6.64	9.32	9.33
FP1	-8.56	10.37	11.01	11.62
FP2	-9.58	9.58	14.65	16.24
FT7	-5.07	11.52	6.60	10.80
FT8	-8.02	6.25	1.55	7.22
Fz	-3.19	10.32	8.81	12.81
P3	-3.55	4.22	0.75	3.06
P4	-3.28	8.35	3.39	5.67
Pz	-2.84	1.40	2.98	5.31
TP7	-2.67	8.00	2.57	5.81
TP8	-3.78	2.19	-	1.45

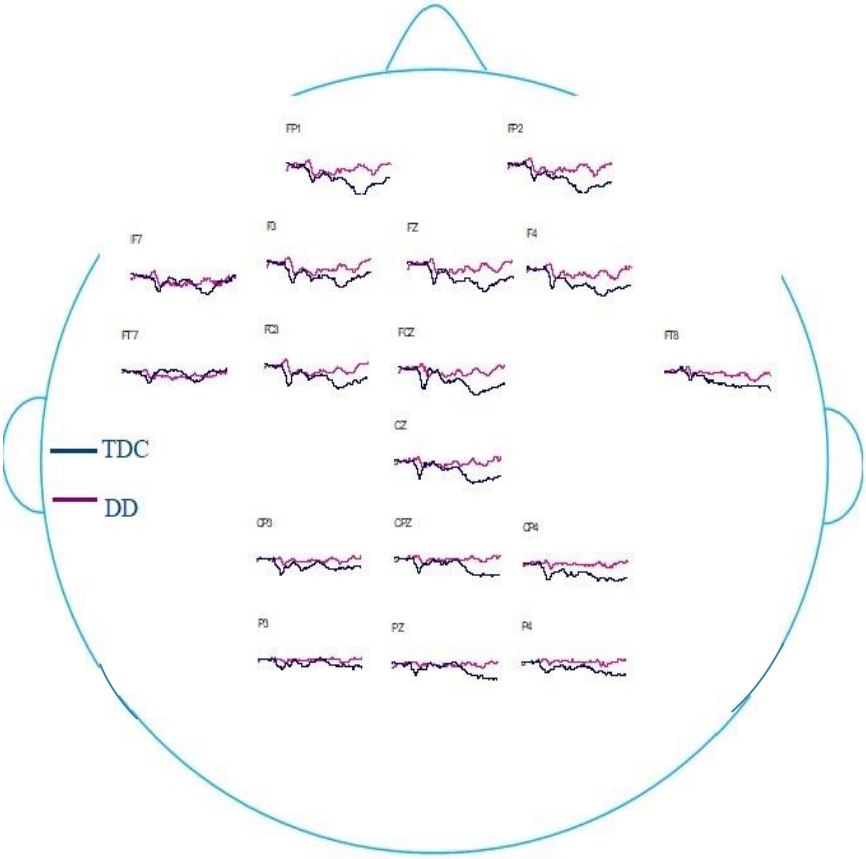

Figure 4.2 Grand averaged ERP waveforms of TDC and children with DD.

Amplitude of ELAN and P600 in TDC

The results are analyzed based on the regions; posterior regions, (P3/4, TP7/8, Oz, FT7/FT8) and anterior regions (F3/4, F7/8, FC3/4, FCz, FP1/FP2), middle (CP3/ CP4, Pz, CPz, Cz, Fz).

The results from Table 4.2 indicate that Median peak amplitude of ELAN in TDC indicated greater peak amplitude for channels such as the fronto-temporal channels, Fronto-parietal followed by Centro-parietal, temporal and Parietal channels. Wilcoxon Signed Rank test was done to compare the performance of children between channels in TDC. Analysis of results revealed that for ELAN amplitude in TDC, there was a significant difference between channels indicted in the table 4.3

Table 4.3

Results of Wilcoxon test across the channels for ELAN Amplitude in typically developing Children.

Wilcoxon test results for ELAN Amplitude in typically developing children

/z/ scores

Electrode sites	CP3	CP4	CPz	Cz	F3	F4	FC3	FC4	FCz	FP1	FP2	FT7	FT8	Fz	P3	P4	Pz	TP7	TP8
CP3	-	1.47	0.51	1.19	2.32*	2.66*	2.04*	2.35*	2.44*	2.79**	2.85**	1.07	1.70	3.18**	1.97	0.34	1.02	0.09	2.54*
CP4	1.47	-	1.30	0.56	1.98	2.41*	1.30	1.91	1.98	2.41*	2.35*	0.51	1.25	3.01**	1.56	0.96	0.11	0.34	2.41*
CPz	0.51	1.30	-	2.21*	1.93	2.85**	0.90	2.04*	2.44*	2.60*	2.91**	0.85	1.81	2.66*	2.84*	0.51	0.28	0.51	2.66*
Cz	1.19	0.56	2.21*	-	1.47	2.29*	0.62	1.41	1.53	2.16*	2.10*	-	1.02	2.55*	2.55*	2.44*	1.19	1.76	2.85**
F3	2.32*	1.98	1.93	1.47	-	0.84	1.47	0.22	0.68	1.91	1.85	0.85	0.45	0.79	2.41*	2.10[a]	1.42	1.97	2.98**
F4	2.68*	2.41*	2.85**	2.29*	0.84	-	1.53	1.01	1.16	1.64	2.06*	0.53	0.15	2.15*	2.41*	2.29*	1.30	1.81	2.74**
FC3	2.04*	1.30	0.90	0.62	1.47	1.53	-	1.22	0.90	2.35*	2.22*	0.11	0.85	2.21*	2.72**	2.44*	1.42	1.93	3.12**
FC4	2.35*	1.91	2.04*	1.41	0.22	1.01	1.22	-	0.40	1.50	1.64	0.40	0.59	2.55*	2.27*	2.22*	1.02	1.35	2.66**
FCz	2.42*	1.98	2.44*	1.53	0.68	1.16	0.90	0.40	-	1.85	2.48*	0.68	0.96	1.98	3.06**	2.95**	1.87	2.27*	3.06**
FP1	2.79**	2.41*	2.60*	2.16*	1.91	1.64	2.35*	1.50	1.85	-	1.09	2.10*	0.59	1.19	3.40**	3.29**	2.89**	3.12**	3.29**
FP2	2.85**	2.35*	2.91**	2.10*	0.85	2.06*	2.22*	1.64	2.48*	1.09	-	2.04*	1.85	1.76	3.29**	3.06**	2.61*	2.72**	3.06**
FT7	1.07	0.51	0.85	-	0.85	0.53	0.11	0.40	0.68	2.10*	2.04*	-	0.51	0.85	1.99	1.41	0.79	2.43*	2.84**
FT8	1.70	1.25	1.81	1.02	0.45	0.15	0.85	0.59	0.96	0.59	1.85	0.51	-	2.78*	1.09	0.66	0.68	0.03	2.54*
Fz	2.10*	1.53	1.53	0.68	1.19	1.78	0.17	0.78	0.34	2.29*	2.41*	0.28	0.62	-	2.89*	2.98**	2.15*	2.44*	3.06**
P3	0.80	0.62	0.97	1.42	1.95	2.40*	1.60	2.04*	2.40*	2.93**	2.75*	1.06	2.57*	2.89**	-	1.56	2.84**	0.84	1.17
P4	0.09	0.40	0.22	0.78	1.22	2.83**	0.84	1.43	1.41	1.99	2.34*	1.09	1.72	2.95**	1.55	-	0.90	0.34	2.06*
Pz	0.96	1.42	1.07	2.21*	2.15*	2.91**	1.87	1.66	2.38*	2.54*	2.98**	1.70	2.84**	2.15*	2.84	0.90	-	0.53	2.38*
TP7	0.29	0.88	0.65	0.77	0.53	1.00	0.53	1.40	0.77	1.26	1.12	1.59	0.65	2.44*	0.84	0.34	0.53	-	2.07*
TP8	0.86	0.25	0.56	0.86	1.47	1.47	1.17	0.96	1.58	2.66**	2.07*	1.88	1.98	3.06**	1.17	2.06*	2.38*	2.07*	-

* p<0.05. ** p<0.01

Analysis of results indicated that in children with TDC, the amplitude of the frontal regions showed greater ELAN effect when compared to central, temporal and parietal regions. Table 4.2 also shows the median peak amplitude and interquartile range for P600 in TDC. The results indicated that the median peak amplitude of P600 in TDC indicated greater peak amplitudes for channels such as the fronto-parietal, frontal, central, parietal and centroparietal channels.

The findings indicate largest amplitude over anterior regions of the scalp in response to morpho-syntactic violations of the sentences. Children in the age range of 8-10years of age as in the present study, show this ERP effect in response to syntactic phrase structure violations wherein syntactically anomalous sentences show greater frontal distributed positive effect. The TDC controls showed a different distribution of the

P600 component in response to the sentence violations than the ELAN component with greater amplitude distributed in left anterior regions than the posterior regions. Wilcoxon Signed Rank test was done to compare the performance of children between channels in TDC. Analysis of results revealed that for P600 amplitude in TDC, there was a significant difference between channels indicted in the table 4.4

Table 4.4

Results of Wilcoxon test across the channels for P600 Amplitude in typically developing Children.

Wilcoxon test results for P600 Amplitude in typically developing children

/z/ scores

Electrode sites	CP3	CP4	CPz	Cz	F3	F4	FC3	FC4	FCz	FP1	FP2	FT7	FT8	Fz	P3	P4	Pz	TP7	TP8
CP3	-	0.85	0.85	2.49*	2.66*	2.49*	3.12**	2.15*	3.35**	3.35**	3.12*	1.22	0.72	3.18**	1.97	0.34	1.02	0.09	2.54*
CP4	0.85	-	0.40	1.91	2.21*	2.41*	1.93	2.10*	2.44*	3.01**	3.12*	1.53	1.60	3.01**	1.76	0.96	0.11	0.34	2.41*
CPz	0.85	0.40	-	2.41*	2.44*	2.22*	2.38*	2.04*	2.72*	3.23**	3.01*	1.19	0.84	2.66*	2.04*	0.51	0.28	0.51	2.66*
Cz	2.49*	1.91	2.41*	-	1.19	1.09	0.62	0.40	2.66*	2.55*	2.89*	0.28	2.16*	2.55*	2.55*	2.44*	1.19	1.76	2.85*
F3	2.66*	2.21*	2.44*	1.19	-	0.85	0.96	1.42	0.34	1.93	1.76	0.09	2.54*	0.79	2.41*	2.10	1.42	1.97	2.98**
F4	2.49*	2.41*	2.22*	1.09	0.85	-	0.05	2.06*	0.28	2.10*	2.15*	0.22	2.41*	2.15*	2.41*	2.29*	1.30	1.81	2.74*
FC3	3.12**	1.93	2.38*	0.62	0.96	0.05	-	0.96	0.96	2.55*	2.38*	0.17	2.32*	2.21*	2.72*	2.44*	1.42	1.93	3.12**
FC4	2.15*	2.10*	2.04*	0.40	1.42	2.06*	0.96	-	1.25	2.66*	2.84*	0.47	2.27*	2.55*	2.27*	2.22*	1.02	1.35	2.66*
FCz	3.35**	2.44*	2.72*	2.66*	0.34	0.28	0.96	1.25	-	1.76	2.38*	0.45	2.66*	1.98	3.06**	2.95*	1.87	2.27*	3.06**
FP1	3.35**	3.01**	3.23**	2.55*	1.93	2.10*	2.55*	2.66*	1.76	-	0.34	1.30	2.95**	1.19	3.40**	3.29**	2.89*	3.12**	3.29**
FP2	3.12**	3.12**	3.01**	2.89*	1.76	2.15*	2.38*	2.84**	2.38*	0.34	-	1.81	2.89*	1.76	3.29**	3.06**	2.61*	2.72*	3.06**
FT7	1.22	1.53	1.19	0.28	0.09	0.22	0.17	0.47	0.45	1.30	1.81	-	1.85	0.85	1.99	1.41	0.79	2.43*	2.84*
FT8	0.72	1.60	0.84	2.16*	2.54*	2.41*	2.32*	2.27*	2.66*	2.95**	2.89**	1.85	-	2.78	1.09	0.66	0.68	0.03	2.54*
Fz	3.18**	3.01**	2.66*	2.55*	0.79	2.15*	2.21*	2.55*	1.98	1.19	1.76	0.85	2.78*	-	2.89*	2.95*	2.15*	2.44*	3.06*
P3	1.97	1.76	2.04*	2.55*	2.41*	2.41*	2.72*	2.27*	3.06**	3.40**	3.29**	1.99	1.09	2.89*	-	1.56	2.84*	0.80	1.17
P4	0.34	0.96	0.51	2.44*	2.10*	2.29*	2.44*	2.22*	2.95**	3.29**	3.06**	1.41	0.66	2.95**	1.56	-	0.90	0.34	2.06*
Pz	1.02	0.11	0.28	1.19	1.42	1.30	1.42	1.02	1.87	2.89**	2.61*	0.79	0.68	2.15*	2.84*	0.90	-	0.53	2.38*
TP7	0.09	0.34	0.51	1.76	1.97	1.81	1.93	1.35	2.27*	3.12*	2.72*	2.43*	0.03	2.44*	0.80	0.34	0.53	-	2.07*
TP8	2.54*	2.41*	2.66*	2.85*	2.98**	2.74*	3.12**	2.66*	3.06**	3.29**	3.06**	2.84*	2.54*	3.06**	1.17	2.06*	2.38*	2.07*	-

* p<0.05. ** p<0.01

Latency of ELAN and P600 in TDC

Table 4.5 shows the median and interquartile range values for latency measure of ELAN and P600 in TDC. The analysis of results indicated that the median latency for ELAN in TDC showed longer latency in fronto-parietal and frontal channels when compared to centro-parietal, parietal and temporal channels. The results also indicated that latency for P600 showed longer latency in fronto-parietal, frontal when compared to centro-parietal, fronto-temporal, parietal and temporo-parietal channels.

Table 4.5

Median and IQR for ELAN and P600 latency (in ms) in TDC

Electrode sites	ELAN Latency (in ms) TDC		P600 Latency (in ms) TDC	
	Median	*IQR*	*Median*	*IQR*
CP3	172.00	52.00	653.00	176.25
CP4	166.00	39.00	694.00	124.00
CPz	181.00	62.00	675.00	222.50
Cz	178.00	49.00	704.00	212.75
F3	192.00	46.50	639.00	180.50
F4	176.50	32.00	741.00	345.50
F7	180.00	37.00	699.00	130.00
F8	172.50	56.00	694.50	187.50
FC3	186.00	54.00	698.00	192.75
FC4	169.50	38.50	698.00	351.25
FCz	196.00	62.50	636.00	134.00
FP1	201.00	60.00	704.00	249.75
FP2	198.00	61.00	698.00	177.00
FT7	169.00	47.00	630.00	104.75
FT8	165.00	17.00	584.00	638.00
Fz	168.00	30.00	739.00	137.25
P3	176.00	40.00	624.00	660.50
P4	172.00	22.25	655.00	295.00
Pz	177.00	30.00	662.00	237.25
TP7	161.00	14.50	546.00	694.00
TP8	159.00	48.75	-	629.50

Table 4.5 shows median and interquartile range for ELAN and P600 latency (in ms) in TDC. The analysis of results as indicated in table 4.5 revealed that the median peak latency was found across channels and ranged between 159.00 to 201.00 ms. The latency ranged from lowest to highest among channels centro-parietal channels (CP3, CP4), Pz, Cz, frontal channels (F3, F7, F8), parietal channels (P4, Pz) and fronto-parieatal channels (FP1, FP2). The results also revealed shorter latency for channels in the left hemisphere compared to the right hemisphere for ELAN component. Further analysis of results in table 4.3 of median peak latency for P600 indicated that the latency ranged from 546.00 to 741.00 across channels –frontal, parietal, centro-parietal, occipital, temporo-

parietal and fronto-central channels. The results also revealed shorter latency for channels in the left hemisphere compared to the right hemisphere for P600 component. The results of amplitude and latency measures for ELAN indicated a greater ELAN effect for anterior regions than the posterior regions in TDC. A greater positivity effect was indicated in the P600 component distributed in the anterior regions when compared to posterior regions. Wilcoxon Signed Rank test was done to compare the performance of children between channels in TDC. Analysis of results revealed that for ELAN and P600 Latency in TDC, there was a significant difference between channels indicted in the tables 4.6 and 4.7 respectively.

Table 4.6

Results of Wilcoxon test across the channels for ELAN latency in typically developing Children.

Wilcoxon test across the channels for ELAN latency in typically developing Children

/z/ scores

Electrode sites	CP3	CP4	CPz	Cz	F3	F4	FC3	FC4	FCz	FP1	FP2	FT7	FT8	Fz	P3	P4	Pz	TP7	TP8
CP3	-	0.34	1.70	1.39	2.50*	1.21	2.46*	0.72	2.13*	2.35*	2.35*	0.80	0.03	0.85	0.83	1.11	2.41*	0.88	0.66
CP4	0.34	-	0.54	0.79	1.44	1.06	1.04	1.67	1.72	1.82	1.80	0.85	0.37	0.22	0.35	0.47	0.75	0.59	-
CPz	1.70	0.54	-	0.51	0.88	0.28	0.66	0.19	1.64	1.57	1.51	0.08	0.82	0.47	0.17	0.25	1.28	0.77	0.76
Cz	1.39	0.79	0.51	-	1.92	1.15	1.33	0.59	2.01*	1.99	2.07*	0.56	0.51	0.15	0.40	0.53	1.19	0.77	0.96
F3	2.50*	1.44	0.88	1.92	-	0.84	0.09	1.08	0.19	1.39	1.66	1.18	1.59	1.85	1.06	0.15	0.25	1.59	1.12
F4	1.21	1.06	0.28	1.15	0.84	-	0.71	1.02	0.21	1.99	1.92	0.06	1.12	0.75	0.04	0.48	0.12	2.07*	1.47
FC3	2.46*	1.04	0.66	1.33	0.71	0.09	-	0.74	0.85	1.50	1.93	0.90	1.30	0.18	0.71	0.15	0.76	1.26	1.17
FC4	0.72	1.67	0.19	0.59	1.08	1.02	0.74	-	0.53	2.04*	1.96	0.73	0.37	0.03	0.15	0.10	0.31	0.70	0.77
FCz	2.13*	1.72	1.64	2.01*	0.19	0.21	0.85	0.53	-	0.76	0.87	1.05	1.44	1.02	0.80	0.14	0.28	1.24	1.18
FP1	2.35*	1.82	1.57	1.99	1.39	1.99	1.50	2.04*	0.76	-	0.14	2.15*	3.10**	2.13*	1.02	0.66	0.25	2.52*	2.38*
FP2	2.35*	1.80	1.51	2.07*	1.66	1.92	1.93	1.96	0.87	0.14	-	1.71	2.89**	2.04*	1.06	0.52	0.15	2.36*	2.19*
FT7	0.80	0.85	0.08	0.56	1.18	0.06	0.90	0.73	1.05	2.15*	1.71	-	1.19	0.17	0.04	0.34	0.99	1.71	1.88
FT8	0.03	0.37	0.82	0.51	1.59	1.12	1.30	0.37	1.44	3.10**	2.89**	1.19	-	0.51	0.93	0.94	1.42	0.56	1.30
Fz	0.85	0.22	0.47	0.15	1.85	0.75	0.18	0.03	1.02	2.13*	2.04*	0.17	0.51	-	0.22	0.72	1.33	1.60	0.53
P3	0.83	0.35	0.17	0.40	1.06	0.04	0.71	0.15	0.80	1.02	1.06	0.04	0.93	0.22	-	0.13	0.84	1.01	1.60
P4	1.11	0.47	0.25	0.53	0.15	0.48	0.15	0.10	0.14	0.66	0.52	0.34	0.94	0.72	0.13	-	0.27	0.41	0.53
Pz	2.41*	0.75	1.28	1.19	0.25	0.12	076	0.31	0.28	0.25	0.15	0.99	1.42	1.33	0.84	0.27	-	0.53	0.86
TP7	0.88	0.59	0.77	0.77	1.59	2.07*	1.26	0.70	1.24	2.52*	0.36	1.71	0.56	1.60	1.01	0.41	0.53	-	0.50
TP8	0.66	-	0.76	0.96	1.12	1.47	1.17	0.77	1.18	2.38*	0.19	1.88	1.30	0.53	1.60	0.53	0.86	0.50	-

* p<0.05. ** p<0.01

Table 4.7

Results of Wilcoxon test across the channels for P600 latency in typically developing Children.

Wilcoxon test across the channels for P600 Latency in typically developing Children

/z/ scores

Electrode sites	CP3	CP4	CPz	Cz	F3	F4	FC3	FC4	FCz	FP1	FP2	FT7	FT8	Fz	P3	P4	Pz	TP7	TP8
CP3	-	0.62	0.38	0.65	0.72	0.56	0.84	0.14	0.54	1.59	1.19	1.28	2.48*	1.91	1.53	1.16	1.64	2.10*	3.23**
CP4	0.62	-	0.62	0.43	0.14	0.56	0.25	0.53	0.08	1.53	0.90	1.60	2.60*	0.93	2.66*	1.64	0.40	2.10*	3.23**
CPz	0.38	0.62	-	0.97	0.56	1.28	0.79	0.10	0.03	1.70	1.19	1.30	2.22*	1.98	1.22	0.34	0.42	2.04*	3.17**
Cz	0.65	0.43	0.97	-	0.22	0.80	0.02	0.15	0.75	1.53	0.69	1.35	2.66*	1.19	2.13*	1.81	0.54	2.10*	3.23**
F3	0.72	0.14	0.56	0.22	-	-	0.68	0.05	0.56	1.39	0.28	2.34*	1.97	0.69	2.22*	0.82	0.45	2.57*	3.17**
F4	0.56	0.56	1.28	0.80	-	-	0.54	0.47	0.34	1.25	0.31	1.22	2.13*	0.34	2.27*	1.53	0.79	1.59	2.98**
FC3	0.84	0.25	0.79	0.02	0.68	0.54	-	0.97	0.79	0.42	0.22	1.98	2.55*	0.93	2.44*	1.67	1.07	2.72*	3.35**
FC4	0.14	0.53	0.10	0.15	0.05	0.47	0.97	-	0.39	1.16	0.40	1.08	1.78	1.13	1.85	0.59	0.53	1.47	2.90**
FCz	0.54	0.08	0.03	0.75	0.56	0.34	0.79	0.39	-	1.42	1.07	1.09	2.32*	1.85	2.21*	1.64	0.31	1.98	3.23**
FP1	1.59	1.53	1.70	1.53	1.39	1.25	0.42	1.16	1.42	-	0.79	2.38*	2.86*	0.25	2.78*	2.15*	1.70	2.66*	3.35**
FP2	1.19	0.90	1.19	0.69	0.28	0.31	0.22	0.40	1.07	0.79	-	1.96	2.38*	0.80	2.95**	1.81	1.07	2.38*	3.40**
FT7	1.28	1.60	1.30	1.35	2.34*	1.22	1.98	1.08	1.09	2.38*	1.96	-	0.90	2.48*	1.01	0.03	1.59	1.17	2.84**
FT8	2.48*	2.60*	2.22*	2.66*	1.97	2.13*	2.55*	1.78	2.32*	2.86**	2.38*	0.90	-	2.72*	0.15	1.57	2.61*	0.03	2.19*
Fz	1.91	0.93	1.98	1.19	0.69	0.34	0.93	1.13	1.85	0.25	0.80	2.48*	2.72*	-	2.98**	2.10*	1.87	2.72*	3.35**
P3	1.53	2.66*	1.22	2.13*	2.22*	2.27*	2.44*	1.85	2.21*	2.78*	2.95**	1.01	0.15	2.98*	-	1.42	1.41	0.31	2.19*
P4	1.16	1.64	0.34	1.81	0.82	1.53	1.67	0.59	1.64	2.15*	1.81	0.03	1.57	2.10*	1.42	-	1.05	1.02	2.69*
Pz	1.64	0.40	0.42	0.54	0.45	0.79	1.07	0.53	0.31	1.70	1.07	1.59	2.61*	1.87	1.41	1.05	-	2.15*	3.35**
TP7	2.10*	2.10*	2.04*	2.10*	2.57*	1.59	2.72*	1.47	1.98	2.66*	2.38*	1.17	0.03	2.72*	0.31	1.02	2.15*	-	2.66*
TP8	3.23**	3.23**	3.17**	3.23**	3.17**	2.98**	3.35**	2.90**	3.23**	3.35**	3.40**	2.84*	2.19*	3.35**	2.19*	2.69*	3.35**	2.66*	-

* p<0.05. ** p<0.01

4.2.1. Performance of children with dyslexia (DD) on ERP (ELAN & P600) amplitude measures

Amplitude of ELAN and P600 in DD

The peak latency and amplitude of ELAN and P600 was analyzed in children with dyslexia. Table 4.8 shows median amplitude measure of ELAN and P600 for DD at different channels respectively.

Table 4.8

Median and IQR for ELAN and P600 amplitude in DD

Electrode sites	ELAN Amplitude (in μV) DD		P600 Amplitude (in μV) DD	
	Median	*IQR*	*Median*	*IQR*
CP3	-9.68	2.96	-	0.63
CP4	-9.39	4.62	4.63	7.31
CPz	-7.58	8.11	1.99	5.55
Cz	-9.94	10.44	2.49	5.77
F3	-9.55	19.90	2.12	7.02
F4	-7.39	11.79	2.24	6.58
F7	-12.81	26.06	5.36	11.57
F8	-5.71	18.63	7.77	7.04
FC3	-12.38	12.13	3.89	8.10
FC4	-13.24	12.26	-	4.23
FCz	-8.80	10.28	3.06	9.33
FP1	-9.33	19.78	1.67	9.39
FP2	-15.17	12.61	2.36	9.57
FT7	-11.48	11.05	0.91	7.42
FT8	-4.21	-	1.26	7.88
Fz	-11.29	8.44	0.96	5.0
P3	-7.89	4.00	-	7.20
P4	-5.08	23.74	0.30	6.83
Pz	-7.58	15.46	3.93	6.93
TP7	-3.82	-	1.81	3.61
TP8	-2.62	-	0.57	6.89

The results from Table 4.8 indicate that the median peak amplitude of ELAN in DD indicated greater peak amplitude for channels such as the frontal, fronto-central channels, fronto-parietal followed by Centro-parietal, Parietal and temporal channels. Wilcoxon Signed Rank test was done to compare the performance of children between channels in DD. Analysis of results revealed that for ELAN amplitude in DD, there was a significant difference between channels indicted in the table 4.9.

It can be observed from Table 4.8 that, the median peak amplitude of P600 in DD revealed higher peak amplitude in frontal, Centro-parietal followed by fronto central, fronto parietal, parietal and temporal channels.

Wilcoxon Signed Rank test was done to compare the performance of children between channels in DD. Analysis of results revealed that for ELAN and P600 amplitude in DD, there was no significant difference between channels observed as indicted in the table 4.9 and 4.10.

Table 4.9

Results of Wilcoxon test across the channels for ELAN amplitude in DD.

Wilcoxon test across the channels for ELAN amplitude in DD.

	/z/ scores																		
Electrode sites	CP3	CP4	CPz	Cz	F3	F4	FC3	FC4	FCz	FP1	FP2	FT7	FT8	Fz	P3	P4	Pz	TP7	TP8
CP3	-	0.36	1.75	1.34	1.34	-	1.82	1.34	1.34	-	1.34	0.73	1.34	1.36	1.54	1.60	2.31*	1.78	1.33
CP4	0.36	-	1.99	1.46	0.53	-	1.21	1.34	-	0.44	0.53	0.73	0.44	1.01	1.49	1.82	0.86	1.47	1.25
CPz	1.75	1.99	-	0.67	1.09	0.94	2.20*	1.60	1.82	1.06	1.46	1.21	044	0.53	0.97	0.36	0.53	0.86	0.53
Cz	1.34	1.46	0.67	-	1.09	1.09	1.75	1.34	2.02*	1.09	1.09	1.34	-	0.05	0.44	0.44	1.02	0.35	0.61
F3	1.34	0.53	1.09	1.09	-	0.53	0.40	0.44	0.13	1.09	0.53	0.44	-	0.41	0.25	-	0.52	0.26	0.26
F4	-	-	0.94	1.09	0.53	-	1.21	1.06	1.46	0.73	1.48	1.82	-	0.08	0.62	1.34	0.40	0.24	0.31
FC3	1.82	1.21	2.20*	1.75	0.40	1.21	-	0.53	0.52	0.94	0.67	1.82	1.34	1.24	1.51	1.60	0.54	1.15	0.87
FC4	1.34	1.34	1.60	1.34	0.44	1.06	0.53	-	0.44	-	0.44	-	-	0.26	0.05		1.09	0.08	0.17
FCz	1.34	-	1.82	2.02*	0.13	1.46	0.52	0.44	-	-	1.09	0.44	-	0.17	0.38		1.01	0.15	0.31
FP1	-	0.44	1.06	1.09	1.09	0.73	0.94	-	0.13	0.73	0.73	0.44	-	0.96	0.86		0.03	1.33	0.56
FP2	1.34	0.53	1.46	1.09	0.53	1.48	0.67	0.44	1.09	-	-	1.06	-	1.07	1.24	1.34	0.28	0.70	1.06
FT7	0.73	0.73	1.21	1.34	0.44	1.82	1.82	-	0.44	0.44	1.06	-	1.34	0.96	0.53	1.60	0.62	1.17	0.56
FT8	1.34	0.44	0.44	-	-	-	1.34	1.34	-	-	-	1.34	--	0.31	0.26	0.44	0.78	0.26	0.88
Fz	1.60	0.36	2.02	2.02*	-	2.02*	0.10	-	0.67		0.67	-	-	-	0.05	1.34	0.94	0.05	0.08
P3	1.46	1.60	0.13	1.06	1.60	0.73	1.82	1.60	1.60	1.34	1.64	1.06	-	0.05	-	1.34	1.37	0.26	0.35
P4	1.60	1.82	0.36	0.44	-	1.34	1.60	-	-	-	1.32	1.60	-	-	0.88	0.53	0.86	0.08	0.25
Pz	1.82	1.46	1.75	0.53	1.34	0.53	1.60	1.34	1.34	-	1.34	1.06	-	0.94	1.37		-	1.02	0.62
TP7	1.34	1.34	1.60	1.34	-	-		-	-	-	-	-	-	0.05	0.26		1.02	-	0.44
TP8	1.34	1.60	-	1.34	-	0.44	1.60	1.34	1.34	-	0.44	0.44	-	0.08	0.35		0.62	0.44	-

* p<0.05. ** p<0.01

Table 4.10

Results of Wilcoxon test across the channels for P600 amplitude in DD.

Wilcoxon test across the channels for P600 amplitude in DD.

	/z/ scores																		
Electrode sites	CP3	CP4	CPz	Cz	F3	F4	FC3	FC4	FCz	FP1	FP2	FT7	FT8	Fz	P3	P4	Pz	TP7	TP8
CP3	-	2.58*	2.49*	2.31*	1.27	1.86	1.95	0.56	1.64	1.33	1.86	1.33	1.15	1.36	1.54	1.47	2.31*	1.78	1.33
CP4	2.58*	-	1.17	1.64	1.43	0.73	0.53	2.22*	1.92	0.59	0.03	0.94	0.86	1.01	1.49	1.24	0.86	1.47	1.25
CPz	2.49*	1.17	-	0.71	0.15	0.17	0.23	0.94	0.70	-	0.73	0.44	0.26	0.53	0.97	0.45	0.53	0.86	0.53
Cz	2.31*	1.64	0.71	-	0.25	0.03	0.86	0.35	0.17	1.07	0.86	0.53	0.23	0.05	0.44	0.17	1.02	0.35	0.61
F3	1.27	1.43	0.15	0.25	-	-	0.94	0.25	0.08	1.48	0.96	0.17	0.03	0.41	0.25	0.07	0.52	0.26	0.26
F4	1.86	0.73	0.17	0.03	0.08	-	0.80	0.78	0.03	0.66	1.48	0.07	0.47	0.08	0.62	0.39	0.40	0.24	0.31
FC3	1.95	0.53	0.23	0.86	0.94	0.80	-	0.80	0.87	-	0.10	0.71	0.24	1.24	1.51	0.78	0.54	1.15	0.87

FC4	0.56	2.22*	0.94	0.35	0.25	0.78	0.80	-	0.70	0.56	1.15	0.44	0.62	0.26	0.05	0.35	1.09	0.08	0.17
FCz	1.64	1.92	0.70	0.17	0.08	0.03	0.87	0.70	-	0.97	1.02	0.54	0.03	0.17	0.38	0.23	1.01	0.15	0.31
FP1	1.33	0.59	-	1.07	1.48	0.66	-	0.56	0.97	-	0.29	0.15	0.54	0.96	0.86	0.62	0.03	1.33	0.56
FP2	1.86	0.03	0.73	0.86	0.96	1.48	0.10	1.15	1.02	0.29	-	0.44	0.62	1.07	1.24	0.94	0.28	0.70	1.06
FT7	1.33	0.94	0.44	0.17	0.53	0.07	0.71	0.44	0.54	0.15	0.44	-	0.26	0.96	0.53	0.25	0.62	1.17	0.56
FT8	1.15	0.86	0.26	0.23	0.03	0.47	0.24	0.62	0.03	0.54	0.62	0.26	-	0.31	0.26	0.29	0.78	0.26	0.88
Fz	1.36	1.01	0.53	0.05	0.41	0.08	1.24	0.26	0.17	0.96	1.07	0.96	0.31	-	0.05	-	0.94	0.05	0.08
P3	1.54	1.49	0.97	0.44	0.25	0.62	1.51	0.05	0.38	0.86	1.24	0.53	0.26	0.05	-	0.88	1.37	0.26	0.35
P4	1.47	1.24	0.45	0.17	0.07	0.39	0.78	0.35	0.23	0.62	0.94	0.25	0.29	-	0.88	-	0.86	0.08	0.62
Pz	2.31*	0.86	0.53	1.02	0.52	0.40	0.54	1.09	1.01	0.03	0.28	0.62	0.78	0.94	1.37	0.86	-	1.02	0.25
TP7	1.78	1.47	0.86	0.35	0.26	0.24	1.15	0.08	0.15	1.33	0.70	1.17	0.26	0.05	0.26	0.08	1.0	-	0.44
TP8	1.33	1.25	0.53	0.61	0.26	0.31	0.87	0.17	0.31	0.56	1.06	0.56	0.88	0.08	0.35	0.25	0.62	0.44	-

* p<0.05. ** p<0.01

Latency of ELAN and P600 in DD

Table 4.11 shows the median and interquartile range values for latency measure of ELAN and P600 in DD. The analysis of results indicated that the median latency for ELAN in DD showed longer latency in fronto-parietal and temporal channels when compared to centro-parietal, parietal and frontal channels. The results also indicated that latency for P600 showed longer latency in frontal, centro-parietal, frontal, fronto-parietal compared to, parietal, temporal channels.

Table 4.11

Median and IQR for ELAN and P600 Latency in DD

Electrode sites	ELAN Latency (in ms) DD		P600 Latency (in ms) DD	
	Median	*IQR*	*Median*	*IQR*
CP3	250.00	21.50	-	836.25
CP4	264.50	43.75	846.50	567.00
CPz	249.00	72.00	803.50	938.50
Cz	248.50	102.00	732.50	890.50
F3	244.00	136.00	610.00	810.50
F4	275.00	137.00	734.50	874.75
F7	257.50	87.50	766.00	183.50
F8	249.50	48.00	1023.00	470.00
FC3	258.50	85.50	864.00	643.75
FC4	245.50	115.00	878.50	586.50
FCz	261.50	90.50	800.00	496.00
FP1	263.50	159.50	876.00	924.00
FP2	266.00	112.00	755.50	892.00
FT7	245.00	89.50	619.50	839.25
FT8	299.50	-	615.00	842.75
Fz	260.50	78.25	659.50	831.50
P3	248.00	31.50	-	836.25
P4	280.50	52.25	850.50	965.50
Pz	249.00	47.50	780.50	919.50
TP7	273.00	-	614.50	826.50
TP8	265.00	-	690.50	795.25

Wilcoxon Signed Rank test was done to compare the performance of children between channels in DD. Analysis of results revealed that for ELAN and P600 Latency in DD, there was no significant difference between channels indicted in the table 4.12 and 4.13 respectively.

Table 4.12

Results of Wilcoxon test across the channels for ELAN Latency in DD.

Wilcoxon test across the channels for ELAN Latency in DD																			
	/z/ scores																		
Electrode sites	CP3	CP4	CPz	Cz	F3	F4	FC3	FC4	FCz	FP1	FP2	FT7	FT8	Fz	P3	P4	Pz	TP7	TP8
CP3	-	0.73	0.73	1.34	1.34	1.06	-	1.34	0.44	-	1.00	0.73	1.34	1.60	1.60	1.06	1.60	1.34	1.34
CP4	0.73	-	0.42	1.09	0.53	-	0.67	1.00	1.06	1.34	0.53	0.36	1.00	0.73	0.53	0.73	1.60	1.34	1.06
CPz	0.73	0.42	-	0.55	0.36	1.21	0.31	0.44	1.09	1.60	-	-	0.44	0.40	0.44		1.46	1.60	1.06
Cz	1.34	1.09	0.55	-	0.36	1.06	0.40	1.00	1.35	0.73	1.09	0.44	-	0.40	1.00	1.00	0.44	1.34	1.34
F3	1.34	0.53	0.36	0.36	-	0.44	0.40	-	0.27	1.82	1.06	0.53	-	0.55	1.60		1.34	-	0.44
F4	1.06	-	1.21	1.06	0.44	-	0.67	-	0.73	0.73	0.40	1.60	-	0.40	1.06	1.00	1.60	-	0.44
FC3	-	0.67	0.31	0.40	0.40	0.67	-	-	0.10	0.67	1.48	0.53	1.34	-	0.36	1.60	-	-	0.53
FC4	1.34	1.00	0.44	1.00	1.34	-	0.53	-	0.44	-	0.44	-	-	0.81	0.44	-	1.00	-	1.34
FCz	0.44	1.06	1.09	1.35	0.27	0.73	0.10	0.44	-	0.94	1.09	0.44	-	0.73	0.27	-	0.44	-	0.44
FP1	-	1.34	1.60	0.73	1.82	0.73	0.67	-	0.94	-	0.36	1.34	-	1.09	1.34	-	-	-	-
FP2	1.00	0.53	-	1.09	1.06	0.40	1.48	0.44	1.09	0.36	-	1.60	-	1.48		0.44	1.34	-	0.44
FT7	0.73	0.36	-	0.44	0.44	0.36	0.53	-	0.44	1.34	1.60	-	-	1.06	0.53	1.60	1.60	-	0.44
FT8	1.34	1.00	0.44	-	-	-	1.34	-	-	-	-	1.34	1.34	-	-	0.44	-	-	-
Fz	1.60	0.73	0.40	0.40	0.40	0.40	-	0.81	0.73	1.09	1.48	1.06	-	-	1.46	1.34	1.34	-	-
P3	1.60	0.53	0.44	1.00	1.34	1.06	0.36	1.34	0.27	1.34	-	0.53	-	1.46	-	-	1.06	1.34	1.34
P4	1.06	0.73	-	1.00	-	1.00	1.60	1.00	-	-	0.44	1.60	0.44	1.34	1.00	1.00	1.06	-	-
Pz	1.60	1.60	1.46	0.44	0.27	1.60	-	1.00	0.44	-	1.34	1.60	-	1.34	1.06	1.06	1.60	-	1.34
TP7	1.34	1.34	1.60	1.34	0.44	-	-	-	-	-	-	-	-	-	1.34	-	1.34	1.60	-
TP8	1.34	1.06	1.06	1.34	-	0.44	0.53	1.34	0.44	-	0.44	0.44	-	-	1.34	-	-	-	-

* p<0.05. ** p<0.01

Table 4.13

Results of Wilcoxon test across the channels for P600 Latency in DD.

Wilcoxon test across the channels for P600 Latency in DD

/z/ scores

Channels	CP3	CP4	CPz	Cz	F3	F4	FC3	FC4	FCz	FP1	FP2	FT7	FT8	Fz	P3	P4	Pz	TP7	TP8
CP3	-	2.04*	1.78	0.86	0.66	1.06	2.17*	0.05	1.80	0.26	0.62	0.53	0.35	1.12	0.28	0.56	1.37	0.86	0.08
CP4	2.04*	-	0.90	1.25	1.53	1.09	0.15	2.49*	0.78	1.18	0.87	0.94	1.49	1.36	1.95	1.37	1.20	1.16	1.80
CPz	1.78	0.90	-	1.06	1.15	0.87	0.39	1.80	0.15	1.06	0.73	1.06	1.42	1.42	1.95	0.71	0.84	0.94	1.77
Cz	0.86	1.25	1.06	-	0.66	0.39	1.09	1.47	0.17	0.25	0.23	0.26	0.47	0.56	0.80	0.08	0.47	0.71	0.96
F3	0.66	1.53	1.15	0.66	-	0.71	1.56	0.45	1.06	0.29	0.66	0.44	0.03	0.21	0.66	0.15	0.80	0.08	0.35
F4	1.06	1.09	0.87	0.39	0.71	-	1.49	1.17	0.31	0.15	0.53	0.54	0.62	0.44	1.06	0.47	0.72	0.59	0.94
FC3	2.17*	0.15	0.39	1.09	1.56	1.49	-	1.81	0.52	1.41	1.36	1.53	1.41	1.78	2.13*	0.86	1.02	1.51	1.43
FC4	0.05	2.49*	1.80	1.47	0.45	1.17	1.81	-	1.86	0.56	1.24	0.62	0.71	0.56	0.05	0.97	1.49	0.53	0.29
FCz	1.80	0.78	0.15	1.17	1.06	0.31	0.52	1.86	-	0.88	0.54	1.42	1.29	0.93	1.36	1.09	0.31	1.33	1.72
FP1	0.26	1.18	1.06	0.25	0.29	0.15	1.41	0.56	0.88	-	0.28	0.10	0.07	0.25	0.56	0.17	1.01	0.08	0.76
FP2	0.62	0.87	0.73	0.23	0.66	0.53	1.36	1.24	0.54	0.28	-	0.35	0.78	0.35	0.80	0.31	0.59	0.43	1.15
FT7	0.53	0.94	1.06	0.26	0.44	0.54	1.53	0.62	1.42	0.10	0.35	-	0.25	0.51	1.00	0.05	0.80	0.15	0.25
FT8	0.35	1.49	1.42	0.47	0.03	0.62	1.41	0.71	1.29	0.07	0.78	0.25	-	0.23	0.35	0.05	1.02	0.08	0.29
Fz	1.12	1.36	1.42	0.56	0.21	0.44	1.78	0.56	0.93	0.25	0.35	0.51	0.23	-	0.76	0.00	0.94	0.25	0.44
P3	0.28	1.95	1.95	0.80	0.66	0.06	2.13*	0.05	1.36	0.56	0.80	1.00	0.35	0.76	-	0.88	1.68	0.62	0.05
P4	0.56	1.37	0.71	0.08	0.15	0.47	0.86	0.97	1.09	0.17	0.31	0.05	0.05	-	0.88	-	0-.65	-	0.45
Pz	1.37	1.20	0.84	0.47	0.80	0.72	1.02	1.49	0.31	1.01	0.59	0.80	1.02	0.94	1.68	0.65	-	0.70	1.33
TP7	0.86	1.16	0.94	0.71	0.08	0.59	1.51	0.53	1.33	0.08	0.43	0.15	0.08	0.25	0.62	-	0.70	-	0.35
TP8	0.08	1.80	1.77	0.96	0.35	0.94	1.43	0.29	1.72	0.76	1.15	0.25	0.29	0.44	0.05	0.45	1.33	0.35	-

* p<0.05. ** p<0.01

4.2.2 Comparison of performance of typically developing children (TDC) and children with dyslexia on ERP (ELAN & P600) measures

Amplitude measure

Table 4.14 shows the median and interquartile range values for amplitude of ELAN component in TDC and DD. The comparison of amplitude between TDC and DD indicated greater amplitude in children with DD when compared to TDC. The amplitude was found to be the greatest for the centro-parietal region (CP4) and the least for the fronto-temporal and temporo-parietal regions. Results on Mann-Whitney U test revealed that there was a significant difference between TDC and DD in terms of amplitude measure of ELAN for CP3 ($|z|$=2.40, p<0.05) and CP4 ($|z|$=2.26, p<0.05) channels.

The median values for amplitude indicated greater amplitude observed in DD when compared to TDC (see table 4.4). There was no significant difference between TDC and DD across all other channels CPz (|z|=1.67, p>0.05),Cz(|z|=1.55, p>0.05),F3 (|z|=0.97, p>0.05),F4(|z|=0.29, p>0.05),F7 (|z|=1.10, p>0.05), FC3(|z|=1.87, p>0.05), FC4(|z|=0.74, p>0.05), FCz(|z|=1.40, p>0.05),FP1 (|z|=0.16, p>0.05),FP2 (|z|=0.37, p>0.05),FT7 (|z|=0.82, p>0.05), FT8(|z|=1.04, p>0.05), Fz(|z|=1.79, p>0.05), P3(|z|=1.86, p>0.05), P4 (|z|=1.48, p>0.05), Pz (|z|=1.78, p>0.05), TP7 (|z|=0.46, p>0.05), TP8(|z|=0.67, p>0.05) except CP3 and CP4.

Table 4.14

Median and IQR for ELAN amplitude (in μV) and P600 amplitude (in μV) in TDC and DD

Electrode sites	ELAN Amplitude (in μV)				P600 Amplitude (in μV)			
	TDC		DD		TDC		DD	
	Median	*IQR*	*Median*	*IQR*	*Median*	*IQR*	*Median*	*IQR*
CP3	-3.08	4.88	-9.68	2.96	1.85	3.86	-	0.63
CP4	-3.90	5.75	-9.39	4.62	3.75	3.99	4.63	7.31
CPz	-3.86	3.42	-7.58	8.11	2.08	3.81	1.99	5.55
Cz	-5.53	6.54	-9.94	10.44	7.28	8.74	2.49	5.77
F3	-4.90	7.84	-9.55	19.90	6.69	15.36	2.12	7.02
F4	-5.80	4.63	-7.39	11.79	5.88	10.96	2.24	6.58
F7	-5.07	8.66	-12.81	26.06	10.35	25.76	5.36	11.57
F8	-4.42	19.63	-5.71	18.63	9.63	6.11	7.77	7.04
FC3	-4.86	5.72	-12.38	12.13	6.07	9.72	3.89	8.10
FC4	-3.29	8.73	-13.24	12.26	4.65	9.09	0	4.23
FCz	-6.30	6.64	-8.80	10.28	9.32	9.33	3.06	9.33
FP1	-8.56	10.37	-9.33	19.78	11.01	11.62	1.67	9.39
FP2	-9.58	9.58	-15.17	12.61	14.65	16.24	2.36	9.57
FT7	-5.07	11.52	-11.48	11.05	6.60	10.80	0.91	7.42
FT8	-8.02	5.66	-4.21	-	1.55	7.22	1.26	7.88
Fz	-3.19	10.32	-11.29	8.44	8.81	12.81	0.96	5.0
Oz	-2.01	4.57	-	-	-	1.83	-	-
P3	-3.55	4.22	-7.89	4.00	0.75	3.06	0	7.20
P4	-3.28	8.35	-5.08	23.74	3.39	5.67	0.30	6.83
Pz	-2.84	1.40	-7.58	15.46	2.98	5.31	3.93	6.93
TP7	-2.67	8.00	-3.82	-	2.57	5.81	1.81	3.61
TP8	-3.78	2.19	-2.62	-	-	1.45	0.57	6.89

Further, the analysis of Mann-Whitney U test results for amplitude of P600component indicated that there was a significant difference between TDC and DD for the channels CP3 (|z|=2.75, p<0.01), Cz (|z|=2.21, p<0.05), F3 (|z|=2.07, p<0.05), F7 (|z|=2.92, p<0.01), F8 (|z|=2.47, p<0.05), FCz (|z|=2.75, p<0.01), FP1 (|z|=2.68, p<0.01), FP2 (|z|=2.67, p<0.01), and Fz (|z|=3.16, p<0.01). The median amplitude of P600 across channels indicated that greater amplitude was observed in TDC than LD across these significant channels i.e., Cz, F3, F7, F8, FCz, FP1, FP2 and Fz and CP3 and other channel there was no significant difference observed CP4(|z|=0.54, p>0.05),CPz (|z|=0.65, p>0.05), F4(|z|=1.74, p>0.05), FC3(|z|=1.35, p>0.05), FC4(|z|=1.93, p>0.05), FT7 (|z|=1.33, p>0.05), FT8(|z|=0.13, p>0.05), P3(|z|=0.65, p>0.05), P4 (|z|=0.91, p>0.05), Pz (|z|=0.26, p>0.05), TP7 (|z|=0.45, p>0.05), and TP8(|z|=1.62, p>0.05).

Latency measure

Table 4.15 shows the median and interquartile range values for latency measure of ELAN component in TDC and DD. The comparison of median peak latency measure for ELAN component between TDC and DD indicated a longer latency in children with DD when compared to TDC. The latency was found to be longer across all the channels. Results on Mann-Whitney U test revealed that there was a significant difference between TDC and DD for latency measure for channels, CP3 (|z|=3.27, p=0.001), CP4 (|z|=3.11, p<0.01), CPz (|z|=2.90, p<0.01), Cz (|z|=2.34, p<0.05), F4 (|z|=2.01, p<0.05), F7 (|z|=2.3, p<0.05), F8 (|z|=2.79, p<0.01), FC3 (|z|=2.58, p=0.01), FCz (|z|=2.49, p<0.05), FP1 (|z|=2.31, p<0.01), FP2 (|z|=3.15, p<0.05), FT7 (|z|=2.40, p<0.05), FT8 (|z|=2.24, p<0.05), Fz (|z|=2.65, p<0.01), P3 (|z|=3.12, p<0.01), P4 (|z|=2.61, p<0.01), Pz (|z|=2.45, p<0.05), TP7 (|z|=2.49, p<0.05), TP8 (|z|=2.54, p<0.05). There was no significant difference observed across F3 (|z|=1.17, p> 0.05), FC4 (|z|=1.48, p> 0.05) channels.

Table 4.15

Median and IQR for ELAN latency (in ms) and P600 latency (in ms) in TDC and DD

Electrode sites	ELAN Latency (in ms)				P600 Latency (in ms)			
	TDC		DD		TDC		DD	
	Median	*IQR*	*Median*	*IQR*	*Median*	*IQR*	*Median*	*IQR*
CP3	172.00	52.00	250.00	21.50	653.00	176.25	0	836.25
CP4	166.00	39.00	264.50	43.75	694.00	124.00	846.50	567.00
CPz	181.00	62.00	249.00	72.00	675.00	222.50	803.50	938.50
Cz	178.00	49.00	248.50	102.00	704.00	212.75	732.50	890.50
F3	192.00	46.50	244.00	136.00	639.00	180.50	610.00	810.50
F4	176.50	32.00	275.00	137.00	741.00	345.50	734.50	874.75
F7	180.00	37.00	257.50	87.50	699.00	130.00	766.00	183.50
F8	172.50	56.00	249.50	48.00	694.50	187.50	1023.00	470.00
FC3	186.00	54.00	258.50	85.50	698.00	192.75	864.00	643.75
FC4	169.50	38.50	245.50	115.00	698.00	351.25	268.50	810.50
FCz	196.00	62.50	261.50	90.50	636.00	134.00	800.00	496.00
FP1	201.00	60.00	263.50	159.50	704.00	249.75	354.00	877.00
FP2	198.00	61.00	266.00	112.00	698.00	177.00	755.50	892.00
FT7	169.00	47.00	245.00	89.50	630.00	104.75	619.50	839.25
FT8	165.00	17.00	299.50	-	584.00	638.00	615.00	842.75
Fz	168.00	30.00	260.50	78.25	739.00	137.25	659.50	831.50
Oz	197.00	46.00	-	-	-	678.75	-	-
P3	176.00	40.00	248.00	31.50	624.00	660.50	-	836.25
P4	172.00	22.25	280.50	52.25	655.00	295.00	280.50	965.50
Pz	177.00	30.00	249.00	47.50	662.00	237.25	780.50	919.50
TP7	161.00	14.50	273.00	-	546.00	694.00	614.50	826.50
TP8	159.00	48.75	265.00	-	-	629.50	304.50	795.25

The analysis of results for latency measure in P600 component between TDC and DD revealed that the latency was found to be longer across most of the channels (see table 4.15). The longest latency within DD was found to be across parietal channels, followed by temporo-parietal, frontal channels, fronto-parietal, fronto-temporal, fronto-central and central channels. Results on Mann-Whitney U test revealed that there was a significant difference between TDC and DD in terms of latency measure of P600 for channels, F7 ($|z|=1.96$, $p<0.01$) and TP8 ($|z|=1.73$, $p<0.05$). There was no significant difference across all the other channels CP3($|z|=1.19$, $p>0.05$),CP4($|z|=1.28$, $p>0.05$), CPz ($|z|=1.00$, $p>0.05$), Cz($|z|=0.11$, $p>0.05$) F3 ($|z|=0.76$, $p>0.05$), F4($|z|=0.22$, $p>0.05$), F8($|z|=1.33$, $p>0.05$),FC3($|z|=0.61$, $p>0.05$), FC4($|z|=1.18$, $p>0.05$), FCz ($|z|=1.44$, $p>0.05$), FP1 ($|z|=0.96$, $p>0.05$), FP2 ($|z|=0.13$, $p>0.05$), FT7 ($|z|=0.17$, $p>0.05$), FT8($|z|=0.67$, $p>0.05$), Fz($|z|=0.57$, $p>0.05$) P3($|z|=0.47$, $p>0.05$), P4

(|z|=0.46, p>0.05), Pz (|z|=0.61, p>0.05), and TP7 (|z|=0.67, p>0.05) studied in the present study.

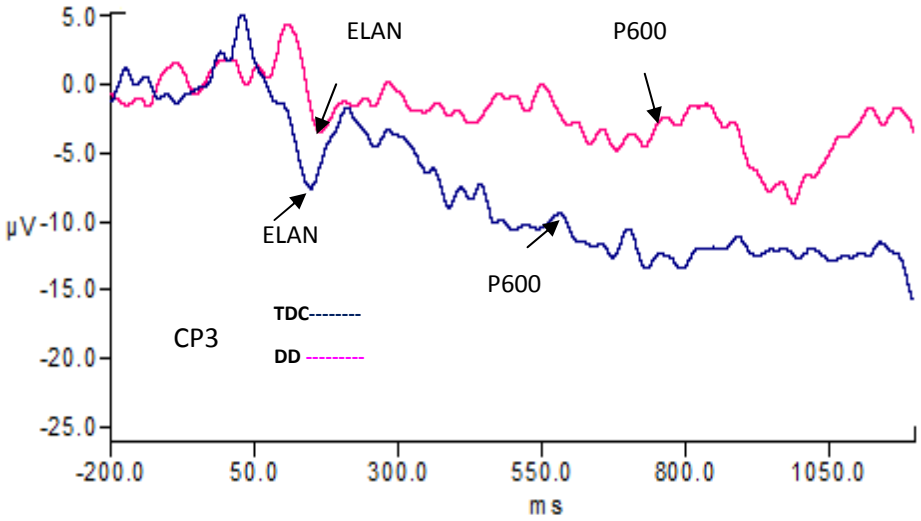

Figure 4.3 Grand average waveform at CP3 channel for children with TDC and DD for violated sentences

In summary, it can be seen from Figure 4.5 that for the syntactic violation condition, control children showed a significant ELAN effect in TDC and a significant P600 effect in centro-parietal channels in TDC. In contrast, the children with dyslexia demonstrated a rather different pattern by showing an ELAN effect only and lack of P600 effect in DD. Late positivity was observed in children with TDC when compared to TDC.

The topographical scalp distribution of activity from 0 ms to 1300 ms in 100 ms interval is shown in Figure 4.4 and Figure 4.5 for children with TDC and DD respectively. From the Figure 4.4, the activity was seen more in the anterior regions, spreading towards spreading towards the coronal areas and parietal areas towards the left regions for violated sentences in TDC and the activation is spread out in these regions. On the other hand, in children with DD, the activation is occurring much later as indicated in Figure 4.5 and the activation regions seem to be more restricted when compared to TDC. Also the activation seems to spread out to the right hemisphere. In the

present ERP data of this study, it indicates that the P600 tends is delayed in the individuals with dyslexia.

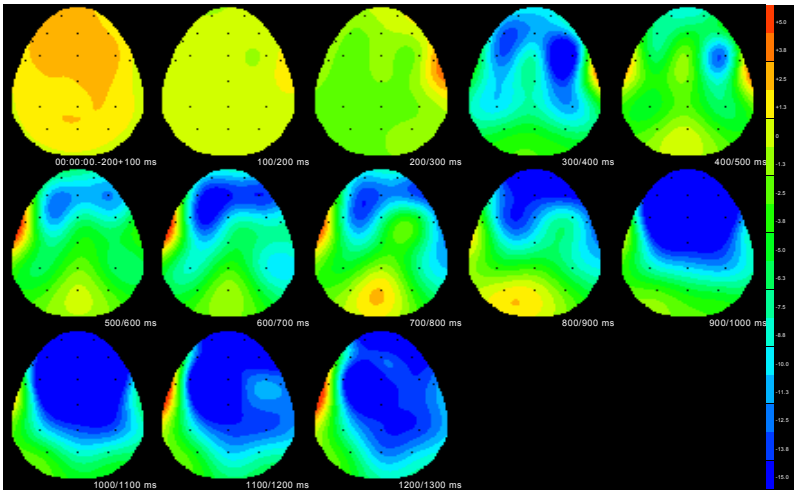

Figure 4.4 Scalp distribution for positivity and negativity for syntactically violated sentences in TDC

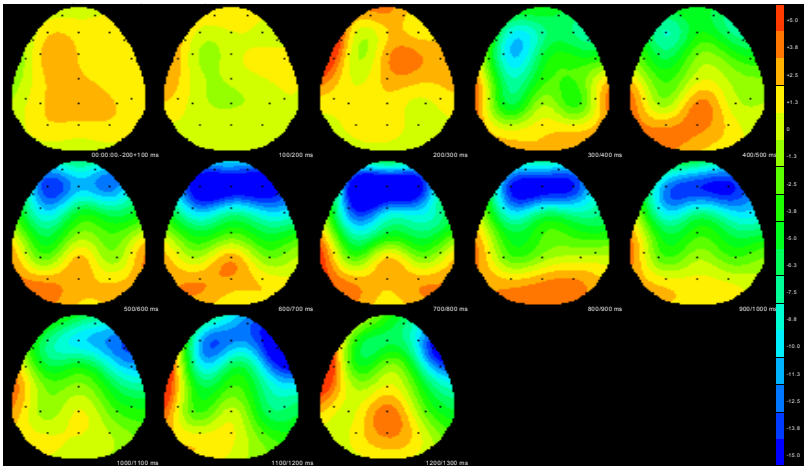

Figure 4.5 Scalp distribution for positivity and negativity for syntactically violated sentences in children with DD

4.3 Correlation of behavioural measures and ERP (ELAN and P600) measures in typically developing children (TDC) and children with dyslexia.

Spearman correlation was done to study the relation between behavioral measures (accuracy for word and non-word; reaction time for word and non-word) and ELAN

(latency & amplitude) and P600 measures (latency & amplitude) across 24 channels in typically developing children and children with developmental dyslexia (DD).

4.3.1 Relation between behavioral and ELAN and P600 in TDC.

The results indicated that in TDC, for ELAN latency there was a significant negative correlation found for reaction time of correct word and latency at CP3 channel (r=-0.611, at p=0.015), whereas for remaining channels there was no correlation found for reaction time of correct word. Also, there was no correlation found for ELAN latency across 21 channels and reaction time of wrong word and reaction time of correct and wrong word.

Similarly in TDC, for ELAN amplitude there was a significant positive correlation of reaction time of wrong word and amplitude at CP3 channel (r=0.564, at p=0.028); significant positive correlation of reaction time of wrong word and amplitude at FC3 channel (r=0.611, at 0.016); significant negative correlation of reaction time of correct word and amplitude at FP1 channel (r=-0.560, at 0.037); significant negative correlation of correct word and amplitude at Oz (r=-0.857, at 0.014). For other remaining channels there was no correlation found for reaction time of correct word and wrong word. Also, there was no correlation found for ELAN amplitude across 21 channels and accuracy of correct and wrong word.

With regard to P600 latency, in TDC, there was a significant negative correlation of reaction time of wrong word and latency at channel FP2 (r=-0.561, at 0.026); reaction time of wrong word and latency at channel FT7 (r=-0.645, at 0.032) and accuracy of correct word and latency at channel TP7 (r=-0.807, at 0.009). For other remaining channels there was no correlation found for accuracy and reaction time of both correct and wrong word.

When P600 amplitude in TDC was considered, there was a significant negative correlation of reaction time of wrong word and amplitude at channels such as Cz (r=-0.618, at 0.019); F4 (r=-0.650, at 0.022); FC3 (r=-0.682, at 0.005); FC4 (r=-0.727, at 0.007); FCz (r=-0.621, at 0.013); FP1 (r=-0.632, at 0.011); FP2 (r=-0.768, at 0.001); Fz (r=-0.682, at 0.005). Also, there was significant negative correlation of reaction time of correct word and amplitude at TP7 (r=-0.750, at 0.020) and significant negative correlation of accuracy of correct word and amplitude at Oz (r=-0.971, at 0.001). For remaining channels and accuracy and reaction time of correct and wrong words there was no relation was found.

4.3.2 Relation between behavioral and ELAN and P600 in children with DD

The results indicated that in children with DD, for ELAN latency there was a significant positive correlation of accuracy of wrong word and latency at channel Cz (r=0.986, at p=0.000), whereas for other remaining channels there was no correlation found for ELAN latency and accuracy and reaction time of correct and wrong word.

Similarly in children with DD, for ELAN amplitude there was a significant negative correlation of accuracy of wrong word and amplitude at F3 channel (r=-0.900, at p=0.037); significant negative correlation of reaction time of wrong word and amplitude at P3 channel (r=-0.900, at 0.037); For other remaining channels there was no correlation found for accuracy and reaction time of correct word and wrong word.

With respect to P600 latency, in children with DD, there was a significant positive correlation of accuracy of correct word and latency at channel TP8 (r=0.793, at 0.033); reaction time of correct word and latency at channel FT7 (r=-0.645, at 0.032); significant negative correlation of accuracy of correct word and latency at channel FT8 (r=-0.810, at 0.015) and significant positive correlation of accuracy of correct word and latency at

channel Cz (r=0.740, at 0.023). For other remaining channels there was no correlation found for accuracy and reaction time of both correct and wrong word.

When P600 amplitude in children with DD was considered, there was a significant positive correlation of accuracy of correct word and amplitude at channel TP8 (r=0.829, at 0.021); significant negative correlation of reaction time of wrong word and amplitude at channel FC4 (r=-0.929, at 0.003). For remaining channels and accuracy and reaction time of correct and wrong words there was no relation was found.

DISCUSSION

5.1. Comparison of performance ofTDC and DD on behavioral measures

Results of behavioral task also showed that the children with DD required more time than the age matched TDC to carry out grammaticality judgment task. Often children with DD are found to have problems in phonological awareness and decoding which may lead to longer time in the lexical processing which could be attributed to the poor performance of children with dyslexia in the present study. According to phonological deficit theory (Liberman, 1973; Snowling, 2000), the ability to segregate and manipulate the speech sounds is affected in children with dyslexia which will also result in slower lexical access speed as evidenced in the present study.

Another possible explanation for the observed deficit in children with dyslexia is poor verbal short term memory. According to (Snowling, 2001) poor verbal short term memory is another manifestation of children with dyslexia. The children are required to keep the heard utterance in their short term memory while it is being processed; when incapable to do so will may lead to longer processing time on grammaticality judgment task. Thus the poor performance of children with dyslexia in the present study could likely be the result of poor verbal short term memory. A general deficit in children with dyslexia such as poor/ reduced attention could also affect the outcome of the performance. Nicolson and Fawcett (1994) stated that a general (non phonological) deficit which reflect in slower stimulus classification speed and a linguistic (phonological) deficit which reflect in slower lexical access speed may be contributing to the slowness of children with dyslexia. This reason holds good here also.

The observed deficits in children with dyslexia could also be explained through auditory temporal processing deficit theory. According to auditory temporal processing

deficit theory (Tallal, 1980; Tallal, Miller, & Fitch, 1993), children with dyslexia have a deficit in rapid auditory processing. That means these children may have difficulties in developing sufficiently rapid processing rates for word recognition. This could be a reason for the longer reaction times for children with dyslexia in the present study. Children with dyslexia were also reported to have speech perception difficulties (Godfrey et al, 1981). Such speech perception deficits may lead to deficits in the ability to manipulate and process speech sounds and may also result in longer duration for lexical processing as seen in the performance of children with dyslexia in the present study.

Children with dyslexia were also reported to have poor performance on auditory tasks such as frequency discrimination (McAnally & Stein, 1996; Ahissar et al, 2000), temporal order judgement (Nagarajan et al, 1999; Tallal, 1980) and also poor categorical perception of certain sound contrasts (Adlard & Hazan, 1998; Mody et al., 1997; Serniclaes et al, 2001). All these auditory processing deficits will interfere with the identification of phonological cues that are typical for spoken word recognition and hence the children with dyslexia may perform poorer and slower compared to normal participants. Poorer performance on temporal related tasks in dyslexia could be related to temporal processing deficits explained in lieu of the temporal processing deficit theory (Tallal, 1980). Sub groups of children with dyslexia are found to exhibit difficulties in auditory related tasks such as spectral parameters of frequency. Hence, a deficit in spectral related characteristics such as frequency discrimination could lead to poorer perception of speech sounds which require fine-grained auditory discrimination such as minimal pairs of words. Literature has suggested that phonological problems in dyslexia are often due to a more fundamental deficit in auditory temporal processing mechanisms (Tallal, 1980). It was also reported that children with dyslexia showed impaired discrimination and sequencing brief and rapid acoustic stimuli when compared normal

peers (Tallal, 1980; Tallal,Miller& Fitch, 1993). Further basic perceptual deficits could result in a host of deficits which include disruption in terms of development of the phonological system. This disruption could lead to problems in reading and spelling (Nagarajan et al., 1999; Wright et al., 1997).

The poor performance of the children with dyslexia in the present study could also be described using cerebellar theory (Nicolson & Fawcett, 1990; Nicolson, Fawcett &Dean, 2001), which says that cerebellar abnormality is the cause for the difficulties suffered by the children with dyslexia. The cerebellum is involved in speech perception (Mathiak, Hetrich, Grodd & Ackermann, 2002) and the automatization of any skill, whether motor or cognitive. So in children with dyslexia, abnormality in the cerebellum may lead to deficit in speech perception which will consequently result in poor performance on lexical processing. A weak capacity to automatize would also have an impact on the lexical processing and can result in longer reaction time as evidenced in the present study.

The present study shows that differences concern reaction times rather than accuracy. The children with dyslexia were as accurate as TDC of the same age in grammaticality judgement task. Despite the fact that they were not less accurate, they were slower than age-matched peers in judging the violated and canonical sentences. Thus, children with dyslexia seem to be delayed and not deviant from the syntactic development point of view.

5.2. Performance of TDC and children with DD on ERP (ELAN & P600) measures

ERP studies on the development of language comprehension using similar syntactic constructions showed that early syntactic processes, in particular, as reflected by

the early anterior negativity, develop gradually (Hahne et al., 2004), such that an adultlike ELAN emerges only in 13-year-olds, whereas 10-year-olds show a left anterior negativity effect. Similar results were found in the present study with significant left anterior negativity effect between children 8 to 10 year old.

For the syntactic violation, a combined pattern, comprising a bilaterally distributed early anterior negativity and a P600, was observed only for the control children. The children with DD showed a different pattern containing a delayed anterior negativity restricted to the left hemisphere and a P600 component. In line with from Hahne et al. (2004), a bilaterally distributed early anterior negativity (100–300 msec) was observed for the control children. In contrast, for children with DD, no early anterior negativity was observed. The ELAN is assumed to reflect early processes of phrase structure building based on word category information, and Hahne and Friederici (1999) showed that these processes are highly automatic. This suggests that in TDC, a neural processing system, as observed in adults, has been established for the processing of default structures in sentences but may continue to develop to be adult-like for complex structures (Hahne et al., 2004). Longer latency in ELAN in children with dyslexia indicate that probably the process required process syntactic components is still not automatic and phrase structure building for word category is still building in children with dyslexia. The results probably reflect that reflect syntactic phrase structure building processes that are not developed to the same degree as generally expected for their age (Sabisch, Hahne, Glass, Suchodoletz, & Friederici, 2006). The results showed that many children with DD were able to perform the syntactic violation task with poorer accuracy which means that they process phrase structure information, but are unable to do it automatically and as early as the TDC of the same age.

Children with DD are often found to show lack of sensitivity to subject-verb agreement morphology (Jiménez et al., 2004; Rispens, Roeleven, & Koster, 2004), impaired inflectional morphology (Altmann, Lombardino, & Puranik, 2008; Joanisse, Manis, Keating, & Seidenberg, 2000) as well as weakness in morphological awareness tasks (Leikin & Hagit, 2006). With respect to the phrase structure violation condition as in the present study, the P600 component generally reflects processes of syntactic reanalysis and repair that emerge during a late processing phase after the parser has detected a syntactic violation (Friederici, 2002). These processes are assumed to be highly controlled (Hahne & Friederici, 1999). The presence of the P600 thus indicates that both children with DD and control children are sensitive to the syntactic violation in phrase structure. This could be also be explained using the *Processing limitation hypothesis.* It is possible that children with dyslexia could have deficits in the autonomous subcomponents such as the phonology, lexicon and problems at these levels could be leading to problems in processing syntax. Each component has its own parser which is responsible for rule access and resolution of ambiguity. This system works in a bottom-up way: the analysis of linguistic input must take place rapidly at the lower levels (phonology) in order to transfer the information to higher levels (semantics and syntax) (Bar-Shalom et al., 1993). This means that if the analysis in lower level components is disturbed in some way, the transfer to higher level components will also be disrupted. Hence, even though for instance the syntax module itself is intact, a person can still have trouble using syntax to understand a sentence because of a phonological problem. It could be possible that in the present study there could be some children with DD who show phonological problems or lexicon, thus leading to problems in syntactic comprehension.

Further this could also be explained for reading problems in DD due to a failure to automatize word recognition (Yap, 1993). In order to read aloud quickly one needs to be

able to resort to an automatic process, which is found to be difficult in children with DD (Yap, 1993; Nicolson &Fawcett, 1999; Fawcett, Nicolson & Dean, 1996). Further, the morpho-syntactic difficulties can be explained using the *Surface hypothesis* (Leonard, 1998) which is often explained in SLI. According to this hypothesis children with DD may be having difficulties in processing morpho-syntax due to poor auditory perception often reported as difficulties in DD. Due to this the children are unable to build up stable phonological representations which are necessary for decoding letters into sounds. According to Leonard (1998), grammatical elements have weak phonetic features (they often are unstressed syllables characterised by short duration, low fundamental frequency and amplitude) making them vulnerable to distorted perception in children who have perceptual problems such as children with DD.

According to recent neurocognitive models (Friederici, 2002), a biphasic electrophysiological pattern (LAN/P600) is normally expected in response to morphosyntactic violations, while an N400 component is expected in response to semantic violations. Differences in these electrophysiological components have been sporadically reported in participants with dyslexia (Leikin, 2002; Rispens, Been, & Zwarts, 2006, Sabisch, Hahne, Glass, von Suchodoletz, & Friederici, 2006; Russeler, Becker, Johannes, & Münte, 2007), even if linguistic difficulties did not emerge from standardized tests of language comprehension. Rispens et al. (2006) have investigated the presence and latency of the P600 component in response to subject-verb agreement violations in Dutch-speaking adults with DD. Despite the absence of differences between participants with dyslexia and control participants in judging the grammaticality of the sentences, ERP data revealed subtle differences between groups, particularly related to latency (the P600 tended to peak later in the dyslexic group compared to the control group). Sabisch et al. (2006) compared the electrophysiological responses to syntactic

violations (phrase structure) in typically developing German-speaking children (aged 10 to 12) with those of children with DD and children with SLI. No behavioral difference was found in the sentence correctness judgment task between control and children with dyslexia, while children with SLI generally performed worse. Concerning ERPs, a similar P600 was found in all groups. However, instead of the early starting LAN shown by control children in response to syntactic violations, children with dyslexia presented a delayed LAN (300-600 ms) that was even more delayed in the SLI group (700-1000 ms). These results are discussed in terms of the delay, in children with dyslexia and SLI, of the early and presumably highly automatic processes of phrase structure building.

Similar to the findings of Rispens et al. (2006) who investigated the presence and latency of the P600 component in response to subject-verb agreement violations in Dutch-speaking adults with DD, in the present study it was found that despite the absence of differences between participants with dyslexia and control participants in judging the grammaticality of the sentences, ERP data revealed subtle differences between groups, particularly related to latency (the P600 tended to peak later in the dyslexic group compared to the control group). The findings of the present study are also in line with Sabisch et al. (2006) who found no behavioral difference in the sentence correctness judgment task between control and children with dyslexia. However, in ERP measure instead of the early starting LAN shown by control children in response to syntactic violations, children with dyslexia presented a delayed LAN (300-600 ms). These results are indicative of early and highly automatic processes of phrase structure building in the TDC when compared to DD.

In a recent developmental language event-related potential (ERP) study, Hahne and colleagues [2004] showed that children between 6 and 13 years of age listening to syntactically incorrect passive sentences displayed a late positivity similar to the P600

recorded in adult ERP studies. The effect was smaller than those in adults, with latency decreasing with age. Six-year-olds, in contrast to the older age groups, did not show an early left anterior negativity (ELAN) effect. According to Hahne and colleagues (2004), ELAN seems to reflect highly automatic structure-building processes. These processes appear not to be established at age 7, but gradually develop toward adult like processing during late childhood (Friederici, 1983).

Studies also show that preschool children (36 and 48- month-olds) also display a late positive wave in response to morphosyntactic violations in sentences, with largest amplitudes over anterior regions of the scalp for 48-month-olds between 500 and 1500 ms (Silva-PereyraJ, Rivera-Gaxiola & Kuhl, in press). Visual inspection of the waveforms indicated that greater positivity was observed for the TDC when compared to DD children for the subject-verb agreement violations. The positivity was greater for violated sentences than the grammatical canonical sentences. The positivity was however greater for the anterior regions than the posterior or central regions. Noteworthy was that the positivity was greater for left channels than the right channels of the anterior regions in TDC when compared to DD. This supposedly indicates that the processing of information in TDC for syntactic processing is controlled by language areas in both the hemispheres, which is more likely than what is observed in children with DD. Longer latencies in children with DD are indicative that processing grammatical information of words in sentences is slower in dyslexia (Leikin, 2002). The results of the study showed that ELAN was found in both TDC and DD, greater in the left anterior region. For P600, grammaticality effect was observed in the left and right parietal region in TDC. Both the TDC and DD showed significant effects of grammaticality in right posterior region and in the midline. There was a significant difference in the left posterior region for TDC. However, there was no significant grammaticality effect observed in DD.

The ELAN has been assumed to reflect a highly automatic processing phase of syntactic processing (Hahne &Friederici,, 1999). The results of the present study indicate that the children with dyslexia also make use of this early mechanism and they are able to detect the violations like the TDC.

Overall, the distribution pattern of P600 in response to sentence violations in DD was relatively similar to the TDC. The regions in the TDC in which a robust P600 was seen was also observed in children with DD as well. However there were differences found in certain regions as well. It was observed that in the grand average ERP that ungrammatical violated sentence elicited increased positivity than the grammatical canonical sentences. The findings also suggested that greater positivity in left posterior regions in TDC and right posterior in DD. In a study conducted by Pugh et al. (2000) it was reported that left temporo-parietal brain circuit was found to be disrupted in dyslexia. A functional problem in the left posterior region could be explained for weaker activation in left region and relative greater activation in right posterior region in dyslexia. Delayed linguistic processing as indicated by various studies (Brandeis et al., 1994; Helenius et al., 1999, and Leikin, 2002) could have resulted in longer latency responses in children with DD. Similar results are suggested from the behavioral data indicating slower processing time in children with DD. Greater restricted distribution of P600 further indicates less synchronous active neurons functioning (Rispens, 2004).

SUMMARY AND CONCLUSION

Very few studies have been conducted in the Indian context with respect to ERPs at the level of sentence processing in children with dyslexia, though there is ample behavioral research in the area of language comprehension in children with dyslexia with a subgroup presenting with significant language deficits.The aim of the present study was to compare the behavioral correlates with event related potential (ERP) correlates of implicit morpho-syntactic processing in children with DD in comparison to typically developing children (TDC). The present study intended to explore responses to subject-verb agreement in DD, measuring performance on behavioral tasks and measuring ERP responses.

The participants included two groups – group 1 including with 30 typically developing children in the age range of 8-10 years old and group 2 including 15 children with DD in the age range of 8-10 years. Both behavioral and ERP data were analyzed. The behavioral data included reaction time (RT) in milliseconds and accuracy for canonical and violated sentences. The ERP data included latency (in milliseconds) and amplitude (in micro volts) of the ELAN at 22 different channels.

On behavioral tasks the findings of the study suggested that children with DD took longer time to respond to grammaticality judgment when compared to TDC as indicated by the results of reaction time measure. However, the children with DD were able to accurately respond to grammaticality judgment similar to TDC. This could be explained with phonological deficit theory (Liberman, 1973; Snowling, 2000), as children with dyslexia have poorer ability to segregate and manipulate speech sounds, which could result in slower lexical access speed in turn affecting the processing of sentences.

Often children with DD are found to have problems in phonological awareness and decoding which may lead to longer time in the lexical processing, which could be attributed to the poor performance of children with dyslexia in the present study. According to phonological deficit theory (Liberman, 1973; Snowling, 2000), the ability to segregate and manipulate the speech sounds is affected in children with dyslexia which will also result in slower lexical access speed as evidenced in the present study. Slower sentence processing speed could also be attributed to poor verbal short-term memory (Snowling, 2001) wherein children with dyslexia find it hard to process the heard utterance in their short-term memory, while it is been processed, leading to longer processing time on grammaticality judgment task.

According to (Snowling, 2001) poor verbal short-term memory is another manifestation of children with dyslexia. The children are required to keep the heard utterance in their short term memory while it is being processed; when incapable to do so will may lead to longer processing time on grammaticality judgement task.

The present study shows that differences concern reaction times rather than accuracy. The children with dyslexia were as accurate as TDC of the same age in grammaticality judgement task. Despite the fact that they were not less accurate, they were slower than age-matched peers in judging the violated and canonical sentences. Thus, children with dyslexia seem to be delayed and not deviant from the syntactic development point of view.

The comparison of performance of TDC and children with dyslexia was made on ERP measures. The analysis revealed results on performance of TDC and children with dyslexia on ERP measures the median peak amplitude and latency of ELAN and P600 components. The findings revealed that Visual inspection of the waveforms

indicated that greater positivity was observed for the TDC when compared to DD children for the subject-verb agreement violations. The positivity was greater for violated sentences than the grammatical canonical sentences. The positivity was however greater for the anterior regions than the posterior or central regions. What was interesting observation was that the positivity was greater for left channels than the right channels of the anterior regions in TDC when compared to DD. This supposedly indicates that the processing of information in TDC for syntactic processing is controlled by language areas in both the hemispheres, which is more likely than what is observed in children with DD. Longer latencies in children with DD are indicative that processing grammatical information of words in sentences is slower in dyslexia (Leikin, 2002). The results of the study showed that ELAN was found in both TDC and DD, greater in the left anterior region. For P600, grammaticality effect was observed in the left and right parietal region in TDC. Both the TDC and DD showed significant effects of grammaticality in right posterior region and in the midline. There was a significant difference in the left posterior region for TDC. However, there was no significant grammaticality effect observed in DD.

The ELAN has been assumed to reflect a highly automatic processing phase of syntactic processing (Hahne & Friederici, 1999). The results of the present study indicate that the children with dyslexia also make use of this early mechanism and they are able to detect the violations like the TDC. The distribution pattern of P600 in response to sentence violations in DD was relatively similar to the TDC. The regions in the TDC in which a robust P600 was seen was also observed in children with DD as well. However there were differences found in certain regions as well. It was observed that in the grand average ERP that ungrammatical violated sentences elicited increased positivity than the grammatical canonical sentences. The findings also suggested that greater positivity in left posterior regions in TDC and right posterior in DD. In a study conducted by

Pugh et al., (2000) of a left temporo-parietal brain circuit to be disrupted in dyslexia. A functional problem in the left posterior region could be explained for weaker activation in left region and relative greater activation in right posterior region in dyslexia. Delayed linguistic processing as indicated by various studies (Brandeis et al., 1994; Helenius et al., 1999, and Leikin, 2002) could have resulted in longer latency responses in children with DD. Similar results are suggested fromthe behavioural data indicating slower processing time in children with DD. Greater restricted distribution of P600 further indicates less synchronous active neurons functioning (Rispens, 2004).

The findings of the current study have provided us doors to understand an on-line measure for assessment of syntactic processing through ELAN and P600, which could help to explore the level of processing deficit in children with dyslexia. Clinically, this seems to substantiate behavioral measure but also give a better description of the level of processing deficit in children with dyslexia. Identifying the level of processing deficit may facilitate specific intervention strategies by professionals in the clinical set up. Utilization of the findings from the present study may also serve as norms for children in the age range 8-10 years with Kannada as native language background.

Limitations of the study

Owing to the inclusion and exclusion criteria for participants in the current study, data was small and limited. A larger sample would give a better conception of generalization for results. The results can also be viewed for localization and/or lateralization of language functions through ERPs in the clinical population such as dyslexia.

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Appendix 1

List of Sentences

1. ರಾಮುಊರಿನಿಂದಬಂದನು.
/ra:mu u:rininda bandanu/.

2. ರಾಮುಊರಿನಿಂದಬಂದೆ.
/ra:mu u:rininda bande/.

3. ಗೀತಾನೀರನ್ನುಕುಡಿದಳು.
/gi:ta ni:rannu kuDida|u/ .

4. ಗೀತಾನೀರನ್ನುಕುಡಿದನು.
/gi:ta ni:rannu kuDidanu/.

5. ವಿಮಾನಆಕಾಶದಲ್ಲಿಹಾರುತ್ತದೆ.
/vima:na a:ka:ʃadalli ha:ruttade/.

6. ವಿಮಾನಆಕಾಶದಲ್ಲಿಹಾರುತ್ತಾಳೆ.
/vima:na a:ka:ʃadalli ha:rutta:|e/.

7. ಮಗುಆಟಆಡುತ್ತದೆ.
/magu a:Ta a:Duttade/.

8. ಮಗುಆಟಆಡುತ್ತಾಳೆ.
/magu a:Ta a:Dutta:|e /.

9. ನಕ್ಷತ್ರಗಳುರಾತ್ರಿಮಿಂಚುತ್ತವೆ.
/nakʃatraga|u ra:tri minʧuttave/.

10. ನಕ್ಷತ್ರಗಳುರಾತ್ರಿಮಿಂಚುತ್ತದೆ.
/nakʃatraga|u ra:tri minʧuttade/.

11. ಕಮಲಮನೆಗೆಬರುತ್ತಾಳೆ
/kamala manege barutta:|e/.

12. ಕಮಲಮನೆಗೆಬರುತ್ತಾನೆ.

/kamala manege barutta:ne/.

13. ಹಸುರಸ್ತೆಯಲ್ಲಿಬರುತ್ತಿದೆ.
/hasu rastejalli baruttide/.

14. ಹಸುರಸ್ತೆಯಲ್ಲಿಬರುತ್ತಾನೆ.
/ hasu rastejalli barutta:ne/.

15. ನಾವುಟಿ.ವಿ.ನೋಡುತ್ತೇವೆ.
/na:vu Ti.vi. nODutte:ve/.

16. ನಾವುಟಿ.ವಿ.ನೋಡುವಳು.
/na:vu Ti.vi. nODuvaɭu/.

17. ಗಣೇಶಕಥೆಯನ್ನುಓದಿದನು.
/gaNe:ʃa kat^{h}ejannu o:didanu/.

18. ಗಣೇಶಕಥೆಯನ್ನುಓದಿದಳು.
/gaNe:ʃa kat^{h}ejannu o:didaɭu/.

19. ನನ್ನಸ್ನೇಹಿತನಗುತ್ತಾನೆ
/nanna sne:hita nagutta:ne/.

20. ನನ್ನಸ್ನೇಹಿತನಗುತ್ತಾಳೆ.
/nanna sne:hita nagutta:ɭe/.

21. ರಾಮಬಾಲನ್ನುಎಸೆದನು.
/ra:ma ba:lannu esedanu/.

22. ರಾಮಬಾಲನ್ನುಎಸೆದಳು
/ra:ma ba:lannu esedaɭu/.

23. ಅವನುಅಂಗಡಿಗೆಹೋದನು
/avanu angaDige hOdanu/.

24. ಅವನುಅಂಗಡಿಗೆಹೋದಳು.
/avanu angaDige hOdaɭu/.

25. ರಾಜ ಬೇಟೆ ಆಡುತ್ತಿದ್ದಾನೆ.
/ra:ʤa be:Te a:Duttidda:ne/.

26. ರಾಜ ಬೇಟೆ ಆಡುತ್ತಿದ್ದಾಳೆ.
/ra:ʤa be:Te a:Duttidda:ɭe/.

27. ಅವಳು ಹಾಲು ಕುಡಿತಾ ಇದ್ದಾಳೆ
/avaɭu ha:lu kuDijutta: idda:ɭe/.

28. ಅವಳು ಹಾಲು ಕುಡಿಯುತ್ತೇ
/avaɭu ha:lu kuDijutte:/.

29. ಅವರು ಜಗಳ ಆಡುತ್ತಾರೆ.
/avaru ʤagaɭa a:Dutta:re/.

30. ಅವರು ಜಗಳ ಆಡುತ್ತೀಯಾ.
/avaru ʤagaɭa a:Dutti:ja/.

31. ನಾನು ಮನೆ ಕಟ್ಟುತ್ತೇನೆ.
/na:nu mane kaTTutte:ne/.

32. ನಾನು ಮನೆ ಕಟ್ಟುತ್ತಾರೆ.
/na:nu mane kaTTutta:re/.

33. ನೀನು ಚಪ್ಪಾಳೆ ಹೊಡೆಯುತ್ತೀಯಾ.
/ni:nu cappa:ɭe hoDejutti:ja/.

34. ನೀನು ಚಪ್ಪಾಳೆ ಹೊಡೆಯುತ್ತೇನೆ.
/ni:nu cappa:ɭe hoDejutte:ne/.

35. ಅವರು ಮೀನನ್ನು ಹಿಡಿಯುತ್ತಾರೆ.
/avaru mi:nannu hiDijutta:re/.

36. ಅವರು ಮೀನನ್ನು ಹಿಡಿಯುತ್ತೀಯಾ.
/avaru mi:nannu hiDijutti:ja:/.

37. ನಾಳೆ ಹುಡುಗಿ ಹಾಡುತ್ತಾಳೆ
/na:ɭe huDugi ha:DUtta:ɭe/.

38. ನಾಳೆ ಹುಡುಗಿ ಹಾಡಿದಳು.
/na:ɭe huDugi ha:Didaɭu/.

39. ಅವನು ನಾಳೆ ಬರುತ್ತಾನೆ.
/avanu na:ɭe barutta:ne/.

40. ಅವನು ನಾಳೆ ಬಂದನು.
/avanu na:ɭe bandanu/.

41. ಸೀತೆ ನಿನ್ನೆ ತಿಂದಳು.
/si:te ninne tindaɭu/.

42. ಸೀತೆ ನಿನ್ನೆ ತಿನ್ನುತ್ತಾಳೆ.
/si:te ninne tinnutta:ɭe/.

43. ರಾಮ ಊರಿಗೆ ಹೋಗುತ್ತಾನೆ.
/ra:ma u:rige hogutta:ne/.

44. ರಾಮ ಊರಿಗೆ ಹೋದನು.
/ra:ma u:rige ho:danu /.

45. ಹಕ್ಕಿ ನಿನ್ನೆ ಹಾರುತ್ತಿತ್ತು.
/hakki ninne ha:ruttittu/.

46. ಹಕ್ಕಿ ನಿನ್ನೆ ಹಾರುತ್ತಿದೆ.
/hakki ninne ha:ruttide/.

47. ಸುರೇಶ ಶಾಲೆಗೆ ಹೋಗುತ್ತಾನೆ.

/surEʃa ʃa:lege ho:gutta:ne/.

48. ಸುರೇಶ ಶಾಲೆಗೆ ಹೋಗುತ್ತಿದೆ
/surEʃa ʃa:lege ho:guttide/.

49. ನಿನ್ನೆ ರಾತ್ರಿ ಮಳೆ ಬಂತು.
/ninne ra:tri maḷe bantu/.

50. ನಿನ್ನೆ ರಾತ್ರಿ ಮಳೆ ಬರುತ್ತಿದೆ.
/ninne ra:tri maḷe baruttide/.

51. ನಿನ್ನ ಪುಸ್ತಕವನ್ನು ನಿನ್ನೆಕೊಟ್ಟೆ.
/ninna pustakavannu ninne koTTe/.

52. ನಿನ್ನ ಪುಸ್ತಕವನ್ನು ನಿನ್ನೆ ಕೊಡುತ್ತೇನೆ.
/ninna pustakavannu ninne koDutte:ne/.

53. ನೆಂಟರು ಕಳೆದವಾರ ಹೋದರು.
/nenTaru kaḷedava:ra ho:daru/.

54. ನೆಂಟರು ಕಳೆದವಾರ ಹೋಗುತ್ತಾರೆ.
/nenTaru kaḷedava:ra ho:gutta:re/.

55. ನಾಳೆ ಊರಲ್ಲಿ ಹಬ್ಬ ಇದೆ.
/na:ḷe u:ralli habba ide/.

56. ನಾಳೆ ಊರಲ್ಲಿ ಹಬ್ಬಇತ್ತು.
/na:ḷe u:ralli habba ittu/.

57. ಹುಡುಗಿ ನಿನ್ನೆ ತಿಂದಳು.
/huDugi ninne tindaḷu/.

58. ಹುಡುಗಿ ನಿನ್ನೆ ತಿನ್ನುತ್ತಾಳೆ.

/huDugi ninne tinnutta:ɭe/.

59. ರಸ್ತೆಯಲ್ಲಿಕಾರುಹೋಗುತ್ತಿದೆ
/rastejalli ka:ru ho:guttide/.

60. ರಸ್ತೆಯಲ್ಲಿಕಾರುಹೋಗುತ್ತಿವೆ.
/rastejalli ka:ru ho:guttive/.

Appendix 2

Table 3.1

Participant details

Groups	**N**	**Mean age (in years;months)**	**SD**	**Number of males**	**Number of females**	**Language (based on LPT)**
Control group	30	9;4	0.21	24	06	Age adequate
Clinical group	15	9.2	0.36	11	04	Age adequate

Table 3.2

Profile of Participants in the Clinical Group (N=15)

Participants	Age (in years; months)	Gender	Language scores (on LPT)	ERS results
Participant 1	9;6	M	241	I-II
Participant 2	10;0	M	261	I-III
Participant 3	9;4	F	239	I-III
Participant 4	8;6	M	253	I
Participant 5	9;3	F	260	I-II
Participant 6	8;9	M	240	I-II
Participant 7	9;5	M	256	I
Participant 8	9;6	M	241	I-II
Participant 9	9;1	M	236	I
Participant 10	9;0	F	260	I-II
Participant 11	8;7	M	244	I-II
Participant 12	8;2	F	254	I-II
Participant 13	9;5	M	240	I-II
Participant 14	9;6	M	251	I-II
Participant 15	8;7	M	241	I-II