

Autism Spectrum Disorder and Co-Existing Conditions: A Lexical Decision ERP Study

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Abstract

The current study sought to clarify the nature of lexical decision-making information processing, using a lexical decision paradigm during EEG, in 4 groups: pure-ASD; pure-ADHD; pure-anxiety; and neurotypical controls. We also aimed to understand whether there were differences between groups when ASD presents as a comorbid condition (ASD + ADHD). The P100 and the N170 components of the evoked potential (ERPs) were the focus of analyses. Overall, we found larger P100 amplitudes in the right (relative to the left) hemisphere in neurotypical controls. This early ERP component likely reflects pre-linguistic processing (e.g., the sorting of nouns into categories) at a stage before the language-dominant left hemisphere takes over. We also found that those with pure-ADHD had longer P100 latencies than both the pure-anxiety and pure-ASD groups towards all lexical stimuli. The pure-ADHD group also showed smaller amplitudes toward word stimuli than toward pseudowords and nonwords. The ASD + ADHD group had significantly longer latencies towards pseudowords than the pure-ASD group. A unique pattern of ERPs was therefore observed in the comorbid group, which suggests that the two conditions are separate. This finding is in accord with the latest revision of the Diagnostic and Statistical Manual of Mental Disorders (DSM-V).

Keywords: ADHD, ASD, EEG, Neurotypical, ERP

Introduction

Autism spectrum disorder (ASD) is a lifelong neurodevelopmental condition for which there is no known cause or cure. Autism is a highly variable disorder, the most prominent difficulties of which include aberrant behaviour, poor social skills and disrupted communication skills [1]. Difficulties in social communication and interaction feature in the diagnostic criteria for ASD [1]. While language difficulties are a core characteristic of autism, as with the disorder itself, linguistic functioning can be highly variable within those on the spectrum. Some children develop fluent speech while others never begin to speak at all. Verbal and nonverbal communications are vital the formation of social interactions [2], and thus, any investigation into the nature of linguistic difficulties in ASD may help us to further understand basis of the condition.

Language difficulties in ASD, ADHD and Anxiety

Interestingly, impediments in linguistic information processing have also been documented in attention deficit hyperactivity disorder (ADHD) [3], a condition that commonly occurs together with ASD. Investigating the shared nature of linguistic processing difficulties (if any) in conditions that commonly occur together will contribute to a larger research effort to identify whether coexisting conditions in ASD stem from the same basic cause (i.e., are additional conditions simply characteristics of the ASD itself), or are entirely separate conditions (i.e., the coexisting conditions are true separate conditions).

Difficulties with language in ASD may be driven by poor language acquisition and processing. These include problems with phonology [4]; semantics [5]; syntax [6] and prosody [7]. Those with ADHD are known to have pragmatic language deficits and are significantly more likely to have a language disorder such as Specific Language Impairment or dyslexia [8], a profile that is also seen in ASD [9]. These difficulties contribute to problems with academic achievement and cognition in ADHD [10]. To the best of our knowledge, there are no known deficits in linguistic information processing in those with generalised anxiety

disorder. ADHD and anxiety commonly occur together with ASD as a comorbid condition [11]. Exploration of a core deficit evident in both conditions will assist to further investigate the shared nature of these conditions when they occur together.

Brain Networks Involved in Language Difficulties

While language difficulties in ASD are well documented, there are no clear links to underlying the brain networks involved. fMRI studies show that individuals with autism produce more activation in the Wernicke's area (located in the superior temporal gyrus and involved in the understanding of speech and language), but less in the Broca's area (located in the frontal lobe and involved in the production of speech and language) than typically developing controls [12]. Further, this study found that patterns of functional connectivity between cortical areas involved in language processing are significantly reduced in those with autism than in typically developing controls [13]. Taken together, this evidence suggests dysfunctional integration of information related to language in autism.

In ADHD, difficulties may be driven by atypical hemispheric laterality. Most right-handed neurotypical adults have left hemisphere dominance for language [14], therefore a more symmetrical pattern of activation or a dominant right hemisphere, is considered atypical. One study demonstrated that individuals with ADHD were impaired relative to typically developing controls in discriminating words and

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Received: September 17, 2015; Accepted: September 19, 2015; Published: October 20, 2015

Citation: Saunders A, Kirk IJ, Waldie KE (2015) Autism Spectrum Disorder and Co-Existing Conditions: A Lexical Decision Erp Study. Clin Exp Psychol 1: 101. doi:10.4172/cep.1000101

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non-words on a lexical decision task. This impairment was mapped to reduced left hemisphere, and increased right hemisphere involvement during the task compared to neurotypical controls [15]. Research has also found atypical lateralisation of language in those with ASD [16].

Measuring Language Difficulties Using EEGG

A common approach to investigate how the brain processes stimuli is through the use of electroencephalography (EEG). The event related potentials (ERPs) that are recorded using EEG give information about the speed in which certain stimuli are processed. A typical ERP consists of a composite waveform of positive and negative components that are labelled according to their polarity and post-stimulus latency. The amplitude at the peak of each positive or negative waveform is argued to index the neural resources devoted to a specific task at any one time [17]. Larger amplitudes reflect more neural activity and are correlated with the amount of cognitive effort required [18]. The post-stimulus time at which a peak occurs after is known as the latency, and is argued to measure processing speed [19]. Early ERP components, such as the N150 that occurs 130-150ms post stimulus, mark the automatic lexical classification of a word (Spironelli & Angrilli, 2009). Late ERP components like the N300 or the N400 are related to phonological and semantic processing, respectively [20].

ASD children with very disrupted linguistic functioning show markedly smaller N1 amplitudes in response to auditory evoked stimuli than neurotypical controls [21]. Another study that investigated semantic processing in ASD by measuring the N400 component, found that the N400 was significantly higher in neurotypical controls versus those with autism in response to musical or linguistic stimuli [22]. Therefore, while there has been research to demonstrate altered N1 and N400 ERP's in autism in response to various auditory stimuli, it is unclear whether the ERP waveform will differ from neurotypical controls in response to a lexical decision task. In ADHD, ERP research in the absence of specific language impairment is rare. Resting state EEG research does however demonstrate increased N100 latencies and decreased P300 amplitudes in those with ADHD [23].

In addition, a study that investigated laterality by measuring ERP's in each hemisphere during a simple reaction time experiment found that those with ADHD demonstrated that compared with controls, attention-deficit/hyperactivity disorder-combined participants demonstrated significantly faster left-to-right transfer, whereas attention-deficit/hyperactivity disorder-inattentive participants had significantly slower right-to-left transfer [24]. More information is needed to establish the nature of ERP's with specific relation to lexical stimuli in ADHD without specific language impairment.

Aims and Hypotheses

The current study aimed to determine whether there is an underlying neurological difference in the processing of lexical information in any of the three experimental groups compared to each other and to neurotypical controls, using a lexical decision paradigm.

First, we expected to see the shortest latencies and/or largest amplitudes in the left hemisphere for the word condition in the neurotypical controls, given that the left hemisphere is specialised for language processing. We also expected to see shorter latencies and/or larger amplitudes in the right hemisphere, relative to the left, for the ADHD group, given that language previous research has demonstrated that language may not be typically lateralised in ADHD. If language is typically organised in ASD or anxiety then we would expect to see

typical latencies and/or amplitudes in these groups.

Second, we expected to see shorter latencies for word- relative to non-word stimuli in neurotypical controls. The P100 and the N170 would also be larger in the left hemisphere for word- relative to non-word stimuli. If language is not typically organised in ADHD (or in the other groups), we would not expect to see this left hemisphere preference for words relative to non-words.

Third, if the comorbid group is better conceived of as one condition and not two, we would expect no significant group differences in either the hemisphere, latency, or the amplitude of the N170 or the P100 (between the ASD-pure and ASD-comorbid groups).

Methods

Participants

The initial sample consisted of 83 subjects. There were 10 subjects that were excluded from the final analyses: English not first language $n = 1$; dyslexia $n = 1$; not enough correct trials $n = 8$. The final sample consisted of 73 subjects that were split into four experimental groups (ADHD $n = 21$; anxiety $n = 12$; ASD $n = 20$; control $n = 20$). All subjects had normal or corrected-to-normal vision and no history of head injury. The overall sample consisted of 37 females and 36 males. However, within each group the sex ratio was not evenly split (Table 1). This imbalance is in line with literature which suggests autism has a 4.3:1 ratio for boys to girls [25], and anxiety is much more common in females than males [26,27]. The mean age of all participants was 25.86 years, with a range from 16.3 years to 54.2 years.

Recruitment

Participants were recruited using advertisements placed around the University of Auckland City Campus and on a participant recruitment website. Subjects were also recruited through organisations such as Altogether Autism, the Phobic Trust, the Parent and Family Centre and the ADHD Association. Once subjects contacted the researcher with their interest in the study, they were given an initial questionnaire to ensure they fit the study criteria. Participants must have no history of head injury, no history of comorbid depression or schizophrenia, be right-handed and have English as their first language. Participants were given \$20 in vouchers for their participation in this study.

Control subjects consisted of mainly undergraduate students and had no history of any psychological illness (e.g., depression, anxiety). Subjects who fell into the experimental groups were diagnosed with their respective conditions by a registered medical professional prior to participation. From the 21 subjects in the ADHD group, 4 had an additional diagnosis of anxiety. In the ASD group, 6 subjects had a diagnosis of high functioning autism with no coexisting conditions; 10 had a diagnosis of high functioning autism and coexisting anxiety or OCD; and the remaining 4 had a diagnosis of coexisting high functioning autism and ADHD. The anxiety group included those with a diagnosis of anxiety or OCD.

	ADHD		anxiety		ASD		control	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Age	26.72	(7.81)	25.99	(8.13)	26.11	(10.16)	24.65	(6.61)
IQ	117.95	(8.2)	117.3	(8.46)	110.25	(11.84)	111.37	(12.24)
Female	13		9		5		9	
Male	9		3		15		20	

Table 1: Mean age, IQ and gender distribution for each experimental group in the lexical decision study.

Stimuli

Stimuli consisted of black words or non-words centrally presented one at a time on a computer monitor. The non-word condition was split into two types, legitimate non-words (i.e., non-words that follow English language rules) and illegitimate non-words (i.e., words that do not follow English language rules), as shown in Table 2.

Thus, there were three conditions in the experiment: real words; pseudowords; and non-words. All words were generated using the MRC Psycholinguistic Database (Wilson, 1998), and consisted of common English nouns. All non-word stimuli were presented using the ARC non-word database (Rastle, Harrington & Coltheart, 2002). All words were either 4 or 5 letters long. Words were presented in black Times New Roman Font point-size 72 in the centre of a blank screen on E-prime software [28] and were displayed on a colour computer monitor with a screen resolution of 800 x 600 pixels. Overall, participants responded with the correct response 94.17% of the time.

Procedure

The experiment consisted of two trial blocks plus one practice block. Each block consisted of 120 trials, making the total number of trials for each participant 240. Participants were asked to respond with a different hand for each block (either left or right hand) and the hand for the first block was counterbalanced among participants (i.e., either respond with left or right hand first). The stimuli appeared on the screen for 800ms and was followed by a 1500ms inter-stimulus interval. The study design followed a 'go no-go' paradigm, where participants were asked to respond by pressing the space bar if they thought the word was a real English word, and to not respond if it was a non-word.

EEG Acquisition

EEG recordings were conducted in an electrically shielded room (Model L3000; Belling Lee, Enfield, England) using 128-channel Ag/AgCl electrode nets [29]. The Geodesic sensor net distributes electrodes from nasion toinion and from left to right mastoids at uniform intervals. EEG was recorded continuously (1000-Hz sample rate; 0.1–100 Hz analogue bandpass) with Electrical Geodesics Inc. amplifiers (300-M Ω input impedance). A MacIntosh computer was used to acquire the data using NetStation software and this was then stored on the computer's hard disk. Electrode impedances were kept below 40 k Ω , an acceptable level for this system [29]. Common vertex (Cz) was used as a reference, resulting in a total of 129 electrodes. Triggers were programmed into E-Prime and synchronised with the EEG acquisition software via a parallel port. Triggers were sent as a function of word type and hand

(six in total: real words, pseudowords and non-words for left and right hands) at stimulus onset, and again upon response (two types: correct, or incorrect), thus resulting in two triggers per trial.

EEG Processing

Data was segmented using custom in-house software (WinView) into ERP waveforms with any artifacts removed according to the guidelines set out in [30]. EEG data was segmented as a function of trigger type. Data included both correct and incorrect trials to increase power (results were not significantly different when incorrect trials were omitted). Each epoch was centred around the stimuli trigger (i.e., the trigger that was sent when the stimuli was displayed). Each epoch was 100ms pre stimulus and 500ms post-stimulus giving a total epoch segments of 600ms. Any epochs with bad channels, eye blinks, too much drift or noise were discarded from analyses. From the 237 trials for each participant, 217.93 epochs on average were accepted. Epochs were collapsed across hand and averaged as a function of word type (Real word, Pseudoword and non-word) giving a total of 3 ERP's. These ERP's were then grand-averaged and filtered using a 30-Hz low-pass Butterworth Filter [31].

ERP Analysis

The three grand averaged waveforms for each participant were visually inspected for the P100 and N170 component. The N170 is a large negative deflection at occipito-temporal sites approximately 140 - 190ms after stimulus onset [32]. The electrodes selected for 59, 64, 65, 66, 68, 69, 70, 83, 84, 89, 90, 91, 94 and 95. These electrodes are located in the occipito-temporal region and are consistent with the electrodes used in similar research [33,34]. From each ERP the amplitude and latency of the P100 (i.e., the first most positive peak). For the N170, the amplitude and latency of the first most negative peak (i.e., the most negative deflection) was collected for each participant, for each hemisphere (left and right).

Statistical Analyses

Data were analysed using SPSS version 20 software. The main statistical technique used was mixed analysis of variance (ANOVA). Initial analyses showed no significant main effects or interactions with gender. Therefore, analyses were collapsed across this dimension to increase statistical power. All p-values were considered significant at the .05 level. Follow-up analyses were conducted with *Bonferroni* correction to the alpha level.

Results

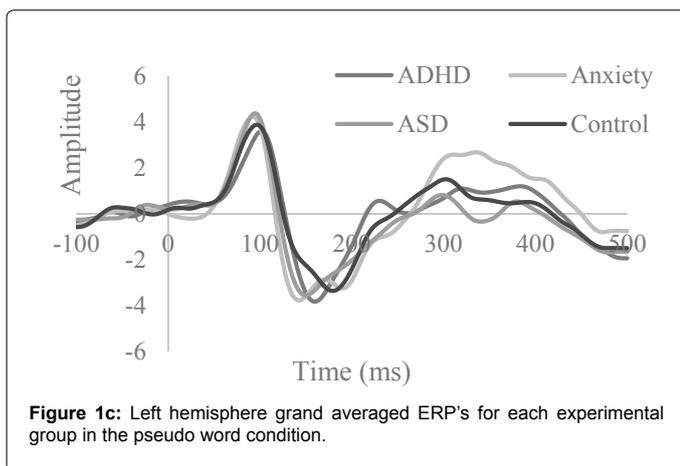
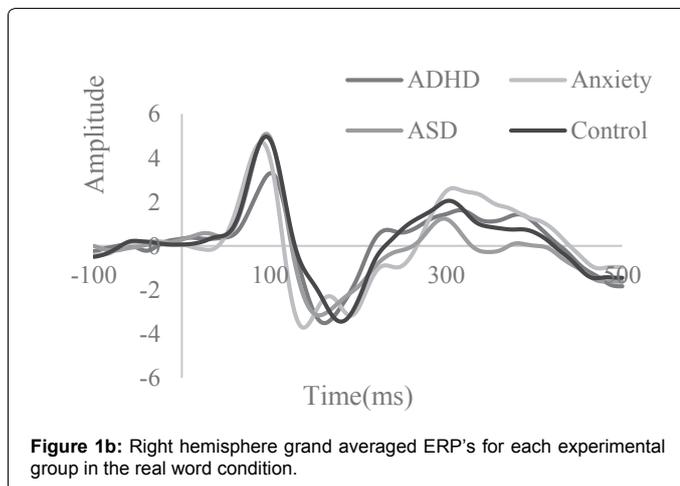
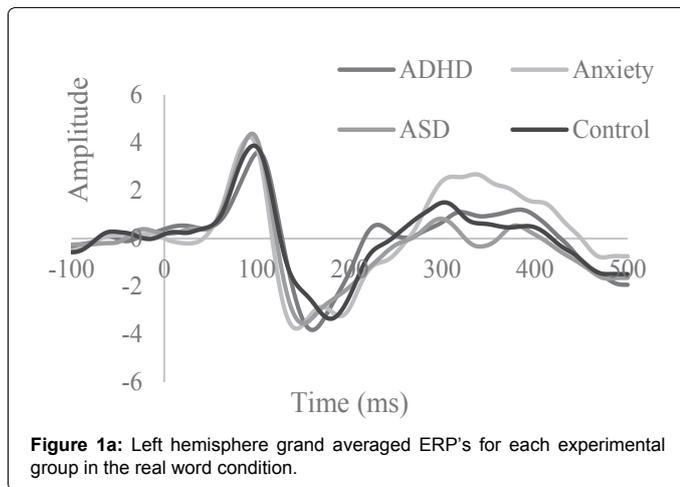
A one-way ANOVA was conducted to investigate whether there were any differences in accuracy in the three conditions (real-word, pseudoword, illegitimate word) for each experimental group. There were no significant group differences. Grand averaged ERPs (across the P100 and N170 components) for each pure experimental group and for each hemisphere and condition are illustrated in Figures 1a–f.

A 4 (group) x 2 (hemisphere) x 3 (pseudoword, real word, illegitimate word) repeated measures ANOVA was conducted on the latency of the P100. There was a significant main effect of group, $F(3,67) = 3.44, p = .022$. Those with pure-ADHD had longer P100 latencies ($M = 106.08, SE = 2.61$) than both the anxiety ($M = 96.14, SE = 2.61, p = .046$) and ASD ($M = 94.35, SE = 3.37, p = .05$) groups. There were no other significant effects.

A 4 (group) x 2 (hemisphere) x 3 (pseudoword, real word, illegitimate word) repeated measures ANOVA was conducted on

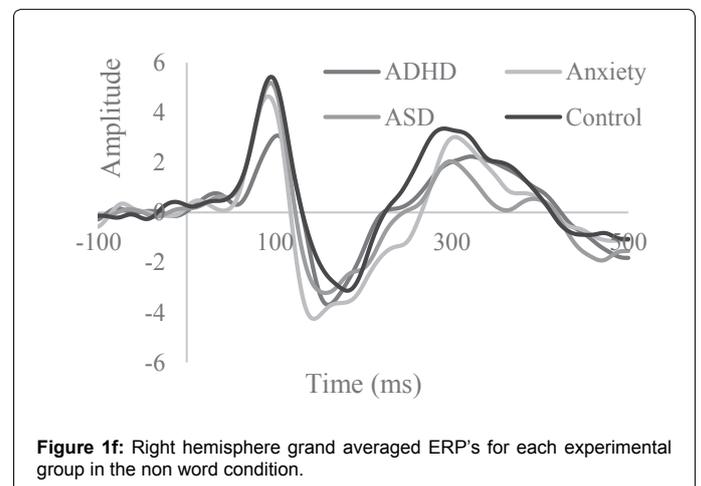
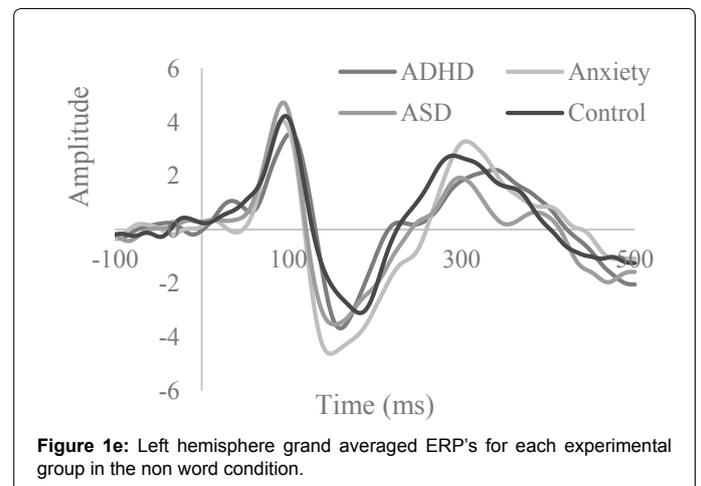
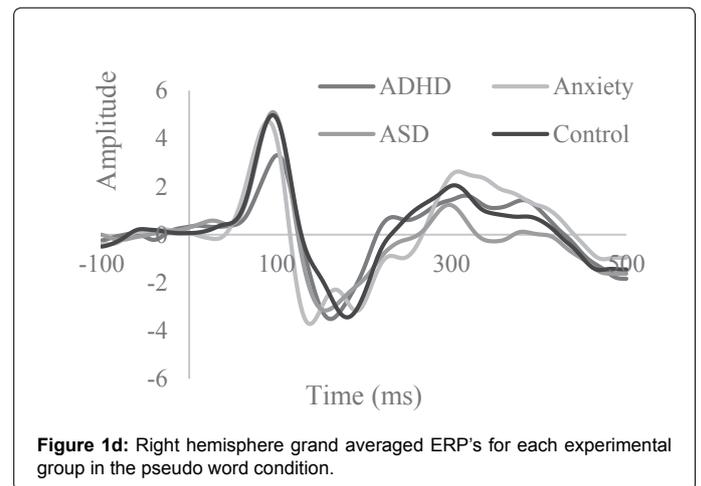
Real word condition	Pseudoword condition	Non word condition
Fruit	Whols	cauv
Pansy	Clett	esuyp
Pizza	Fenth	siopp
Squid	Vacso	ycli
mango	Sevit	defv
Olive	Defas	loqu
Spoon	Noxu	oofl
Horse	Yave	knyze
Panda	Rona	makt
Coral	Muge	tapht
Thyme	Frit	wynts
Crown	Cari	ghict
thumb	Dide	sloiz

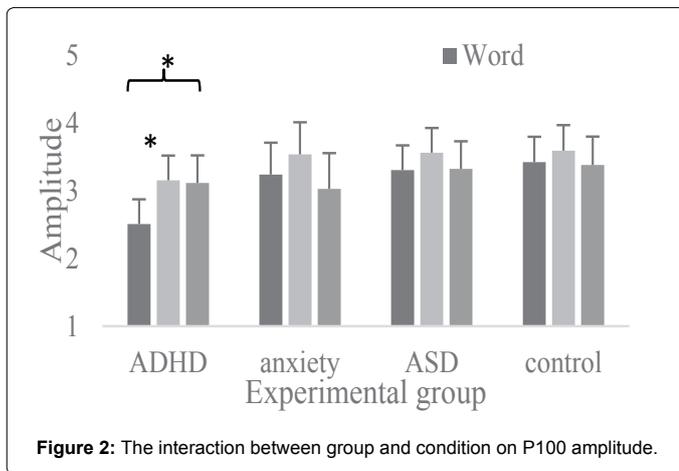
Table 2: Examples of the stimuli used for each condition in the lexical decision experiment.



the amplitude of the P100. There was a significant main effect of hemisphere, $F(1,67) = 7.08, p = .010$, where amplitudes were larger in the right ($M = 3.48, SE = .22$) than left hemisphere ($M = 3.05, SE = .21$). There was a significant main effect of condition, $F(2,67) = 6.75, p = .002$, where the P100 amplitude was significantly larger in the pseudoword condition ($M = 3.46, SE = .20$) than in the word condition ($M = 3.12, SE = .20, p = .001$) and the nonword condition ($M = 3.21, SE = .22, p = .047$). There was also an interaction between condition

and experimental group that was approaching significance, $F(6,67) = 1.93, p = .081$ (Figure 2). For the ADHD group only, amplitudes toward word stimuli ($M = 2.51, SE = .37$) were significantly smaller than toward pseudowords ($M = 3.16, SE = .37, p = .001$) and nonwords ($M = 3.12, SE = .41, p = .004$). In addition, for the control group only, amplitudes in the right hemisphere ($M = 3.89, SE = .42$) were significantly larger than in the left hemisphere ($M = 3.04, SE = .39, p = .007$).





Two separate 4 (group) x 2 (hemisphere) x 3 (pseudoword, real word, illegitimate word) repeated measure ANOVAs were conducted on the latency and the amplitude of the N170. There were no significant main effects or interactions on either the latency or the amplitude.

With regard to comorbidity, one-way ANOVAs were conducted on the latency of the P100 for each word condition separately. In the real word condition, there was a significant effect of group, ($F(2,26) = 6.81, p = .005$), where the pure-ADHD group had significantly longer latencies than the pure-ASD group ($p = .003$). In the Pseudoword condition there was also a significant main effect of group, ($F(2,26) = 4.48, p = .022$), where the pure-ADHD group had significantly longer latencies than the pure-ASD group, ($p = .05$), and the ASD + ADHD group had significantly longer latencies than the pure-ASD group ($p = .039$). In the nonword condition, there was a significant effect of group, ($F(2,23) = 4.85, p = .019$), where the pure-ADHD group had significantly longer P100 latencies than the pure-ASD group ($p = .02$).

One-way ANOVAs were also conducted to investigate if there were differences between the comorbid versus pure groups on the latency or amplitude of the N170 in each condition. There were no significant effects in any condition.

Discussion

The main aim of the current study was to investigate whether there are neurophysiological differences in lexical information processing between ASD, ADHD, anxiety and neurotypical controls, as measured by ERPs in a lexical decision paradigm. The second aim of the current study was to investigate the role of comorbidity in ASD by determining whether any differences in lexical information processing are altered when examining the pure conditions versus the comorbid condition (i.e., ASD + ADHD). Overall, we expected to see larger P100 or N170 amplitudes and shorter P100 or N170 latencies in the left hemisphere for the control group. If lexical processing deficits were present in ADHD, ASD and/or anxiety, then we expected to see a different pattern of amplitudes and latencies to the control group.

Our results showed that, in the control group, P100 amplitudes were larger in the right hemisphere and there were no significant hemisphere differences between latencies. This early, right-sided dominance was not shared, to the same extent, by the ASD, anxiety or ADHD groups. We expected to see a pattern of left hemisphere specialisation for the control group in particular given previous research [35].

There are three main possibilities for this finding. It is most likely

that the P100 component reflects pre-lexical processing, whereas later components reflect lexical processing. A study investigating early and late ERP components in lexical decision making found significantly larger ERP's in the right over the left hemisphere in the P100 component, with left hemisphere specialisation only occurring in later components [36]. This suggests that the left hemisphere does not take over the processing of lexical information until 200-300ms after stimulus presentation. Later ERP components reflect processing of lexical information, rather than recognition [37]. This theory is supported by the fact that the electrodes at which the maximal P100 is seen in the current study are located over the occipito-temporal region of the brain, an area responsible for early visual processing [38]. Further support for this theory will be outlined with reference to the N170 component later in the discussion.

The second possibility is closely related to the first. It is possible that the P100 reflects participants' organisation of the stimuli into categories rather than the processing of this information. One body of research suggests the role of the right hemisphere in language is underestimated. The right hemisphere may be involved in categorisation and recognition of language, whereas the left hemisphere is involved in the processing and production of lexical information [39]. To illustrate, one study found larger N170 components in the right over the left hemisphere when categorising objects in your area of expertise (e.g., knowledge of dogs or cats) [40]. The words used in the current study 'English condition' were nouns. Participants may have visualised the object being named and used a 'sorting technique' to classify it as a real English word, rather than process the lexical information. Thus, the larger P100 ERP in the right hemisphere could reflect neural activity involved in sorting, rather than analysing the lexical value of the stimuli when using nouns.

Third, smaller latencies of the P100 in the left hemisphere could reflect a more streamlined neural process for the recognition of lexical stimuli. That is, the left hemisphere does not need to exert cognitive effort at this early stage, which only involves recognition of the stimuli. In support of this hypothesis, EEG studies have demonstrated that the amplitude of the P100 in lexical decision tasks is modulated by word length, that is shorter words have a smaller P100 than larger words [41,42]. As the words in the current study were 4-5 letters in length, it is possible that little cognitive effort was required to recognise these words, and thus a lower amplitude P100 was produced. However, given the established body of research demonstrating left-hemisphere dominance for lexical processing, this 'streamlined theory' is perhaps less likely than those suggested above.

Despite the unexpected pattern of amplitudes in the control group, the ASD, anxiety and ADHD group did not show the same pattern of ERP's. In these groups, there were no significant differences between the left and right hemispheres in the amplitude or latency of the P100. Given that this pattern of ERP's is different to that seen in neurotypical controls, it is likely that there is a dysfunction in the recognition of lexical stimuli present in these groups. This is in line with previous literature that has demonstrated patterns of atypical laterality for those with ADHD Rolfe et al., and ASD [16]. An overall dysfunction of brain systems may contribute to this atypical pattern of brain activity. If the right-lateralised P100 reflects a right hemisphere specialisation for pre-linguistic processing, and this is not evident in ADHD, this may explain the high rate of language impairment seen in ADHD [8]. A lack of differentiation between the right and left hemisphere in the P100 suggests that the ADHD brain may not recruit specialised brain networks for non-linguistic versus linguistic processes.

Visual inspection of the ERPs, from Figure 1, indicate that neurotypical controls show a similar pattern to that of the pure-ASD and pure-anxiety groups. As some participants in the ASD group had a comorbid diagnosis of ADHD, the atypical pattern of lateralisation may, in part, be explained by the addition of ADHD. However, it is also possible that there is a specific deficit in the P100 associated with ASD only. While no significant differences were found in the pure-anxiety group, it is possible that this can be explained by lack of statistical power, rather than a true effect given the small sample size of pure-anxiety group.

In addition, analysis of the P100 revealed an overall longer latency for the pure-ADHD group as opposed to the pure-ASD and pure-anxiety groups. Slow processing speed, defined as the overall speed of completing a task while maintaining accuracy, is a core deficit in those with ADHD and significantly impacts reading fluency by Jacobson et al. [44]. The overall longer latencies shown by those with ADHD would certainly reflect this slower processing speed.

Overall analysis of the ERPs also revealed that, for the ADHD group only, there were significantly smaller P100 amplitudes toward the word stimuli as opposed to the non-word or pseudoword stimuli. This could indicate a specific deficit in the recognition of linguistic stimuli over other visual stimuli. As previously stated, smaller amplitudes may reflect reduced neural resources dedicated to that process [17]. An inability to devote neural resources in the pre-linguistic processing stage would certainly have lead-on effects for when more effortful linguistic processing systems were to take over. Thus contributing to an overall pattern of lexical difficulties in those affected.

There was also an overall effect of (larger) amplitude evoked by pseudowords relative non-words or real words. This finding fits with the current theory that larger amplitudes reflect more cognitive effort. It is relatively easy to distinguish real English words and non-words (that have a non-typical structure) from one another, but distinguishing a pseudoword (a non-word that has a typical English language structure) is more difficult. Research has demonstrated that the recognition of pseudowords produces significantly more brain activation than real words [43]. The overall larger amplitudes evoked by pseudowords likely reflects the effort required to process the information and assign the word a correct classification.

The pure-ADHD group showed significant longer N100 latencies compared to the pure-ASD group in all three conditions, indicating that ADHD and ASD have different ERP profiles evoked in the recognition of lexical information. While both (pure) conditions are associated with lexical processing difficulties, these difficulties appear to stem from different origins. It is not uncommon for the same phenotype (e.g., difficulties with language) to be driven by different cognitive difficulties in individuals with developmental disorders (Ozonoff & Jensen, 1999). Lexical decision processing deficits in ADHD are associated with longer latencies, which is associated with slower processing speed, whereas slow processing speed was not evident in ASD. This finding is in line with the idea that slow processing speed is a core deficit in those with ADHD [44], and hypersensitivity to stimuli is a key characteristic of those with ASD [45]. Thus the phenotype of linguistic difficulties, while shared by both conditions, stems from distinct underlying neurological deficits in each condition.

There were no significant Group or Condition effects on either the N170 latency or amplitude. This was unexpected, given that this ERP component is thought to reflect lexical classification of a word, and earlier studies have found that this is affected in ADHD and ASD in particular [21,23]. Visual inspection of the ERP waveforms (and

means and standard deviations for each experimental group) did show some differences in this component. These differences may have not reached significance due to low statistical power. For example, for neurotypical controls, the latency of the N170 was earlier in the left than the right hemisphere for the word condition only. As this hemisphere specialisation was evident in the word condition, and not the pseudoword or nonword conditions, this suggests that the left hemisphere is beginning to take over lexical processing at about 200 ms post-stimulus (i.e., words are not processed immediately in the left hemisphere in particular).

The current findings also suggests that when ASD and ADHD present together, they are indeed separate conditions [46], and early lexical processing is differentially affected in this group versus those with a pure version of each condition. Therefore, interventions targeted to those with comorbid ASD and ADHD need to be aware that a treatment plan that is appropriate for those with ASD or ADHD alone may not be appropriate or effective in this population. For example, children with a comorbid ADHD + ASD diagnosis may benefit from the same behavioural interventions to improve linguistic functioning as those with ADHD alone. Similarly, therapies that improve overall processing speed may be beneficial for those with a comorbid diagnosis, even though slower processing is not a core deficit of those with ASD alone.

Limitations and Conclusions

The most important limitation of the current study is the small sample sizes of each group, particularly the comorbid ASD + ADHD group. This likely contributed to non-significant effects on the N170 component due to lack of statistical power. A larger scale study may have allowed for a more robust statistical analysis.

The current study has a number of strengths. Firstly, the use of concurrent EEG during lexical decision making allowed us to measure the underlying neurological activity in millisecond timing. We found, for example, that the right hemisphere is involved in very early lexical processing. However, our task was not designed to specifically manipulate the conditions. Future research into the exact function of early ERP components is needed to establish whether these components are indeed involved in pre-lexical processing, and to clarify the role of the right hemisphere involved in this processing.

This study demonstrates the importance of measuring factors related to pre-linguistic processing in those with a neurodevelopmental condition. If very early processing is altered in those with ADHD and ASD, then treatments targeted to improve linguistic functioning need to take this into account. Specifically, language deficits may not be associated with the processing of the lexical stimuli itself, but the (pre-lexical) process of recognising, categorising and sending the information to the appropriate brain networks. Therefore, more basic sensory integration therapies may be a more appropriate avenue for the treatment of word and nonword processing difficulties in these groups.

In summary, the current study found that a pattern of larger P100 amplitudes was found in the right versus the left hemisphere particularly in neurotypical controls. It is likely that this early ERP component reflects pre-linguistic processing, at a stage before the left hemisphere (which is specialised for language) takes over. Another possibility is that the P100 reflects the sorting of nouns into categories and this process is dominant in right-hemisphere networks. In addition, the atypical pattern of results demonstrated in the pure ADHD and ASD groups fit with the behavioural difficulty with language seen in these conditions. The pattern of ERPs, while different from neurotypical

controls, were also different in the two pure conditions. With regard to comorbidity, when ASD and ADHD was presented as a comorbid condition a unique pattern of ERPs was produced. The current study contributes to a growing body of evidence that suggests that, when ASD and ADHD are presented together, the difficulties cannot simply be accounted for by the ASD diagnosis. That is, ADHD should not be regarded as simply a more severe form of ASD when the two disorders are diagnosed in the same individual.

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