Attentional Blink Effects of Linguistic and Non-Linguistic Stimuli in Aphasia

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Abstract

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Aphasia literature has shown that aphasia is not a loss of language, but an impairment in the language processing system. Recent literature has suggested this breakdown in language performance must be explained by underlying cognitive mechanisms involved in accessing the representations and not the loss of the language information itself. The *attentional mechanism* has been suggested to fulfill the criteria of this impaired cognitive mechanism that could account for the deficits experienced by people with aphasia (PWA). An attentional blink (AB) task was used to gain further understanding of the role of the attentional mechanism in PWA as compared with neurotypical adults, in both linguistic and non-linguistic conditions. Analysis conducted investigated the strength, magnitude, and length of AB across participants and time intervals of stimulus presentation. Analysis revealed no significant differences between groups or conditions, likely due to small sample size and limited data points. Despite limitations, patterns observed showed that PWA had a tendency to higher susceptibility to AB than neurotypical participants, suggesting that it may be useful to continue investigating the relationship between attention and both linguistic and non-linguistic processing in PWA.

Introduction

Aphasia is an acquired neurogenic language disorder, usually occurring secondary to stroke in the left, language-dominant hemisphere. Disruption in the intricate processes necessary for language can manifest through many deficits, one of the most prominent deficits being anomia, or impaired word retrieval (Dell 1997). Extensive research has been conducted over the years investigating anomia and impaired mechanisms for language processing, as well as possible treatment procedures to overcome these deficits. In order to further develop treatment procedures, there is still a great deal to learn about these mechanisms crucial to language processing. This study was conducted with the intent of gaining a deeper understanding of the disruptions in those mechanisms that result in anomia.

Early aphasia research developed the concept that the errors produced by individuals with aphasia are within the same realm as the errors produced by neurotypical individuals who may have a “slip of the tongue” (Freud, 1891/1953). These errors exist along a continuum of naming errors from the normal “slip of the tongue” to severe anomia. Those with aphasia experience these anomic deficits more frequently and often to a greater degree than neurotypical individuals. This was later coined as the *continuity thesis* (Dell et al., 1997).

An Interactive Spreading Activation Theory of Word Retrieval

The continuity thesis was used in development of the interactive spreading activation theory of lexical retrieval (Dell et al., 1997). Spreading activation theory is used to explain the lexical network from mapping the conceptual representation of an object to the phonological form of the name and, ultimately, producing the word that will name the object (Dell, 1986). This

complex process involves the coordination of conceptual, pragmatic (i.e., contextual information needed for appropriate language), syntactic, and phonological information (Dell et al., 1997).

The process begins with a concept. The conceptual representation of the idea desired to be expressed (e.g., *cat*) contains semantic information. For example, semantic information activated for *cat* includes that it is a noun for something with four legs that purrs. Semantic information is then mapped to a lemma, spreading activation to non-phonological information of a word along with a frame from which the phonological form can be built. Grammatical information (e.g., number, singular and not plural, representing one cat) is also tagged onto the lemma. The lemma then enters the stage of phonological access and activation spreads to the phonological information allowing the various parts to be brought into the phonological frame (e.g., the sounds are strung together that make the word "cat", beginning with /k/).

At each level, there are nodes containing the necessary information (e.g., a semantic node contains information about the concept, a phoneme node contains information about how a specific phoneme sounds, the features of the phoneme, where the articulators must be placed to produce the phoneme, etc.). The model proposes what each level contributes to the lexical retrieval process; however, it also contains an interactive mechanism. The levels are all connected by excitatory, bidirectional connections. These connections run both top-down and bottom-up, allowing for activation to spread throughout the levels and units (e.g., from semantics to word to phoneme units and vice versa). Connections grow stronger through learning and experience. Refer to Figure 1 for illustration of this network.

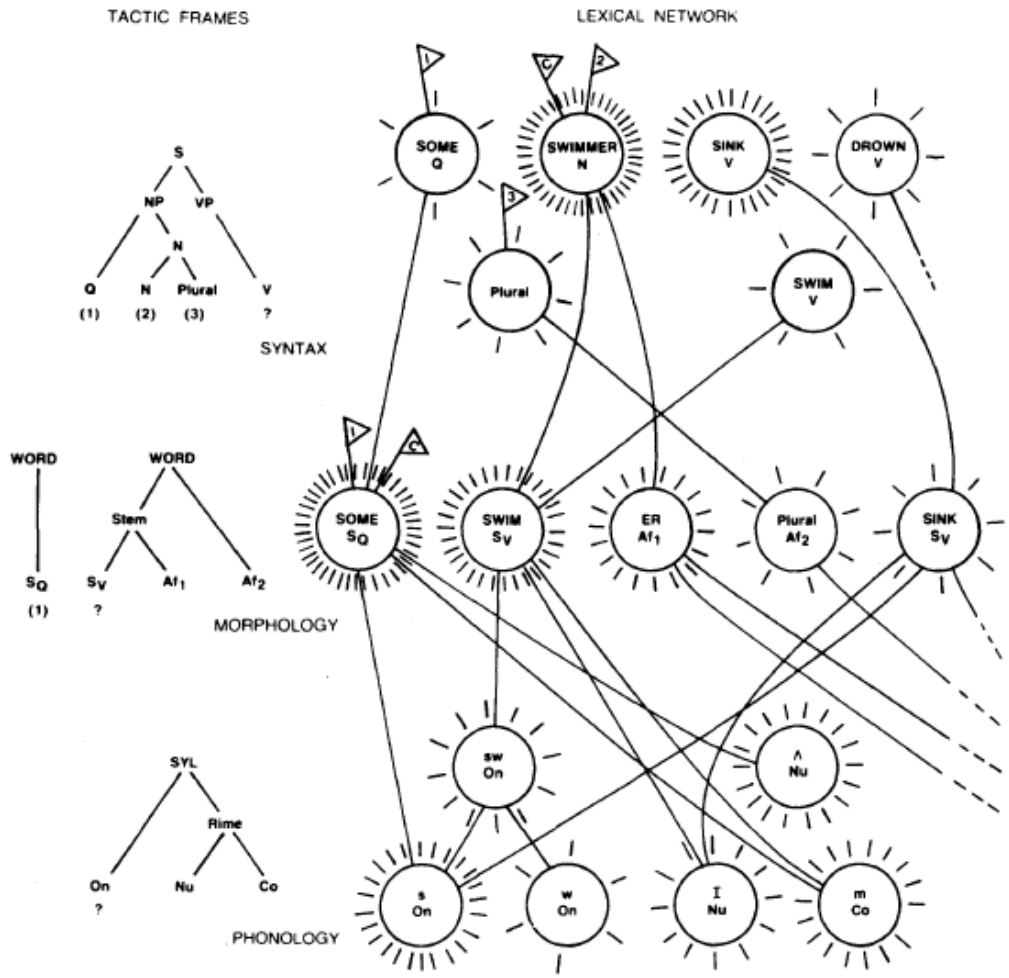

Figure 1. Conceptualization of two-level interactive model of lexical retrieval model used in the spreading activation theory (Dell, 1986)

In this interactive network, the semantic nodes send information to word nodes and then the information is sent to phonological nodes, while the phonological nodes are also sending information to the word nodes, which then send the information to the semantic nodes. Even while the semantic nodes are gaining activation, the word and phonological nodes will also begin gaining activation (Dell et al., 1997).

A computer-simulated model was used to create a system that mirrored this theory for typical lexical retrieval (Dell et al., 1997). In Figure 2, each circle represents a node of information in the simulated model. When a node is selected, it receives a jolt of activation. For example, if a picture of a cat is presented, visual processes outside this model identify the picture

and a jolt of activation is sent to the semantic nodes for features related to a "cat" (such as the gray nodes illustrated in Figure 2). The activation spreads throughout the lexical network, across all levels (via the interactive mechanism). The nodes will react based on the amount of activation received, known as the internal *weight* of the connection, and that node's *decay rate*. The weight of the connection relates to how often it is activated, therefore leading to how strong the connection becomes to other nodes. That weight is assumed to be related to learning and recent experience.

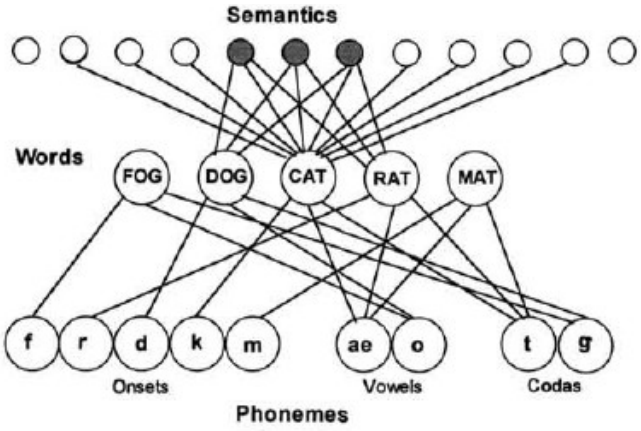

Figure 2 Illustration of lexical network for the interactive model of two-step spreading activation for lexical retrieval. Connections are excitatory and bidirectional. The nodes shaded grey represent the common semantic features of "dog", "cat" and "rat" (Dell et al., 1997).

Each node also produces *noise* relatively constantly, without any activation. In addition, when a node receives a jolt of activation, this increases the noise it produces (Dell et al., 1997). This noise produced by a node affects the related nodes, as they will then receive some activation from this input and noise. For example, suppose a picture of a cat is presented. Following the previous example, when the picture is identified and the semantic nodes for "cat" are given a jolt of activation, the activation spreads across levels. Considering the interactive process, while the lemma for "cat" is being activated, activation is sent to the phonological nodes for "cat". The

phonological nodes for “cat” reinforce the target by sending activation back to the lemma level. Simultaneously, activation is also spread to other lemmas that share those phonemes in the same syllable positions, such as “hat” and “sat”. Additionally, the phonological nodes for “dog” will also be activated because the lemma of “dog” was activated. All of these activated nodes are affected by the input and noise of their related neighbors, as well as their own decay rates (e.g., nodes that are activated less will decay faster). As the whole system interacts, sending information throughout the levels, the nodes for “cat” continuously receive activation as the most related to the concept presented. Finally, as the word “cat” continues to receive the most activation from its related neighbors, relative to its competitors, it is selected for production as the most activated word node. While the simulated model concludes at this step, it is assumed that the phonological information is then linked to slots in a phonological frame (i.e., the number of syllables, stress pattern etc..) and is then sent to be translated into articulatory codes (Dell and Schwartz, 1997).

This simulation was motivated by the interactive spreading activation model to illustrate this theory of lexical retrieval and, subsequently, lesions made in the model were used to simulate error patterns observed in those with aphasia (Dell et al. 1997). In addition, naming task trials were conducted with aphasic and non-aphasic speakers to assess their responses in relation to the simulated errors produced in the model. The spreading activation two-step lexical retrieval model simulated aphasic naming deficits to allow further understanding of alterations (i.e., lesions) in the network that account for various errors in aphasic and nonaphasic speakers. Generally, the 23 participants with aphasia presented with greater and more variable errors than the neurotypical adults.

In simulating these errors, two mechanisms within the interactive model were introduced as possible explanations of the errors (Dell et al., 1997). The first possible mechanism that led to errors when altered is *activation transmission*. This indicates that there may be a reduced ability to transmit activation between levels in the network. This relates to the previously described weight in connections across nodes. For example, there is a strong connection between the conceptual representation of "cat" and the sounds /k/, /ae/ and /t/ that form the word. The connection becomes stronger as it is more frequently activated and follows a consistent pattern for activation, leading to the retrieval of the word "cat". When the simulated model created an interruption in the mechanism for activation transmission, the jolt of activation did not follow the typical activation pattern. The noise among all nodes remained too great, resulting in increased errors. The second possibly impaired mechanism indicated is *integrity of representation* (i.e., the ability for the node itself to maintain activation). An impaired ability to maintain activation of the node as a unit may be associated with a greater decay rate, too much noise in the system, or the size of the jolt of activation may not be great enough.

These two mechanisms were surveyed in the simulated model, and the globality assumption was formed. This assumes that possible damage involves all levels in the lexical network. Essentially, the globality assumption states that all nodes (semantic, lexical and phonological) are equal parts of one system that is damaged. It was determined that different error patterns presented at different levels of severity depending on if the lesion involved activation transmission or representational integrity. The simulated model demonstrated a variety of deficit patterns, explainable by both types of activation impairment (i.e., activation transmission and representational integrity), that were consistent with the observations seen across the participants with aphasia. Ultimately, these error patterns still fell along a continuum,

supporting the continuity thesis. The errors presented by those with aphasia, when mimicked in the simulated spreading activation model of lexical retrieval, reflected extreme patterns of neurotypical speakers, thus supporting the idea that aphasia simply extends the natural variation of error patterns in speech (Dell et al., 1997).

Deficits in Aphasia: Disruption in Processing, Not a Loss of Information

The simulated model's support of the continuity thesis is extremely important as aphasia was thought to be a *loss* of language for many years (Benson, 1979). Through the work of many researchers, though, it has been extensively demonstrated that aphasia results from impaired *access* to language, not a loss of the language information itself. Among the research supporting this idea are the results of the simulated model investigating the spreading activation theory, which demonstrated the impairments observed in aphasia are most likely attributed to deficits in activation transmission or representation integrity (i.e., not impairment of the information itself, but of access to the information) (Dell et al., 1997). Additional research models supporting this idea include processing models of language (Saffran et al., 1980) and computational models of language (Martin et al., 1994).

These models and further research continued to provide supportive evidence for the idea of aphasia resulting from impaired *access* to language, as individuals with aphasia demonstrated preserved fundamental properties, rules and representations of language (Hula & McNeil, 2008). This suggests the source of the breakdown may be attributed to the cognitive mechanisms required for the online construction and integration of language. Simply put, the building blocks for language are intact, but the mechanisms putting them together may be impaired. Such examples of the building blocks of language being intact include individuals with aphasia

demonstrating metalinguistic knowledge of language (e.g., awareness that language has a structure that can be manipulated), even if they cannot produce it to the extent they could pre-morbidly (McNeil, 1988). Individuals with aphasia have also been shown to experience priming effects at all levels of the linguistic system (Martin et al., 2004; Baum, 1997; Wilshire & Saffran, 2005). Priming effects occur when exposure to a stimulus influences the response to another stimulus. This often requires linguistically-relevant information to be available in order for the stimulus to be activated or inhibited. Methods that were not linguistic-specific also demonstrated ability to produce language change, such as repetitive transcranial magnetic stimulation (rTMS) (Martin et al., 2007; Naeser et al., 2005). The successful use of rTMS for individuals with aphasia has revealed that stimulation can lead to transient increases or decreases in excitability in targeted cortices beyond the time of stimulation. For example, improved picture naming was revealed to be a possible benefit to using rTMS (Naeser et al., 2005); if the language was in fact lost, this stimulation would not be effective. Additionally, spontaneous recovery has been observed in many cases of aphasia (Lomas & Kertesz, 1978). Deficits observed in aphasia can be transient, allowing for some language representations to recover without any assistance within a couple of days following a stroke.

Variability is also a dominating aspect of aphasia, as the deficits experienced by the individuals vary depending on the day, or sometimes even the hour. A loss of language representation would not allow for variability, as loss of language would imply that it cannot return, and individuals with aphasia have demonstrated that language abilities can fluctuate; while damage to a cognitive apparatus used for language processing would account for within-task variability (Hula & McNeil 2008).

This evidence through priming, demonstrated metalinguistic knowledge, non-linguistic methods (e.g., rTMS), and the phenomenon of spontaneous recovery, reveal that language information remains intact in individuals with aphasia. Additionally, a growing body of literature has demonstrated that neurotypical individuals can present as persons with aphasia when provided an intense cognitive or linguistic load (Dick et al., 2003; Silkes, McNeil, & Drton, 2004). These neurotypical individuals have not lost their language, but an overload of the system impaired their ability to access the information. This finding continues to support the continuity thesis previously discussed. Individuals with aphasia have not lost language; rather, an inefficiency in the system of language processing results in their performance to be consistent with anomic errors on the continuum. All the examples listed above support the growing idea in the aphasia literature that the breakdown in language performance must be explained by underlying cognitive mechanisms involved in *accessing* the representations and not the loss of the information itself. This brings us to the notion that either there is an interfering factor or a lack of accessibility to the building blocks of the linguistic operation (Hula & McNeil 2008).

Working Memory and Verbal Short-Term Memory

The question remains to identify the ambiguous underlying cognitive mechanism that could contribute to language deficits in aphasia. The mechanism at play must account for the above listed phenomena (Hula & McNeil, 2008). This mechanism must change rapidly (moment to moment), not be unique to linguistic computations, must be present in neurotypical individuals as well as those with damage, and must be present under cognitively challenging conditions. While an overwhelming number of cognitive mechanisms exist, it is necessary to investigate a mechanism that fulfills the above listed criteria, and is integral to linguistic processing. Some

cognitive mechanisms recently investigated for possible implications in language processing for individuals with aphasia are working memory (WM) and verbal short-term memory (vSTM).

Short-term memory (STM) is conceptualized as a limited capacity storage mechanism retaining a representation of information that has been temporarily activated from the long-term memory store but decays over time. When an attentional mechanism is added to activated information (i.e., STM), the information is able to be rehearsed, maintained and manipulated for a brief period of time. This is known as working memory (WM). The WM system can be used for operations involving linguistic or nonlinguistic information. WM is likely constrained by time, type of data, quantity of data, difficulty of operations, and available processing resources (Hula & McNeil, 2008). See Figure 3 for an illustration based on the model developed by Nelson Cowan in 1988. We will use this model for our conceptualization of memory storage.

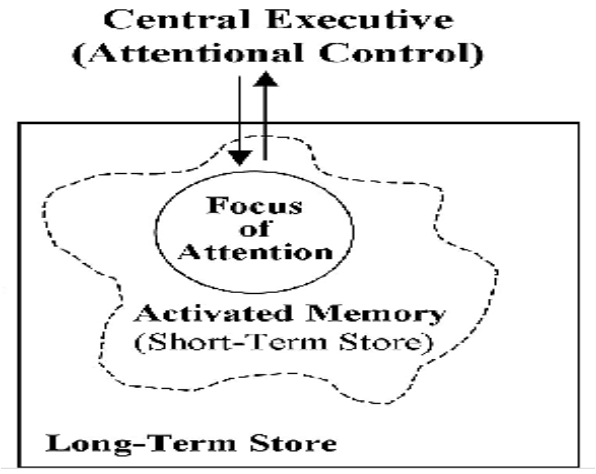

Figure 3 This illustration is the conceptualization of the long-term memory store, containing short-term memory as an activated portion of the memory store. Attention brought to STM allows for WM (Cowan 1988). Illustration: Morrison 2005

Recent research has revealed that verbal short-term memory (vSTM) and language are at least partly supported by the same temporary storage mechanism. Digit and word spans have been used to investigate vSTM in aphasia (Martin & Saffran, 1997). The performance of

individuals with aphasia on word and digit span tasks was consistent with their performance on language tasks; as the individuals received external speech and language therapy for aphasia and improved their word retrieval, their performance on word and digit spans improved as well. As previously discussed, it is possible that individuals with aphasia have trouble maintaining temporary activation of linguistic representations (Dell et al., 1997). The interactive spreading activation occurring for lexical processing is thought to be a form of STM, as the activation allows for maintenance of information over time (Martin & Gupta, 2004). An impairment in the spreading activation process would impact the ability to maintain activation of lexical representations (Martin & Gupta, 2004). The connection between digit/word span performance and language performance has indicated that the breakdowns in aphasia could suggest a deficit in maintaining activation, resulting in a breakdown in vSTM and language.

The Attentional Mechanism

The continuity between language and vSTM status has led to the hypothesis that there is a continuum between the relationship of "lexical processing impairment in aphasia and verbal span…suggesting the impairments of lexical processing and verbal STM share a common underlying deficit" (Martin & Gupta 2004, p. 214). The common mechanism underlying deficits of language and vSTM must fit the criteria listed above (Hula & McNeil, 2008), and be responsible for maintaining temporary activation of vSTM in order for language processing to be conducted properly. An aspect of WM and STM that fulfills the above listed criteria is the *attentional mechanism* (Hula & McNeil, 2008).

The attentional mechanism is a driving source, or power supply, to service cognitive processes for working memory (Hula & McNeil, 2008). It is theorized to be a single pool with

specialized reservoirs, which are limited-capacity. The resources can be flexibly allocated to where they are most needed, but the use of available resources for one task will affect their use in concurrent processing of tasks that require the same resources. The attentional mechanism has been conceived in several ways but for this purpose will be discussed as a processing resource for language within WM (Hula & McNeil, 2008). As previously mentioned, WM can be constrained by the available processing resources used to maintain the information, which is the attentional mechanism. Essentially, the ability to provide attentional resources to WM is necessary for language processing. Therefore, a lack of proper attentional resources allocated can result in aphasic deficits (Hula & McNeil, 2008).

Previous studies conducted to survey attention in aphasia through dual-task methods demonstrated that two tasks require more processing resources than a single task (Herath et al., 2001). However, there are many factors other than attentional resources that can account for these results, and most studies conducted lack the ability to provide direct assurance that a deficiency of attentional resources truly caused the observed effects. This demonstrates a deficit of information in the literature that can directly highlight the role (or impairment) of the attentional mechanism in aphasia when processing multiple stimuli. Literature to date regarding the attentional mechanism in cognitive processing will be discussed to support attention as a possible key player in the deficits observed in individuals with aphasia.

Attentional Bottleneck and Central Attention Models

The existing literature has suggested that a limited supply of attentional resources could create a processing bottleneck (Pashler, 1984). In this bottleneck model, attentional systems can sometimes process information in parallel, but in some conditions may have to process

information serially, resulting in competing tasks for processing. Processing delays are possible when there is a disruption of the timed language construction stream. Timing delays can result in breakdowns of building linguistic representations, as well as breakdowns in the encoding and decoding of linguistic forms. The attentional bottleneck model was developed based on neurotypical individuals; however, it has been theorized that an increase in the number of timing delays within this proposed model could be a potential source of resulting deficits of language processing experienced by individuals with aphasia (Hula & McNeil, 2008).

Since the attentional bottleneck model was proposed, several further models have developed, using it as a basis for theorizing a central processing system. These central processing models were created as tools for illustrating the role of attention in processing stimuli (Pashler, 1984). These models have been condensed to the Central Bottleneck model (CB) and the Single Resource model (SR), to be described together as the central attention models (Hula & McNeil, 2008). These models propose that limitations on performance of a task (e.g., language performance resulting in deficits in aphasia) arise due to serial processing of competing tasks in a limited capacity processing area with regulated resources.

The Central Bottleneck (CB) model proposes three sequential stages of processing for each task (Hula & McNeil, 2008). The precentral stage covers processing up to and including perceptual encoding of the stimulus. However, manipulation of the perceptual factors of a stimulus have revealed some effects on the processing time of the stimulus (Hula & McNeil, 2008), which would suggest perceptual discriminability could also occur in the central stage. The central stage includes response selection (i.e., the translation of an encoded stimulus into an abstract response code) and closely related processing. Finally, the postcentral stage is related to processing of response initiation and/or execution.

Precentral and postcentral stages can run in parallel with any competing task but the central stage can only process one task at a time. The bottleneck caused by serial processing in the central stage can delay or disrupt processing when two stimuli occur in too close succession. This is supported by observed effects of increasing the difficulty of response selection, supposedly affecting the central stage, resulting in increased reaction time than when compared to manipulation of the pre- and post-central processes. Dual-task experiments have also displayed the limitations of the central processing system as individuals demonstrated difficulty in completing a rapidly presented second task. This suggests the second task could not be completed until central processing of the first task was complete.

The Single Resource (SR) model is identical to the CB model, except the SR model allows for some parallel processing in the central stage. It allows some of the limited-capacity pool to be shared in a graded fashion. However, the speed at which the tasks in central processing are completed is determined by the quantity of resources applied. This results in more time needed to accomplish tasks that are sharing central stage processing resources. For example, in a dual processing task when the secondary task increased in difficulty, the reaction time to the primary task was slower (Hula & McNeil 2008). This aspect of the SR model is continually supported by experiments that have revealed participants allocating their resources for multiple simultaneous tasks, due to limited capacity parallel processing (Tombu & Jolicoeur, 2002; Hula & McNeil, 2007).

Central Attention Models and Cognitive Deficits in Aphasia

The central attention models were applied to investigate the cognitive deficits in individuals with Closed-Head Injury (CHI). The results suggested that the cognitive deficits

experienced by individuals with CHI were best characterized as a slowing of central processing (Dell'Acqua et al., 2001). Applying this research to individuals with aphasia, a dual-task experiment was conducted using picture naming and auditory identification (Hula et al., 2007). Individuals with aphasia were found to have a slower response when time between the stimuli was decreased when compared to neurotypical individuals. This supports the idea that those with aphasia have slower central processing and possibly reduced processing capacity or impaired allocation of central processing resources. Additionally, the increased reaction time was found to be substantially larger than the psychological refractory period (PRP) effect. The PRP effect is defined as a steep rise in the secondary task reaction time when the stimulus onset asynchrony (SOA) is decreased. While the individuals with aphasia demonstrated a larger reaction time than the neurotypical participants, they did not demonstrate a large PRP effect. This suggests that the additional processing occurred after the limited-capacity central stage. Using the logic of the central attention models, this would suggest that increased naming latency could be attributed to postcentral stages of processing (Hula et al., 2007). This could imply that naming latency doesn't exactly reflect the timing for central stage processing. This reinforces the need for research highlighting attention apart from actions that require various other cognitive mechanisms, such as demonstrated by the confounding aspects of reaction time. Nevertheless, the findings of this research can be integrated with the central attention models of dual-task performance and models of lexical access (Hula & McNeil, 2008).

In relation to the spreading activation model, the initial activation of the semantic, lemma (word) and phonological nodes is possibly similar to the precentral stage of processing (Dell et al., 1997). The action of selection attributed to the central stage in the CB and SR models can be at work, supplying attentional resources necessary for lexical selection as laid out in the

interactive spreading activation model. This idea was supported by several studies (Hula & McNeil, 2008; McCann et al. 2000), including a dual-task experiment revealing results consistent with lemma selection and phonological word form selection occurring in the central stage (Ferreira & Pashler, 2002). In the dual-task experiment, durations of word-production stages of lemma and phonological word-form selection were manipulated by presenting varying levels of constrained sentences and picture name frequency; additionally, lemma and phoneme selection were manipulated by presentation of conceptually and phonologically related distractors. The various manipulations (with the exception of phoneme-selection manipulations) resulted in delayed responses, indicating lemma and phonological word-form selection are subject to a central processing bottleneck, while phoneme selection is more consistent with the final (postcentral) stage. Phoneme selection, as well as response initiation and execution, have been suggested to be more consistent with occurring in the postcentral stage of the central attention processing model (Ferreira & Pashler, 2002). Figure 4 presents an illustration to compare the interactive activation model to the central attention models.

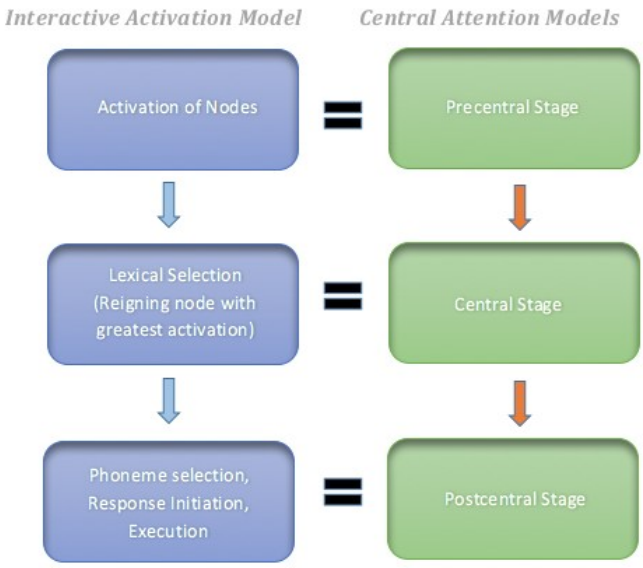

Figure 4. Comparison between the interactive activation model of lexical processing (Dell et al. 1997) and the central attention models (Hula & McNei,l 2008).

This exploration of lexical processing models mapped onto central attention processing models provides greater understanding of the role of attention in language processing. However, the results of research for central processing models (Hula & McNeil, 2008; Ferreira & Pashler, 2002) also display the role of attention as a domain-general cognitive mechanism that serves not only language processing but other non-linguistic processing tasks. Evidence suggests individuals with aphasia have impaired processing of nonlinguistic information as compared to neurotypical adults (Erickson et al., 1996; Korda & Douglas, 1997; Laures, Odell, & Coe, 2003; Laures, 2005). For example, when attention was investigated through arousal (defined as energy mobilization) in individuals with aphasia, decreased performance of processing linguistic and nonlinguistic stimuli was found for individuals with aphasia when compared to neurotypical individuals (Laures, Odell & Coe, 2003). Exploration of perception of rapid visual stimuli by individuals with aphasia also revealed decreased performance when compared to neurotypical individuals (Silkes & Rogers, 2010). The continued research revealing decreased performance in

nonlinguistic tasks for individuals with aphasia supports the idea that impairment in aphasia is of a domain-general mechanism, namely the attentional mechanism.

Impaired ability to allocate domain-general attentional resources in individuals with aphasia could possibly be responsible for the observed slowing of central processing in linguistic and nonlinguistic performance. Both resource and bottleneck models have the potential to explain variable impairments on a moment-to-moment basis, which is a prominent aspect of aphasia.

A Neural Model of Conscious Processing: The Global Neuronal Workspace

The central attention models provide stages of processing and the events believed to occur in each stage. While response selection is attributed to the central stage, there is minimal discussion regarding the actual mechanism of selection (believed to occur in the central stage) and what allows selection to occur with linguistic and nonlinguistic stimuli. In many lexical retrieval models, the actual process of selection occurring is not thoroughly discussed, as it is indicated to be outside of the proposed model.

One explanation, outlining the possible mechanism for selection, lies within a proposed neural model of conscious access developed to explain the entrance of visual stimuli to conscious awareness; this is known as the Global Neuronal Workspace (GNW) model (Dehaene et al., 2011). The GNW model was developed based on neurophysiological data and subjective reports, in an attempt to link the subjective data of observed behaviors or collected participant reports in neuro-psychological research with objective, neuroimaging data. The collection and combination

of these data was used to create a comprehensive view of neural activity that occurs during conscious access.

The conscious access discussed in the GNW model parallels the process of selection as previously discussed in the central attention models. The GNW model is based on the idea of three processing *states* the stimuli must go through to achieve conscious perception. The initial state is subliminal processing (Dehaene et al., 2006). This is a processing state where many stimuli are active, but may never grow stronger. The stimuli in this state have weak bottom-up activation that often dies out quickly, remaining insufficient for further processing, and do not reach any level of our conscious awareness. The next is a preconscious state. In this state, the stimuli have enough activation to be potentially accessible, but have not yet been accessed. The stimuli in this preconscious state are temporarily buffered in a non-conscious store because they have not received top-down attentional amplification. These stimuli may decay, losing activation before top-down attention is applied. Other stimuli may receive the top-down amplification to be brought to conscious processing when the central workspace is freed. The stimuli that receive top-down amplification are brought to the third state of processing in which surges of parieto-frontal activity (i.e., the neural action of top-down amplification) allow conscious processing to be achieved.

The GNW model operates on the assumption that conscious access (parallel to selection) is global information availability, "the selection, amplification, and global broadcasting to many distance areas, of a single piece of information" (Dehaene et al., 2011, p. 56). There are two distinct *phases* for conscious access within the GNW model, namely the preconscious phase and conscious phase (Dehaene et al., 2011). These phases of the GNW model illustrate the action the stimuli go through to reach conscious processing. This relates to the *states* of processing

previously discussed which indicated the processing the stimuli go through during the *phases* of the GNW model. For example, the stimuli in the previously discussed preconscious state could be processed, but are waiting for activation (Dehaene et al., 2006). This preconscious *state* can be mapped onto the first *phase* of the GNW model. This first phase is typically 100-300ms as the stimulus climbs up the cortical hierarchy of processors, primarily in a bottom-up manner and entirely non-consciously (Dehaene et al., 2011). Some stimuli will continue to climb and reach the subconscious state of processing. A subset of these stimuli are then amplified in a top-down manner, allowing entrance to the second phase and bringing them to the conscious state of processing. The second phase is entrance to the GNW, defined as a "distributed set of cortical neurons characterized by their ability to receive from and send back to homologous neurons in other cortical areas horizontal projections through long-range excitatory axons" (Dehaene et al., 2011, p. 56). The stimuli in the second phase are maintained by the sustained activity of a fraction of the neurons of the GNW, and the rest of the GNW neurons are inhibited. The signature of conscious processing is a late global phase characterized by sudden, late and sustained firing of the GNW neurons, increased high-frequency oscillations and long-distance phase synchrony (Dehaene et al., 2011). Essentially, when a stimulus reaches the GNW, *global ignition* occurs, allowing for what we perceive as conscious perception. The second phase can be mapped onto the conscious phase previously discussed, where selection actually occurs. The GNW accumulates information through recurrent top-down and bottom-up loops that work in a competitive manner to achieve global conscious status. The GNW is limited-capacity, only allowing a small number of stimuli to achieve the global conscious status provided by the GNW. This remains consistent with the central attention models previously presented, as they propose a

workspace that can only process a limited amount of information at once (Hula & McNeil, 2008).

The ignition of the GNW activity is often elicited by external stimuli, but the ignition itself is activated within the GNW (Dehaene et al., 2011). This ignition can also fail under specific conditions, in which case the stimuli would remain in a non-conscious state. This can occur because a stimulus did not "gather sufficient self-sustaining [reverberation]" (Dehaene 2011, p.60) and therefore died before it could reverberate to longer axons and trigger late global activation. This can be mapped onto the interactive spreading activation model's suggestion that language errors could be attributed to lack of activation maintenance, or decreased representation integrity, as the stimulus (or node) decays too quickly and cannot remain activated for an adequate period of time (Dell et al. 1997). Global ignition could also be disrupted if another incoming stimulus is simultaneously accessed (Dehaene et al., 2011). These disruptions occur during ignition because the GNW is mobilized as a whole, so if some of the GNW neurons are providing activation to one stimulus, any stimulus too close in succession cannot reach ignition.

Attentional Blink

One experimental condition known to cause ignition failure is inattention due to simultaneous tasks, namely *attentional blink* (Dehaene et al., 2011). Attentional blink occurs when two stimuli are presented in too close succession, known as a Rapid Serial Visual Presentation (RSVP) paradigm, resulting in interference in processing the second stimulus (T2) because processing is still occurring for the first stimulus (T1) (Raymond et al., 1992). It is suggested that the first stimulus elicits an attentional response to facilitate target identification, and attention is allocated episodically to that stimulus. The gate for attentional resources opens

upon detecting a stimulus and does not close until identification is complete. In contrast, if T2 is presented very shortly after T1, or at a long interval following T1, it is fully noticed and reportable. However, attentional blink is thought to occur because the participant's attention is still engaged in processing T1 (typically approximately 200-500ms), and as such the resources are not available to process T2 (Raymond et al., 1992). Manipulating the time between presentation of T1 and T2, known as stimulus onset asynchrony (SOA), is a critical aspect of the attentional blink task.

During computer-simulated trials testing the GNW model, attentional blink was observed to result in failed global ignition. A subset of GNW neurons were activated while the rest were strongly suppressed. In the absence of external input, the computer-simulated GNW neurons fired spontaneously in a top-down manner. These spontaneous ignitions "follow[ed] each other in a never-ending stream" and partially inhibited external stimuli from entering the GNW. This was suggested as a possible cause of the attentional blink, as two simultaneous stimuli cannot be adequately processed (Dehaene et al., 2011). This could be mapped onto the central attention models, suggesting attentional blink could cause response selection to fail.

Attentional blink effects have been found to be altered (e.g., a longer period of time than typical where T2 is not recognized/processed) in non-aphasic individuals with focal cerebral lesions (Rizzo, Akutsu, & Dawson, 2001), in cerebellar lesions (Jiang, Tian, & Wang, 2013), multiple sclerosis (Kavcic & Scheid, 2011), dyslexia (Lacroix et al., 2005), and attention deficit hyperactivity disorder (Amador-Campos, Aznar-Casanova, Bezerra, Torro-Alves, & Sanchez, 2015). These examples support the likelihood that individuals with aphasia, who are known to demonstrate deficits in attention skills (Hula & McNeil, 2008; Korda & Douglas, 1997; Murray, 1999, 2000; Murray, 2002; Villard & Kiran, 2016), may show impaired patterns of attentional

blink. If impaired attentional blink is present in individuals with aphasia, this would suggest an impaired attentional mechanism. The importance of the attentional mechanism in central processing and selection has been supported by the previously discussed information, detailing the possible effects that could occur due to an impaired attentional mechanism. This could account for the deficits experienced by individuals with aphasia.

Summary and Proposed Study

The spreading activation model of lexical retrieval for linguistic processing (Dell et al., 1997), central attention processing models (Hula & McNeil, 2008), and the GNW model of conscious processing (Dehaene et al., 2011) have been explored as possible tools for explaining how a deficit of the attentional mechanism could result in the impairments observed in those with aphasia. In consideration of language processing in individuals with aphasia, it has been demonstrated that the linguistic information is not lost, but the integrity of representation and activation transmission are affected, possibly by an interruption to response selection in the central attentional processing system (i.e., failed global ignition as demonstrated in the GNW model). These models can be effective tools to understanding the mechanisms of domain-general cognitive processing that can result in deficits of language processing, namely the attentional mechanism for our purposes.

The body of literature revealing a domain-general attentional mechanism potentially impaired in individuals with aphasia supports our use of an attentional-blink task to compare non-linguistic stimuli to linguistic stimuli across neurotypical participants and participants with aphasia. The attentional blink effect was observed with visual stimuli within the computer-simulated trails for the GNW model (Dehaene et al., 2011). Results of dual-task experiments

with linguistic and nonlinguistic stimuli have suggested the possible role of the attentional mechanism in explaining the deficits observed in individuals with aphasia (Hula & McNeil, 2008). However, an attentional blink task using linguistic and nonlinguistic stimuli with individuals with aphasia allows for stronger evidence, as it directly surveys the role of the attentional mechanism in processing multiple stimuli.

If the deficits experienced by individuals with aphasia are a result of a slow ability to engage attention, the individuals may not show attentional blink at the interval expected, but may show it later. However, if the deficits are affecting the individual's ability to release or inhibit attention in an appropriately timed manner, the individuals may show attentional blink at the interval expected but also later intervals. Analysis of the patterns observed by individuals with aphasia in an attentional blink task can inform a further understanding of the integrity of pre-conscious processes as they trigger the GNW, allowing for subliminal word retrieval processes leading to explicit awareness of words. The goal of this study was to evaluate the attentional mechanisms across two groups, namely people with aphasia (PWA) compared to neurotypical individuals. Linguistic and non-linguistic conditions for attentional blink tasks were used to gather information regarding the role of attention in PWA. Specifically, the research questions are:

1) How do people with aphasia (PWA) perform on a linguistic attentional blink task compared with neurotypical adults?

   Hypothesis: PWA will require a greater period of time to process T1(as evidenced by greater period of time before recognition of T2) than neurotypical adults.

2) How do PWA perform on a non-linguistic attentional blink task compared with neurotypical adults?

Hypothesis: PWA will require a greater period of time to process T1 (as evidenced by greater period of time before recognition of T2) than neurotypical adults.

Methods

Participants

People with aphasia (PWA)

Five participants with aphasia (PWA) were recruited for participation in this study, although one completed only the non-linguistic task. All PWA ranged from 17 to 134 months post-onset of aphasia due to unilateral, left hemisphere cerebral vascular accident (CVA, i.e., stroke). Participants had no history of other neurologic dysfunction, severe psychiatric conditions, drug or alcohol abuse, or speech or language disorders past the age of 8. Participants passed screening for visual acuity as assessed by tumbling E eye chart and hemi-neglect as assessed by a line bisection task. Visual correction was accepted, contingent upon the participant successfully completing visual assessments with visual correction. All were fluent speakers of

English prior to their injury, with hearing adequate to complete testing and experimental tasks. See Table 1 for demographic information.

Table 1. *PWA Demographic information*

Participant	Age	Education (years)	Months Post Onset	Severity
11A	56	16	28	Mild
12A	49	11	39	Moderate
13A	64	19	134	Mild
14A	65	18	24	Mild
15A	77	18	17	Mild

Speech-language testing and evaluation of central executive skills (e.g., attention, working memory) were conducted for each participant to determine the precise nature of his or her communication deficits and describe related cognitive skills. Severity of aphasia was documented by scores on the Language Battery portions of the Comprehensive Aphasia Test (CAT; Swinburn, Porter, & Howard, 2004). Non-linguistic reasoning skills were determined to be generally intact, as documented by a minimum score of 23 on the Raven's Coloured Progressive Matrices (RCPM ; Raven, 1976). Attention was assessed with a vigilance task (sustained attention/vigilance, and alternating attention), as well as symbol cancellation and trail making tasks (selective attention). The vigilance task involved the examiner verbally presenting a string of letters and prompting the participant to gesturally indicate (e.g., tap the table) when a specific letter was heard in the string. The participants passed the task if they completed the task with less than 2 errors. A forward and backward pointing span task was used to assess non-verbal WM/STM. Recognition of linguistic stimuli was relatively intact, as assessed by using an

alphabet array to test recognition of orthographic letters. See Tables 2 and 3 for assessment scores. Scores are not listed in the table for symbol cancellation, trail making and alphabet array tasks as all PWA achieved 100% accuracy on those assessment measures.

Table 2. *PWA Assessment Scores*

Participant	Raven's (max score = 36)	Letter String (Pass = <2 errors)	Pointing Span (forward, backward)
11A	35	Pass	5/5 4/5
12A	36	Fail	5/5 5/5
13A	32	Pass	5/5 4/5
14A	28	Pass	5/5 3/5
15A	32	Pass	5/5 4/5

Table 3. *PWA CAT Scores*

Participant	11A	12A	13A	14A	15A
Semantic Memory (10)	10	10	10	9	10
Word Fluency (animals, letter "s")	14, 12	2, 0	11, 10	19, 13	7, 7

Recognition Memory (10)	10	10	10	10	10
Comprehension of Spoken Words (30)	30	29	30	28	30
Comprehension of Written Sentences (32)	29	22	32	25	28
Comprehension of Spoken Paragraphs (4)	4	3	4	4	2
Repetition Total (74)	74	52	68	51	62
Naming Total	78	35	74	87	65
Reading Total (70)	70	14	70	67	66

Notes. Number in parenthesis indicates maximum value for each subtest or composite score. Naming total is calculated from subtest scores (max=58) plus word fluency scores.

Neurotypical (non-brain damaged) participants:

Neurotypical participants were included as a comparator control group. There were 5 neurotypical participants. These participants were age-matched to the PWA. Neurotypical participants reported no history of speech-language disorder after the age of 8, no significant history of psychiatric disorders or drug or alcohol abuse, and no history of neurological injury or disease. See Table 4 for demographic information. They were assessed using the Boston Naming Test (Kaplan, Goodglass, & Weintraub, 2001) and Raven's Coloured Progressive Matrices to rule out any possible neural impairment. All participants scored WNL on the assessment measures. See Table 4 for assessment scores. They met the same criteria for hearing and vision as the PWA.

Table 4. *Neurotypical Demographic information*

Participant	Age	Education (years)	Language(s)	Raven's (36)	Boston Naming Test (60)

1N	58	14	English	34	54
2N	63	14	English	34	60
3N	64	17	English	33	55
4N	66	13	English	34	59
5N	54	12	English	34	57

Notes. Number in parentheses indicates maximum score for subtest.

Equipment and Setting

Participants completed evaluation and experimental tasks in a quiet room. For experimental tasks, participants were seated in front of a computer at a comfortable distance from the screen. Stimuli were presented visually on a 20" CRT computer monitor connected to a personal desktop computer running Microsoft Windows 7, using E-Prime for stimulus delivery (E-Prime Professional version 2.0.10.242, 1996-2012, Psychology Software Tools, Pittsburgh, PA). The monitor refresh rate was specified for the stimulus delivery rate needed for each task. All tasks were video-recorded for the purpose of reliability coding by a trained research assistant.

Stimuli

Linguistic condition stimuli were capital letters; non-linguistic condition stimuli were simple, highly familiar shapes (e.g., square, circle, heart). T1 stimuli were presented in white, and T2 was presented as a black X (linguistic) or a black triangle (non-linguistic). All distractor items were black. All stimuli were presented on a dark grey background. Task 1 described below

presented 144 trials across linguistic and non-linguistic conditions. In each condition, there were 36 trials including a T2 stimulus, presenting 6 trials at each of six levels of SOA or presentation rates. The remaining 36 trials did not contain a T2 stimulus. Task 2 described below presented 72 trials, and Task 3 presented 144 trials. Each trial presented a stream of 18-22 letters/shapes stimuli, chosen at random and not repeated within the trial. Stimulus presentation rate was a standard 5.0 Hz (i.e., 5 stimuli per second) for the first two tasks. Task 3 included varying presentation rates from 5.0 to 12.5 Hz. Between stimulus screens, a blank grey screen was presented for the same duration as the stimulus screens. The stimulus presentation protocol was the same for all tasks; the only differences were the responses that participants were asked to make. _

Experimental procedures

Following evaluation, PWA completed the experimental tasks across 2 sessions (one for the linguistic task and the other for the non-linguistic task). The sequence of tasks was alternated by participant, so that half completed the linguistic task on Day 1 and the other half completed it on Day 2. Neurotypical control participants completed all evaluation and experimental tasks across 2 sessions. The sequence of tasks also alternated by participant in the NT group. All participants were asked to complete the following tasks, with at least 10 practice trials provided before data collection began:

Task 1: Dual-target task

This task was a classic RSVP paradigm (Raymond, Shapiro & Arnell 1992). An RSVP paradigm involves a participant identifying a target stimulus within a stream of stimuli (i.e.,

letters or shapes) presented at the same location in rapid succession, with the target item differentiated in some way (i.e., by color). The participants indicated two things: the identity of the white stimulus (T1) and the presence or absence of a black X or triangle (T2) in the stream of stimuli. Zero to five stimuli were presented between T1 and T2, with six different Stimulus Onset Asynchronies (SOA). The presentation time was 5 frames in 1 second, or 200ms per frame. See Figure 5 for an example of screens as presented to participants.

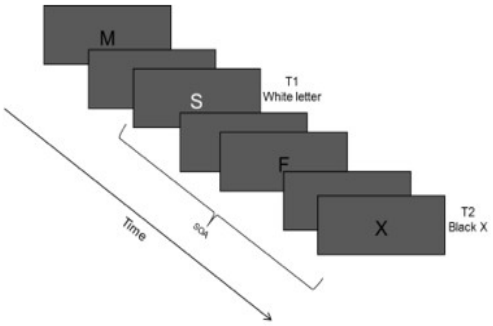

Figure 5. Sample successive screens in partial trial of linguistic RSVP task.

In the *linguistic condition*, neurotypical control participants verbally reported the identity of T1 and the presence or absence of T2. As verbal expression is often a deficit experienced by PWA, reporting for T1 identification was extended to alternate response modalities when necessary, including gestural response (e.g., choosing from among choices on a sheet of paper by pointing) or written response. The majority of the PWA responded in the verbal modality, with some using a gestural modality to supplement their verbal responses. Only one PWA used a gestural response almost exclusively throughout experimental tasks.

In the *non-linguistic condition*, response modality options were the same as linguistic condition. See Figure 6 for an example of screens as they may be presented to participants within a 1 second time frame.

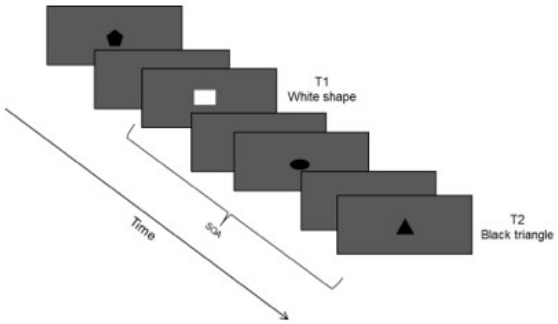

Figure 5. Sample successive screens in partial trial of non-linguistic RSVP task.

Task 2: T2 detection task

This task was designed to provide a comparator for identification of T2 in Task 1. Using the same protocol as Task 1, participants indicated the presence or absence of T2, with no need to identify T1. Response modalities were the same as described for Task 1.

Task 3: T1 detection task

This task used a variety of stimulus presentation rates to provide a measure of overall processing speed by collecting data for how quickly participants engaged T1 presentations. These data informed assessment of the relationship between overall speed of processing and attentional blink (the presence and characteristics). Participants only identified T1, with response modalities as described in Task 1. The protocol was the same as for Tasks 1 and 2, with the exception that presentation rates will vary rather than SOAs. The range of presentation rates included 5, 6.5, 8, 9.5, 11, and 12.5 Hz (i.e., 200 ms, 153 ms, 125 ms, 105 ms, 90 ms, and 80 ms per screen).

Data Analysis

Data from both linguistic and non-linguistic tasks were analyzed based on Rizzo et al. (2001). Details regarding exact analyses used for each task and condition are presented below.

The investigation of attentional blink effects was conducted only for the trials when T1 was accurately identified. This allowed the analyses to reveal the effect of attentional blink for trials when the participants had adequately engaged attention for T1. As such, before the analyses were completed, the raw data from trials when a participant did not accurately identify T1 were identified and removed. Only the 36 trials with a T2 stimulus were considered, as the remaining 36 trials without T2 were foils to fill out the task, not meaningful for analysis of attentional blink effect. See Tables 5 and 6 for information regarding trials included.

Table 5

Task 1 Trials included in Data Analyses for NT Participants

Participant	Linguistic Trials Included (36)	Non-Linguistic Trials Included (36)
1N	35	35
2N	34	34
3N	34	35
4N	33	36
5N	36	36

Table 6

Task 1 Trials included in Data Analyses for PWA

Participant	Linguistic Trials Included (36)	Non-Linguistic Trials Included (36)
11A	35	33
12A	36	34

13A	36	36
14A	N/A	33
15A	36	32

Strength of attentional blink for each participant was initially measured by plotting the percent correct detection of the presence of T2 at each SOA, including only trials in which T1 was correctly identified. This relationship was quantified by calculating d′ (Green & Swets, 1966) for correct detection responses at each SOA for each participant on both Task 1 and Task 2, followed by calculating a ratio between the two measures. This ratio provided an index of sensitivity to T2 that may have been lost when the participant needed to detect it in the presence of an interfering T1 stimulus versus in the non-interference condition; that is, this ratio estimated the magnitude of the attentional blink. A d′ ratio of 1 indicated no loss of sensitivity, meaning no attentional blink was present, while a d′ ratio of 0 indicated the strongest possible attentional blink. Based on this ratio, the *magnitude* of attentional blink was identified for each participant, defined as 1 – (the minimum d′ ratio observed for that participant). Strength and magnitude were both used in analysis to describe varying aspects of the same idea, namely a participant's susceptibility to attentional blink. For the purposes of this study, *strength* allowed for analysis of the loss of sensitivity to T2 and each SOA, speaking to the degree of susceptibility of a participant to attentional blink across all presented SOA intervals and conditions. Strength was also used as a tool to then calculate magnitude. *Magnitude* allowed for a calculation to capture the overall susceptibility of each participant to attentional blink, independent of the SOA at which the blink occurred. *Length of attentional blink for* each participant was identified by determining the highest SOA value at which each participant's ratio was <.80. This measure reflected a participant's ability to release attention from T1 so that they can engage T2.

For Task 3, the speed of T1 recognition for each participant was estimated as the fastest exposure duration at which they accurately identified a minimum of 75% of the target letters. This measure provided an indication of the speed of processing for PWA as compared with typical adults, for both linguistic and non-linguistic stimuli. These data were also analyzed using linear regression, assessing the ability of processing speed to predict the magnitude of attentional blink; this analysis helped to identify the independence of these two constructs.

Results

Rater Reliability

Inter-rater and intra-rater reliability were calculated using Cohen's Kappa Coefficient were conducted for reliability of examiner's recording of participant responses. Both intra-rater and inter-rater reliability coding were conducted for 10% of all trials for each participant. Intra-rater reliability was calculated at 0.98 agreement. The inter-rater reliability coding was conducted by a trained research assistant. The inter-rater reliability was calculated at 0.97 agreement. In both inter-rater and intra-rater reliability calculations, these scores reflect almost perfect agreement (Landis & Koch, 1977).

Linguistic Condition: NT vs PWA Data

The analysis for strength of attentional blink (AB) was calculated from the d′ values reflecting response accuracy in Tasks 1 and 2 (i.e., d′ task 1/d′ task 2). A *blink* was defined as a d′ ratio lower than 1, which occurred when percent accuracy of T2 detection decreased (i.e., when T2 was not detected, an attentional blink occurred). A blink was stronger when the d′ ratio was closer to 0 (e.g., d′ of .2 is stronger than d′ of .8). Simply put, the more often a participant did not detect T2 within a specific SOA interval, the stronger the attentional blink. Two of the five neurotypical (NT) participants demonstrated an attentional blink for at least one SOA, (see Table

7 for NT results). The overall strongest blinks (i.e., d′ closest to 0) across NT participants were observed to occur during SOA intervals 300-400ms. Three of the four PWA demonstrated an attentional blink for at least one SOA (see Table 8 for PWA results). The overall strongest blinks (i.e., d′ closest to 0) for PWA were observed to occur during SOA intervals 100-200ms.

Table 7

Summary of Results from NT Participants in Linguistic Condition

	SOA 100	SOA 200	SOA 300	SOA 400	SOA 500	SOA 600
Mean % Correct T2 detection	89.8 (SD = 9.31)	86.6 (SD = 21.74)	89.8 (SD = 9.31)	93.4 (SD = 14.76)	100 (SD = 0)	100 (SD = 0)
Mean d′ Task 1	4.718 (SD = 1.56)	4.33 (SD = 2.42)	4.02 (SD = 1.91)	4.67 (SD = 1.67)	4.504 (SD = 2.04)	5.417 (SD = 0)
Mean d′ Task 2	5.417 (SD = 0)	4.504 (SD = 2.04)	5.417 (SD = 2.04)	5.417 (SD = 0)	4.504 (SD = 2.04)	5.417 (SD = 0)
AB Strength	0.87 (SD = 0.29)	0.8 (SD = 0.45)	0.74 (SD = 0.35)	0.86 (SD = 0.31)	1 (SD = 0)	1 (SD = 0)

Notes. SD = Standard deviation. AB = Attentional blink.

Table 8

Summary of Results from PWA in Linguistic Condition

	SOA 100	SOA 200	SOA 300	SOA 400	SOA 500	SOA 600
Mean % Correct T2 detection	62.3 (SD = 28.5)	83 (SD = 13.9)	100 (SD = 0)	95.8 (SD = 8.5)	91.5 (SD = 17)	95.8 (SD = 8.5)
Mean d′ Task 1	1.35 (SD = 2.8)	2.09 (SD = 2.33)	5.417 (SD = 0)	4.10 (SD = 2.63)	4.28 (SD = 2.28)	5.417 (SD = 0)
Mean d′ Task 2	4.28 (SD = 2.28)	5.417 (SD = 0)	5.417 (SD = 2.04)	5.417 (SD = 0)	5.417 (SD = 0)	5.417 (SD = 0)
AB Strength	0.46 (SD = 0.63)	0.39 (SD = 0.43)	100 (SD = 0)	0.76 (SD = 0.48)	0.79 (SD = 0.42)	100 (SD = 0)

Notes. SD = Standard deviation. AB = Attentional blink.

Table 9

Average Attentional Blink (AB) Strength - NT and PWA

	SOA 100	SOA 200	SOA 300	SOA 400	SOA 500	SOA 600	Overall Strength
NT Linguistic	0.871	0.8	0.743	0.862	1	1	0.879
PWA Linguistic	0.461	0.386	1	0.758	0.789	1	0.732

Notes. *Strength of Attentional Blink defined as the d' ratio Task 1/Task 2. Average strength calculated for each SOA interval across all participants in each group and condition. Overall strength calculated across all SOA intervals for average of each group/condition.*

The Mann-Whitney U test compared all strength of attentional blink raw values for the participants who showed AB across all SOAs between the two groups. The results of the Mann-Whitney U test analysis were not significant (Z = -1.127, p = .329). However, these results are not very meaningful due to the small sample size and low incidence of attentional blink, particularly for the NT control participants.

The analysis for magnitude was calculated as 1 minus the minimum d′ ratio for each participant across SOA intervals. The larger values indicate a larger maximum attentional blink. As detailed above, this analysis allowed for the maximum attentional blink effect to be quantified, independent of SOA interval. The two NT participants who demonstrated an attentional blink both had similar magnitude values (i.e., .689 and .646). One PWA also had a

similar magnitude value to the NT participants (i.e., 0.646). However, the remaining two PWA who demonstrated an attentional blink had larger magnitude values as compared to the NT participants (i.e., 0.843 and 0.989). See Table 10 for magnitude data in the linguistic condition. These magnitude values reveal that, overall, the PWA demonstrated greater susceptibility to attentional blink than the NT participants.

Table 10

Magnitude of Attentional Blink - NT and PWA, Linguistic Condition.

Participant	Magnitude
1N	N/A
2N	0.689
3N	0.646
4N	N/A
5N	N/A
11A	N/A
12A	0.843
13A	0.646
15A	0.989

Notes. Magnitude of Attentional Blink defined as 1- minimum d′ ratio across all SOA intervals.

The length of attentional blink (i.e., highest SOA value during Task 1 at which a participant's d′ ratio was <0.8) for PWA participants ranged between 200-500ms (see Table 11 for results). The two NT participants that showed an attentional blink had length of attentional blink values at 300ms and 400ms (see Table 11 for results).

Table 11

Length of Attentional Blink - NT and PWA, Linguistic Condition.

Participant	Length of AB (SOA interval)
1N	N/A
2N	400

3N	300
4N	N/A
5N	N/A
11A	N/A
12A	500
13A	200
15A	400

Notes. *Length of Attentional Blink is defined as the highest SOA interval where the d' ratio <0.8. Magnitude defined as 1- minimum d' ratio across all SOAs, and Processing Speed defined as highest presentation rate at which participants achieved above 75% accuracy for T1 identification.*

The relationship between magnitude and processing speed was assessed through data from Task 3. Analysis of processing speed (i.e., the fastest exposure duration at which the participant accurately identified at least 75% of target letters) revealed that all NT participants and PWA achieved above 75% accuracy at the fastest exposure interval, 12.5 Hz. In contrast to the consistent processing speed outcomes for both groups, magnitude values were more variable in both groups. Linear regression to assess the relationship between magnitude and processing speed showed no predictable relationship between these two measures (R square = 0.23, NT; 0.16, PWA).

Non-Linguistic Condition: NT vs PWA Data

Consistent with the analysis for the linguistic conditions, the analysis for strength of attentional blink was calculated as the ratio of d′ calculated from the d′ values reflecting response accuracy in Tasks 1 and 2 (i.e., d′ task 1/d′ task 2). The majority of NT participants demonstrated attentional blink for at least one SOA in the non-linguistic condition, with the exception of one participant who did not show a blink (see Table 12 for NT results). The overall strongest blinks (i.e., d′ closest to 0) across NT participants were observed to occur primarily during SOA intervals 100-200ms. Each of the PWA demonstrated an attentional blink for at least one SOA

(see Table 13 for PWA results). The overall strongest blinks across the PWA were also observed to primarily occur during SOA intervals 100-200ms.

Table 12

Summary of Results from NT Participants in Non-Linguistic Condition

	SOA 100	SOA 200	SOA 300	SOA 400	SOA 500	SOA 600
Mean % Correct T2 detection	78.2 (SD = 30.9)	79.6 (SD = 18.6)	85.2 (SD = 16.5)	96.6 (SD = 7.6)	89.2 (SD = 15.6)	96.6 (SD = 7.6)
Mean d′ Task 1	2.67 (SD = 3.06)	2.68 (SD = 2.50)	3.04 (SD = 2.49)	4.72 (SD = 1.56)	3.75 (SD = 2.29)	4.72 (SD = 1.55)
Mean d′ Task 2	5.417 (SD = 0)	5.417 (SD = 0)	5.417 (SD = 0)	5.417 (SD = 0)	5.417 (SD = 0)	5.417 (SD = 0)
AB Strength	0.49 (SD = 0.52)	0.49 (SD = 0.46)	0.56 (SD = 0.42)	0.87 (SD = 0.29)	0.69 (SD = 0.46)	0.87 (SD = 0.28)

Notes. SD = Standard deviation. AB = Attentional blink.

Table 13

Summary of Results from PWA in Non-Linguistic Condition

	SOA 100	SOA 200	SOA 300	SOA 400	SOA 500	SOA 600
Mean % Correct T2 detection	19.0 (SD = 0.21)	54.4 (SD = 24.1)	81.8 (SD = 18.6)	86.4 (SD = 14.2)	89 (SD = 9.3)	96 (SD = 8.9)
Mean d′ Task 1	-2.77 (SD = 2.49)	0.33 (SD = 1.15)	2.82 (SD = 2.43)	3.10 (SD = 2.16)	3.31 (SD = 1.92)	4.67 (SD = 1.66)
Mean d′ Task 2	5.417 (SD = 0)	5.417 (SD = 0)	5.417 (SD = 0)	5.417 (SD = 0)	5.417 (SD = 0)	4.504 (SD = 2.04)
AB Strength	-0.51 (SD = 0.46)	0.06 (SD = 0.21)	0.52 (SD = 0.45)	0.57 (SD = 0.39)	0.61 (SD = 0.35)	1.20 (SD = 0.44)

Notes. SD = Standard deviation. AB = Attentional blink.

Table 14

Average Attentional Blink (AB) Strength - NT and PWA

	SOA 100	SOA 200	SOA 300	SOA 400	SOA 500	SOA 600	Overall Strength
NT Non-Linguistic	0.493	0.494	0.560	0.871	0.694	0.872	0.787
PWA Non-Linguistic	-0.512	0.060	0.521	0.572	0.611	N/A	0.408

Notes. *Strength of Attentional Blink defined as the d' ratio Task 1/Task 2. Average strength calculated for each SOA interval across all participants in each group and condition. Overall strength calculated across all SOA intervals for average of each group/condition.*

The Mann-Whitney U test compared all strength of attentional blink raw values for the participants who showed attentional blink across all SOAs between the two groups. The results of Mann-Whitney U test analysis were not significant (Z = -1.127, p = .329). However, these results are not very meaningful due to the small sample size and low incidence of attentional blink, particularly for the NT control participants.

The analysis for magnitude was calculated as 1 minus the minimum d′ ratio for each participant across SOA intervals. The larger values indicate a larger maximum attentional blink. As detailed above, this analysis allowed for the maximum attentional blink effect to be

quantified, independent of SOA interval. Two of the four NT participants who demonstrated an attentional blink had identical magnitude values (i.e., .843). The remaining two NT participants had lower magnitude values as compared to the two other NT participants who demonstrated an attentional blink (i.e., 0.751 and 0.646). Three of the four PWA had a similar or identical magnitude values to the NT participants (i.e., 0.689, 0.648 and 0.751). Two PWA demonstrated a very strong attentional blink effect, where they showed a blink within all presented stimuli within a specific SOA interval. This results in a magnitude value of 0, indicating the participant was highly susceptible to attentional blink effect (i.e., within a specific SOA, there was no instance of accurate T2 detection). The two PWA that showed this very strong attentional blink effect, had a larger magnitude value as compared to the NT participants and other PWA (i.e., 0). See Table 15 for magnitude data in non-linguistic condition. These magnitude values reveal that, overall, the PWA demonstrated more susceptibility to attentional blink as group, when compared to the NT participants.

Table 15

Magnitude of Attentional Blink - NT and PWA, Non-Linguistic Condition.

Participant	Magnitude
1N	0.843
2N	0.751
3N	0.843
4N	N/A
5N	0.646
11A	0
12A	0.689
13A	0.648
14A	0
15A	0.751

Notes. *Magnitude of Attentional Blink defined as 1- minimum d' ratio across all SOA intervals.*

The length of attentional blink for PWA primarily occurred in the 500ms SOA interval, with one participant demonstrating a length of 200ms SOA interval (see Table 16 for results). The NT participants showed length of attentional link values between 400-600ms (see Table 16 for results).

Table 16

Length of Attentional Blink - NT and PWA, Non-Linguistic Condition.

Participant	Length of AB (SOA interval)
1N	500
2N	500
3N	400
4N	N/A
5N	600
11A	500
12A	500
13A	500
14A	500
15A	200

Notes. *Length of Attentional Blink* is defined as the highest SOA interval where the d′ ratio <0.8. Magnitude defined as 1- minimum d′ ratio across all SOAs, and Processing Speed defined as highest presentation rate at which participants achieved above 75% accuracy for T1 identification.

The relationship between magnitude and processing speed was assessed through data from Task 3. Analysis of processing speed (i.e., the fastest exposure duration at which the participant accurately identified at least 75% of target letters) revealed that all NT participants and PWA achieved above 75% accuracy at the fastest exposure interval, 12.5 Hz. In contrast to the consistent processing speed outcomes for both groups, magnitude values were more variable in both groups. Linear regression to assess the relationship between magnitude and processing

speed showed no predictable relationship between these two measures (R square = 0.04 NT and PWA).

Discussion

This study investigated how participants with aphasia (PWA) perform on linguistic and non-linguistic attentional blink tasks as compared to neurotypical (NT) adults. The purpose of this study was to determine the feasibility of this protocol and provide preliminary data to better understand the role of the attentional mechanism for cognitive-linguistic processing in PWA.

In both conditions, the results were very similar. Overall the PWA showed more susceptibility to attentional blink (AB), compared to NT participants. No particular patterns regarding sensitivity of SOA intervals were identified across either group. This is surprising, as it was thought that the participants would show AB at a cluster of SOA intervals close in length. It is not clear why there would be no pattern. It is possible that this reflects the high inconsistency of attention in aphasia, which is supported in the literature, as it has been reported that allocation of attentional resources is inconsistent in this population (Hula & McNeil, 2008; Villard & Kiran, 2015).

The analysis of the relationship between magnitude and processing speed also revealed no noteworthy patterns or trends across both conditions. It is unclear exactly why this occurred, but it is possible that this was an issues within the methods. The methods may not have been

sensitive enough to get a complete picture of processing speed in aphasia. This is evidenced by the fact all participants achieved 75% accuracy on the fastest presentation rate (i.e., 12.5 Hz), but several participants achieved accuracy lower than 75% on slower presentation rates. This is not adequately reflected in the regression analysis results, as only the fastest presentation rate in which a participant achieved 75% accuracy was included in the analysis.

In the linguistic condition, the results of this study were consistent with Rizzo et al. (2001). The participants with cerebral lesions in the Rizzo et al. (2001) study showed AB more frequently than NT controls. This validates that the protocol successfully measured performance on a linguistic AB task for comparison across two groups.

If the observed patterns in both conditions continued to emerge in a larger study with more participants, it could suggest that PWA have more difficulty than NT participants releasing attention from T1 and engaging the T2 stimulus. These results in the linguistic condition were expected because aphasia is a disorder of language. Therefore, difficulty with a linguistically-based task would be anticipated. It was also expected in this study that the non-linguistic condition because the literature has supported the idea of attention being a domain-general mechanism. Traditionally, it would be expected that PWA would have more difficulty with the linguistic condition than the non-linguistic condition. However, the models of attention that drove this research study showed the attentional mechanism as a domain-general resource. This supported the decision to include a non-linguistic condition, with the expectation that PWA would show AB in both conditions.

It was predicted that PWA would show AB effects more frequently or more strongly in the linguistic condition as aphasia is a language-based disorder. However, the results indicated stronger AB values and more frequent AB occurrence in the non-linguistic condition across PWA

and NT participants as compared to the linguistic condition. This suggests that perhaps the nature of the non-linguistic task is generally less automatic and therefore more difficult, resulting in more incidence of AB. This idea is supported by the fact that not just the PWA had more difficulty in the non-linguistic condition, but also the NT participants showed more instances of AB and stronger AB values compared to the linguistic condition. It is possible that the less automatic or familiar stimuli (e.g., shapes as non-linguistic stimuli) in the non-linguistic condition make the task harder, leading to higher susceptibility to AB, than the linguistic condition with more automatic or familiar stimuli (i.e., orthographic letters as linguistic stimuli). The greater difficulty of the non-linguistic condition could explain why the NT participants showed more variability in length values for the non-linguistic condition than the linguistic condition. The increased difficulty could result in NT performance closer to that seen in the PWA.

Across both conditions, the common trends found can be linked to the models of attention discussed in the Introduction. The models of attention are tools used to help explain the domain-general attentional mechanism and the role it plays across both linguistic and non-linguistic processing domains. Attention is thought to be a limited resource pool, that can be flexibly allocated for various processes and plays a crucial role in the selection of information and elements across all processing networks. The attentional bottleneck model and subsequent central attention models can be used as tools for understanding this idea, as they portray attention as a limited capacity processing area that can only process a certain amount of stimuli at once. It is possible that the participants in this study engaged attentional resources for processing T1, but then required more time to release attention and engage another stimulus (e.g., T2). An influx of

information in this system can disrupt the timed construction stream which results in breakdowns in the processing system.

These models can be used to understand the incidence of larger AB effects observed in the non-linguistic condition across both groups. These larger AB effects indicate the possibility that the nature of the non-linguistic task presented may be harder overall, and as a task with less familiar stimuli the non-linguistic condition may require more resources for adequate processing. Therefore, as the attentional mechanism is allocating more resources for engaging T1, it may require more time to release those resources. This would leave even fewer resources available to be allocated to subsequently engage T2. Thus the difficulty of the non-linguistic condition would result in increased occurrence of AB and strength of AB.

In the linguistic condition, the results can be explained by mapping the models of attention to the language processing models, including Dell's interactive spreading activation model (Dell et al., 1997). Attention was discussed as a limited resource pool that is necessary for the lexical selection process. If PWA are having trouble releasing attention, as suggested by the results of the AB tasks, then it is possible they are having difficulty with their attentional resources adequately moving across the system to allow the selection process (i.e., lexical retrieval) to occur. A disruption in the flexible movement of attentional resources throughout system could lead to aphasic impairments, which Dell explains partially in terms of activation transmission, or the ability for the signal to travel efficiently and successful through the system. In broad strokes, if the PWA can't release attention appropriately, activation transmission will suffer because the system will not have the resources necessary to move and spread activation to the next required element.

Conclusion

Overall, this study set out to consider feasibility of this protocol and provide preliminary data to better understand the role of the attentional mechanism in cognitive-linguistic processing people with aphasia. This study did not provide definitive results from analyses due to small sample size and limited data points that could be included in analyses (i.e., Mann-Whitney U). In future studies, it would be advised to consider adjustment to methods for analysis of relationship between magnitude and processing, as well as possibly manipulate the presentation rate to challenge the system. Despite limitations, the collected data did reveal trends and patterns suggesting that PWA have higher susceptibility to attentional blink than NT adults for both linguistic and non-linguistic processing. This suggests that it is worthwhile to continue researching the relationship between attention and aphasia for both linguistic and non-linguistic processing.

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