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Aphasia and auditory extinction: Preliminary evidence of binding

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Abstract

Background: McNeil, Odell, and Tseng (1991), and Murray and colleagues (Murray, 2000; Murray, Holland, & Beeson, 1997a, 1997b) have suggested that variability of performance in patients with aphasia may be due to nonlinguistic cognitive variables, such as attention (i.e., resources, capacity, effort), which affect language comprehension and production. Given the research that has supported the relationship between aphasia and attention deficits, it is important to determine what effect this breakdown in attention may have on cognitive processes for individuals with aphasia.

Aims: This study aims to determine if auditory extinction is present in individuals with aphasia, and if so, if this is due to a breakdown in binding. If extinction is found for individuals with aphasia, it would further support the notion that auditory attention difficulties are present among individuals with aphasia, since visual and auditory research has attributed extinction to a breakdown in attention (Baylis, Driver, & Rafal, 1993; Deouell, Bentin, & Soroker, 2000; Deouell & Soroker, 2000). If binding is found to be deficient, the fact that individuals with both left and right hemisphere lesions demonstrate this phenomenon would lead to a number of implications regarding the relationship of attention and aphasia.

Methods & Procedures: Auditory extinction, in which one stimulus is not perceived during double simultaneous stimulation (DSS) presentation, was examined in six individuals with aphasia (aged 42–74 years) and six age-matched healthy adults. Two different experiments were conducted in which the auditory stimuli, consisting of male and female voices speaking the letters “T” or “O”, were systematically varied to investigate whether binding of identification to location contributes to extinction.

Outcomes & Results: Participants with aphasia made more omission errors (extinction) than the control group, and extinction was significantly greater for binding versus nonbinding conditions, suggesting that binding may play a role in extinction for individuals with aphasia.

Conclusions: These data provide preliminary results that auditory extinction exists in individuals with aphasia and may be due to deficits in binding together identification and localisation information. Research on this phenomenon and how it influences language would be a worthwhile endeavour for future studies. Moreover, little is known about assessment of auditory attention in patients with aphasia. Further research in this area can lead to advancements in theoretical and functional assessment for individuals with aphasia who have auditory attention and/or binding deficits and require speech-language pathology intervention.

The study of aphasia has evolved and expanded to include research related to cognitive processes. Studies have focused particularly on attention and short-term memory (Erickson,

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Goldinger, & LaPointe, 1996; LaPointe & Erickson, 1991; McNeil & Kimelman, 1986; McNeil, Odell, & Tseng, 1991). For example, McNeil et al. (1991) suggested that variability of performance in patients with aphasia may be due to non-linguistic cognitive variables, such as attention (i.e., resources, capacity, effort), and suggested a resource allocation framework to explain performance decrements on linguistic tasks. Furthermore, Murray (2000) argued that variable attentional (selective or focused) demands influence the language of individuals with aphasia. She suggested that these patients' focused attention deficits decrease the accuracy and efficiency of both comprehension and production skills. Other studies have shown that individuals with aphasia demonstrate divided attention deficits and these in turn affect performance on comprehension (Murray, Holland, & Beeson, 1997a, 1997b; Tseng, McNeil, & Milenkovic, 1993) and production (Murray, 2000; Murray, Holland, & Beeson, 1998). Given the research that has supported the relationship between aphasia and attention deficits, it is important to determine what effect this breakdown in attention may have on cognitive processes for individuals with aphasia.

One cognitive process that has been a focus of recent research is the role of “binding” information in auditory and visual perception (Baylis, Driver, & Rafal, 1993; Baylis, Gore, Rodriguez, & Shisler, 2001; Shisler, Gore, & Baylis, 2004). Binding refers to the integration of sensory information (identification and location) into a whole, thus resulting in the perception of an object or event. Binding arguably occurs when information is integrated from the dorsal and ventral pathways in audition and vision (Ash-bridge, Walsh, & Cowey, 1997; Corbetta, Shulman, Miezin, & Petersen, 1995; Petersen, Corbetta, Miezin, & Shulman, 1994). The idea that attention is necessary for the binding together of different aspects of visual stimuli was proposed by Treisman and Gelade (1980), and the importance of attention in solving the binding problem has been emphasised by many researchers (e.g., Reynolds & Desimone, 1999). Recent research suggests that individuals with right hemisphere (RH) brain damage have difficulty in binding identification and localisation information in vision (Baylis et al., 2001) and audition (Shisler et al., 2004) due to deficits in attention.

One phenomenon suggested as resulting from a breakdown in attention and therefore binding is called “extinction”. Extinction is typically defined as a failure to respond to a contralesional target during simultaneous presentations of stimuli to both the left and right hemifields, known as double simultaneous stimulation or DSS (Marzi et al., 1996), and is not considered to be due to a sensory deficit (Baylis et al., 2001; Shisler et al., 2004). For example, during DSS, individuals with RH lesions with extinction have shown decreased contralesional performance when asked to respond to two simultaneous targets (left and right) (Baylis et al., 2001; Shisler et al., 2004); however, participants are typically able to correctly identify a single stimulus presented in the contralesional hemifield (single left presentation). Due to the fact that individuals are able to identify a single stimulus on the contralesional side, many researchers have argued that extinction is caused by a deficit in attention and not due to sensory difficulties *per se* (Baylis et al., 1993; Deouell & Soroker, 2000; Volpe, LeDoux, & Gazzaniga, 1979). In other words, information properly enters the sensory system, although the patients may not be aware of its presence (Volpe et al., 1979).

Baylis et al. (1993) and Kanwisher (1991) argued that attention is necessary for binding information together. Treisman and Schmidt (1982) stated that if an object is to be identified then attention must be directed to that object. Baylis et al. (1993) argued that in patients with unilateral RH lesions, specific features may not be combined in the contralateral field, resulting in extinction. Therefore, when deficits in attention are present, as in individuals with RH damage, patients may not be aware of contralesional stimuli because identification and location information was not bound together properly (Baylis et al., 1993, 2001; Shisler et al., 2004). Further support related to extinction came from Deouell and Soroker (2000). They suggest that auditory extinction in RH patients may be due to a disruption of spatial information. In one

study by Deouell and Soroker (2000), patients with right hemisphere lesions were presented with auditory consonant–vowel pairs to their left, their right, or both. On approximately 77% of bilateral trials, patients extinguished the contralesional stimulus. This represents further support for auditory extinction when the attention system is disrupted, as it is in the right hemisphere damaged population.

Additionally, Shisler et al. (2004) found that when patients with RH damage were presented with auditory stimuli in a double simultaneous stimulation (DSS) task that required binding of identification and localisation, participants demonstrated significant extinction for information presented to the left; however, in a similar task that did not require binding, omissions decreased for DSS items presented contralesionally. Note that during this task when single items were presented to the left or right, patients made very few errors. The findings suggest that tasks necessitating binding require attention and if attention is disrupted (such as in RH lesions), deficits in binding may be observed. These results were consistent with previous visual attention experiments (Baylis et al., 1993, 2001) and suggest that auditory extinction may also be due to a failure to bind identity and location information.

As mentioned previously, auditory extinction has been found for RH lesioned patients (Deouell et al., 2000; Deouell & Soroker, 2000; Shisler et al., 2004), and one study found auditory extinction for individuals with aphasia (Castro-Caldas, Guerreiro, & Confraria, 1984). Castro-Caldas et al.'s (1984) results must be interpreted with caution, however, due to the linguistic stimuli used and because headphones were used to present stimuli to participants. Auditory information is less likely to be fused into a single occurrence if presented in free field (Soroker, Calamaro, Glicksohn, & Myslobodsky, 1997), whereas if presented via headphones, simultaneous sounds tend to be fused (Pantev, Elbert, Ross, Eulitz, & Terhardt, 1996).

Extinction in RH patients has been attributed to a deficit in the binding system resulting from attention deficits (Baylis et al., 2001; Shisler et al., 2004). Given that researchers have found that aphasia may also be related to attention deficits (McNeil et al., 1991; Murray, 2000; Murray et al., 1998), it could be predicted that individuals with aphasia may also show a deficit in binding, similar to RH patients (Shisler et al., 2004). There is evidence to suggest that left hemisphere stroke patients demonstrate extinction similar to that seen in right hemisphere patients (Bouma & Ansink, 1988; Castro-Caldas et al., 1984; Poncet, Habib, & Robillard, 1987), although the nature of these deficits is not clear. One possibility explored in this set of studies is that a deficit in binding is related to the exhibition of extinction in this population.

The theory of “binding” would explain that extinction is due in part to a failure to bind featural and location information together, and would predict that without such a requirement for binding of auditory identification and localisation pathway information, extinction may be greatly reduced. This view proposes that the level of extinction is partially dependent on the requirement to bind together information from “what” and “where” auditory pathways, and not due to any simple perceptual competition between stimuli. Therefore, it would be expected that if binding was not required when presenting the same stimuli, extinction would be reduced. In other words, a reduction in extinction may indicate that extinction occurs as a result of more than one factor. If individuals with aphasia demonstrate extinction in the auditory modality for binding tasks, but do not demonstrate as much extinction for nonbinding tasks, it could be argued that individuals with aphasia have deficiencies in the binding system. While this may not be the only theoretical explanation for extinction, this expectation of reduced (not abolished) extinction is consistent with past research in other modalities and populations that has found a decrease in extinction when binding requirements are removed (Baylis et al., 2001; Shisler et al., 2004).

If extinction is found in the current study, it would further support the notion that auditory attention difficulties are present among individuals with aphasia, since visual and auditory research has attributed extinction to a breakdown in attention (Baylis et al., 1993; Deouell et al., 2000; Deouell & Soroker, 2000). If binding is found to be deficient, the fact that individuals with both left and right hemisphere lesions demonstrate this phenomenon would lead to a number of implications regarding the relationship of attention and aphasia. For example, auditory attention for individuals with aphasia might need to be assessed more routinely to determine if these deficits are present. If binding plays a role in this, it could influence assessment procedures of these individuals. Therefore, knowledge about the nature of auditory attention deficits and possibly the ability to bind together information properly can help to advance knowledge in the area of aphasia and attention. If individuals with aphasia have a deficit in binding information, future research on this phenomenon and how it influences language would be a worthwhile endeavour. Moreover, little is known about assessment of auditory attention in patients with aphasia. Further research in this area can lead to advancements in theoretical and functional assessment for individuals with aphasia who have auditory attention and/or binding deficits and require intervention.

The purpose of this study was to determine if auditory extinction deficits exist for patients with aphasia, and if a deficit in binding can account for such performance. The first main prediction was that there would be significant extinction for individuals with aphasia on tasks that required identification and localisation, and that the control group would demonstrate significantly less extinction. The second prediction was that requiring counting only (a nonbinding task) would decrease the amount of extinction to the level of the control group, suggesting that it is binding of information on DSS (not DSS itself), that is impaired in aphasia.

METHOD

Participants

A total of 12 individuals participated in two experiments: a group of individuals with aphasia and an age-matched control group. The group with aphasia included six monolingual, English-speaking individuals with aphasia, selected from Northeast Georgia Medical Center and the Athens Stroke Club. Participant selection criteria were met, which included (a) identification of aphasia by a certified SLP; (b) comprehension of complex commands (for example, “point to picture of what you heard and the side you heard it on”); (c) hearing of single stimuli (“T” or “O”) at 80 dB SPL (C weighted) bilaterally (measured at the sound source) presented from a computer in free field; and (d) identification of mild to moderate aphasia, defined as a severity of 3 or 4 on the *Boston Diagnostic Aphasia Exam* Short Form (Goodglass, Kaplan, & Barresi, 2000). The type and severity of aphasia, age, and aetiology details from medical records of the hospital are provided in Table 1. Percentiles from subtests on the *BDAE* Short Form are listed in Table 2. Note that participants were recruited based on presence of aphasia, rather than any anatomical screen or specific lesion site, since anatomical correlations with the behavioural information is beyond the scope of the current study.

In addition to the participants with aphasia, six healthy older adults were age-matched within a range of 5 years for a control group (age range 43–73). For inclusion in the study, participants had to be able to hear single stimuli (male or female voices saying “O” or “T”) with 100% accuracy on both sides during the screening. Inclusion criteria included negative self-report of neurological disorders such as stroke, transient ischaemic attacks, Parkinson's disease, Alzheimer's disease, psychological illnesses, learning disability, seizures, and attention deficit disorders.

Apparatus

Auditory stimuli for the experiment were produced by digitising the speech of one male and one female speaker, each speaker producing the letters “O” and “T”, using Sound Blaster (Creative Computing Inc.) 24 bit digitalisation. Stimuli were presented via speakers on either side of the patient, using Super Lab Pro software for Windows version 2.01. Participants were seated approximately 1 metre from the speakers, which were placed at a 120° angle, i.e., 60° to the left and right (see Figure 1). The speakers were approximately 0.7 metres from the floor.

Screening

Participants with aphasia were identified at the Northeast Georgia Medical Center and the Athens Stroke Club, and were asked if they would like to participate in the following studies. If patients were interested, they completed a consent form and a screening procedure (to determine ability to follow complex commands), which utilised a subset of the stimuli from the experiment (4 single stimulus presentations and 16 DSS presentations = 20 trials). These were presented through a Nomad II Digital Sound Recorder (Creative Labs) and played through headphones. Participants responded verbally or by pointing, or a combination of both. Passing the screen included understanding the directions to point to a graphic representation of the letter presented and the side it was presented on for single as well as DSS trials. The BDAE short form was then administered to determine type and severity of aphasia. Six age-matched individuals who had a negative history of neurological disorders completed a consent form, a questionnaire, and the above-mentioned screening procedure prior to beginning the experiments.

Procedure

Testing took place in one of three places in order to encourage recruitment: an office in Northeast Georgia Medical Center, Adult Neurogenics Laboratory at the University of Georgia, or at the participant's home. Participants completed three auditory extinction tasks (two experiments) on the computer. For this portion, participants were seated in front of the computer with the speakers equidistant. They were instructed to keep their head forward while the stimuli were being presented. The experimenter monitored head movement throughout the experiment and corrected placement as necessary. Each trial began when the experimenter pressed the “enter” key after a fixation cross had appeared in the middle of the computer screen. The experimenter waited until the participant was ready and then pressed the space bar, which elicited presentation of a voice or blank trial. At this point the participant made his or her report, either by verbally responding or by pointing to a visual representation of the two 2-inch high letters typed on a single piece of paper. Due to participants' limited verbal output, response order was not limited, although directions were presented as a correct answer form was “T on the left”. If pointing was used, participants responded with their non-affected hand. The response was entered into the computer for subsequent scoring by the experimenter. Participants were encouraged to rest as often as they wished, although they rarely did so except at the end of blocks of trials.

Stimuli and design

Stimuli for both experiments were identical. The stimuli consisted of one male and one female voice saying letter names for 300 ms each. These stimuli were combined to create three types of trials used for both experiments (42 presentations per experiment) presented in pseudorandom order:

- Catch trial (6 trials): No stimulus was presented.
- Single trial (12 trials): A male or female voice saying “O” or “T” was presented (a) on the left only or (b) on the right only.

- DSS trial (24 trials): Two stimuli were presented simultaneously to the left and right. These could be (a) the same letter in the same voice, (b) different letters but in the same voice, (c) same letter but in different voices, or (d) different letters in different voices.

Intensity was measured using a Radio Shack Sound Loudness Meter at the sound source. The level of intensity was determined by the participants' responses. For example, if an individual could not detect a single presentation at the starting point of testing (65 dB SPL), the sound was initially increased until single presentations were correctly identified on both sides. If participants could not identify single sounds bilaterally above 80 dB SPL, they were not included in the study ($N = 4$). The sound intensity was always equivalent in both the left and right speakers. This dB level was maintained throughout both experiments. For all participants, the range of intensity was between 68 and 75 dB SPL (C weighted), as measured at the speakers. This results in a maximum presentation of 65 dB SPL at the level of the participants' ears.

Experiment 1

In order to make this experiment analogous to visual experiments conducted in the literature (Baylis et al., 1993; Baylis et al. 2001), and to allow analyses of stimuli relevance, two tasks were completed with different identification requirements. The two tasks in Experiment 1 were: letter identification (task 1) and sex identification (task 2). In both tasks, participants were asked to report the location (right or left) and identify each stimulus or report if they heard nothing. Therefore, when the task was to identify the letter, (e.g., "T" was presented on the left and "O" was presented on the right), the patient was to report "left T/right O". Participants were allowed to respond verbally or point to the location of the letter and a visual representation of "T" or "O". This allowed individuals with expressive language deficits to be included in the study. When the task was to identify sex, if a male voice was presented on the left and nothing was presented on the right, the patient reported "left male/ right nothing". Participants were allowed to respond verbally or point to the location of the voice and a visual representation of "male" or "female". One individual in the experimental group used pointing as her only response mode. All other individuals either responded verbally (Aphasia = 2; Control = 5) or a combination of verbal and pointing (Aphasia = 3; Control = 1) for this experiment. All responses were logged and were categorised in subsequent data analysis as a correct response, an omission error, a mislocalisation error, or a nonomission error. Omission errors were considered of primary importance due to the definition of extinction, although other errors were calculated (i.e., mislocalisations).

Experiment 2

This experiment employed the same stimulus conditions and number of trials, although the task of the patients was simply to count the stimuli (one or two stimuli possible), therefore only the task instructions differed for this experiment. For this task an omission error was logged if the participant counted "1" if two stimuli were presented or "0" if one stimulus was presented. No response additions were found (saying "2" if only one stimulus was presented).

RESULTS

Experiment 1

As described above, the main prediction for Experiment 1 was that there would be a greater proportion of omission errors for DSS trials than for single stimulus trials (regardless of the task), characteristic of extinction. It was also predicted that individuals with aphasia would have a greater proportion of omission errors (i.e., extinction) than the control group.

Extinction on DSS. For both the letter and the sex tasks, the number of omission errors in reporting stimuli increased significantly under conditions of DSS for the group of aphasia participants. The mean error proportions for each group for single and DSS presentations are shown in Figure 2. A within-subject repeated measures 2 (Task) $\times$ 2 (Group) $\times$ 2 (Trial) Analysis of Variance (ANOVA) was completed to determine if the proportion of omission errors for the single presentation condition (single left and right together) differed from the number of omission errors in the DSS condition. The analysis indicated that stimuli on DSS trials were missed significantly more often than single trials, $F(1,5) = 80.509$, $MSE = 336.8$, $p < .0001$, $\eta_p^2 = .942$. This difference performance for single trials and DSS trials reveals that there is clear auditory extinction for both the letter and sex tasks. An additional main effect for Group was found, $F(1,5) = 12.49$, $MSE = 552.3$, $p = .017$, $\eta_p^2 = .714$, and an interaction for Group $\times$ Trial was significant such that the aphasia group had a greater proportion than the control group of omission errors on DSS trials, $F(1,5) = 12.35$, $MSE = 526.9$, $p = .017$, $\eta_p^2 = .712$. No other significant effects were found, with observed power less than .06 for all. A planned comparison of the tasks was completed, which revealed that participants' performance did not differ significantly when identifying letter or sex ($p = .127$, $\eta_p^2 = .025$).

Further analyses were completed to determine if omissions on the left and the right for DSS trials were significantly different. It was predicted that participants with aphasia would have a greater proportion of omission errors for DSS trials contralateral to the lesion (i.e., right sided), similar to that seen in right hemisphere patients (Poncet et al., 1987). A 2 (Task) $\times$ 2 (Group) $\times$ 2 (Side) within-subjects repeated measures ANOVA was performed, with a significant main effect for right side errors found, $F(1,5) = 10.78$, $MSE = 42.87$, $p = .022$, $\eta_p^2 = .683$. Also, a main effect was found for Group, $F(1,5) = 11.89$, $MSE = 29.13$, $p = .018$, $\eta_p^2 = .704$. No other significant effects were noted ($p > .1$, $\eta_p^2 = .387$) with observed power less than .304. This suggests that there was a reduced amount of attention to the contralesional auditory items for individuals with aphasia (see Figure 3), and this pattern was not significant for the control group.

Closer inspection of the error types made by individual participants revealed that four of the six participants with aphasia omitted stimuli on the right and mislocalised the omitted stimulus to the left side (see Table 3). In other words, if a "T" was presented to the left and an "O" to the right and the participant responds "O on the left", it considered that the "omitted" stimulus (i.e., the one presented on the side the participant does not respond to) was mislocalised (reported on the opposite side from presentation). Participant MS mislocalised the omitted stimulus in 30% of the DSS trials presented, while WR mislocalised only 4.1% of the DSS trials, HF mislocalised 16%, and WG 8%. Interestingly, two of the individuals in the control group accurately reported the identity of both stimuli (did not omit), but mislocalised 4% of the DSS trials for the sex identification task. Note that for both groups a majority of errors were made on the sex task of Experiment 1. This suggests that information regarding the stimuli was processed although the response was only partially accurate as suggested in other research (Deouell & Soroker, 2000; Shisler et al., 2004).

Effect of stimulus sameness. The omission error data from the present study are sorted according to sameness on the task-relevant dimension in Figure 4, and sorted according to sameness on the irrelevant dimension in Figure 5. For example, if participants were asked to name the letter, then letter was the relevant dimension and sex was the irrelevant dimension. This analysis was completed to determine if trials that contained the same information dichotically were (1) missed more often than trials that were different, and/or (2) missed more often if the information was relevant to the task at hand (i.e., same sex trial when asked to name the sex). The data shown in these graphs suggest that if stimuli were the same dichotically, auditory extinction increased. That is, when the two fields contained stimuli that were the same, the items were more likely to be omitted than when the two stimuli differed.

In order to test whether there was a significant effect of stimulus sameness, a within-subject repeated measures $2 \text{ (Task)} \times 2 \text{ (Group)} \times 2 \text{ (Relevance)} \times 2 \text{ (Sameness)}$ ANOVA was completed. A significant increase in omission errors was found when two stimuli were the same versus those that differed, $F(1, 5) = 15.72$, $MSE = 1312.86$, $p = .011$, $\eta_p^2 = .759$. Also, a significant effect was found for the increase in the amount of omission errors for the aphasia participants versus the control group, $F(1, 5) = 11.92$, $MSE = 4356.98$, $p = .018$, $\eta_p^2 = .704$. Significant interactions were observed for Task $\times$ Relevance, $F(1, 5) = 9.88$, $MSE = 38.37$, $p = .026$, $\eta_p^2 = .664$, which suggests that omission errors were higher for the task-relevant trials for the sex task. Also, Task $\times$ Group $\times$ Relevance interaction was significant, $F(1, 5) = 16.22$, $MSE = 8.68$, $p = .010$, $\eta_p^2 = .764$, such that this difference on task-relevant trials for the sex task was greater in the aphasia group. Finally, the Task $\times$ Relevance $\times$ Sameness comparison demonstrated a significant effect, $F(1, 5) = 13.68$, $MSE = 126.83$, $p = .014$, $\eta_p^2 = .732$, with more omissions for task-relevant trials for the same stimuli on the sex task. That is, when participants in the aphasia group were asked to name the sex of the voices presented, omission errors increased for same sex trials (relevant) significantly more than for same letter trials (irrelevant). Perhaps it was simply more difficult to name the sex rather than the letter. Although the underlying cause of the significant difference found between the letter task and the sex task for task-relevant trials is unclear, the trend for both the letter and sex tasks is similar (see Figures 5 and 6).

Experiment 2

Experiment 2 did not require binding of identity and location information and therefore was considered a nonbinding task. Thus, if participants with aphasia demonstrated a significant increase in performance for a task that did not require binding, it could be argued that binding was disrupted in patients with aphasia on DSS paradigms (i.e., if proportion of omission errors are significantly lower for the count-only task); however, if participants with aphasia exhibited the same level of performance on this task, this would suggest that alternative explanations for participants' performance are necessary. It was also hypothesised that the control participants would exhibit similar performance on Experiment 2 in comparison to Experiment 1 due to an intact binding system. Additionally, it was expected that the control group would again demonstrate superior performance in contrast to the participants with aphasia.

This experiment employed the same stimuli as Experiment 1, and only required that participants count the number of stimuli as opposed to identifying and localising the stimuli. The expectation was that by not asking individuals to combine identity and location information, the requirement of binding would be removed. If indeed extinction is due in part to a failure to bind these two types of information, then it would be predicted that fewer contralesional stimuli would be missed in DSS trials for this task. Again, participants were allowed to respond verbally or point to a visual representation of "1" or "2". Only one individual pointed solely for a mode of response. All other individuals responded verbally (Aphasia = 5; Control = 6) for this experiment.

Extinction on DSS. The mean proportion of errors of omission in single and DSS trials are also given in Figure 2. A within-subject repeated measures $2 \text{ (Trial)} \times 2 \text{ (Group)}$ ANOVA revealed that there was a significant difference between omissions on single trials and DSS trials, $F(1, 5) = 33.02$, $MSE = 255.06$, $p = .002$, $\eta_p^2 = .869$, with no other significant main effects or interactions ($p > .10$, $\eta_p^2 = .437$). Observed power was .359 for non-significant effects. These comparisons suggest that errors of omission, and therefore significant extinction, are demonstrated when participants are simply required to count stimuli; however, it is not clear if the amount of omission errors are significantly different from those in Experiment 1.

Therefore, in order to determine if these results differed from those observed in Experiment 1, a within-subject repeated measures ANOVA was conducted between the contralesional error

rates for DSS trials in Experiment 1 (task 1 and 2) versus Experiment 2 ($3 \times 2 \times 2$). Note that the stimuli in these two experiments were the same; what differed was whether the participants had to identify and localise stimuli, or simply count. Main effects were found for Trial, $F(1,5) = 64.49$, $MSE = 544.77$, $p < .0001$, $\eta_p^2 = .928$, and Group, $F(1,5) = 9.63$, $MSE = 758.81$, $p = .027$, $\eta_p^2 = .658$, with a planned comparison revealing a significant difference between the two tasks in Experiment 1 versus Experiment 2, $F(1,5) = 12.23$, $MSE = 173.64$, $p = .017$, $\eta_p^2 = .710$. This analysis demonstrated that participants made significantly more DSS errors on the binding task than on the counting task, and there was a significant difference in performance for the aphasia versus the control group. A significant effect for Group $\times$ Trial was also found, $F(1,5) = 9.71$, $MSE = 718.8$, $p = .026$, $\eta_p^2 = .660$, such that significantly more omission errors on DSS trials were observed for individuals with aphasia. An effect approaching significance was also noted for the interaction of Experiment $\times$ Group ($p = .063$, $\eta_p^2 = .424$) with observed power at .539. Planned comparisons combining the two tasks in Experiment 1 versus Experiment 2 revealed significant interactions for Experiment $\times$ Group, $F(1,5) = 14.92$, $MSE = 52.89$, $p = .012$, $\eta_p^2 = .749$, Experiment $\times$ Trial, $F(1,5) = 8.61$, $MSE = 70.63$, $p = .032$, $\eta_p^2 = .633$, and Experiment $\times$ Group $\times$ Trial, $F(1,5) = 9.32$, $MSE = 299.13$, $p = .028$, $\eta_p^2 = .651$. These results demonstrate a significantly greater proportion of omission errors across trials for aphasia participants on the binding tasks (Experiment 1). The data for these comparisons are plotted in Figure 6. Additionally, this suggests that the results for the participants with aphasia were not due to practice effects. If the decrease in omissions had been due to practice effects, both groups would have demonstrated a difference from Experiment 1 to Experiment 2, which was not observed. Control group participants did not demonstrate decreased omission errors for Experiment 2.

GENERAL DISCUSSION

As predicted, significantly more auditory extinction was found in Experiment 1 for the individuals with aphasia versus the control group. This was found when individuals were asked to identify and localise stimuli by reporting which stimulus was present in each sound hemifield. All six individuals with aphasia exhibited extinction; the strength of this finding is notable given the debate as to the frequency and robustness of auditory extinction for individuals with aphasia (Castro-Caldas et al., 1984). One possible reason for reported difficulty in demonstrating auditory extinction could be the use of headphones. Our use of free field loudspeaker presentation for the auditory stimuli may lead to a greater phenomenal sense of auditory objects in space (Pantev et al., 1996).

Also, participants with aphasia demonstrated significant extinction for tasks that required binding, as well as demonstrating significantly decreased extinction on a non-binding task (Experiment 2). This combined result suggests that individuals with aphasia demonstrate similar auditory extinction deficits to right hemisphere stroke patients (Shisler et al., 2004). Furthermore, performance on task-relevant and task-irrelevant trials was similar to that found by Shisler et al. for RH patients where there was a significant effect for sameness on the relevant dimension (sex of the speaker). The pattern of performance observed in the participants with aphasia in the current study is similar to that observed in participants with RH in previous studies. For individuals with RH lesions, Baylis et al. (2001) and Shisler et al. (2004) suggested that these deficits are due to a deficit in the binding system. The results from this research tentatively support the theory that individuals with aphasia demonstrate a deficit in binding, although replication is needed for verification.

On the nonbinding task, performance for individuals with aphasia did not differ significantly from the control group. The aphasia group did demonstrate decreased omission errors on the nonbinding task in comparison to the binding task. This supports the theory of decreased binding in individuals with aphasia. Also in Experiment 2 (the nonbinding task), significant

extinction was found for both groups. Therefore, the finding of extinction in Experiment 2 in both the aphasia and the control groups suggests that the significant auditory extinction may be due to auditory attention deficits in healthy ageing. The control group's performance did not significantly differ in Experiments 1 and 2, suggesting that low-level auditory attention deficits may be present in healthy ageing individuals, but not due to binding. For the control group, this performance was not dependent on whether identification and localisation was required. Since this extinction task is typically used to detect altered attentional processes in stroke patients, it was not expected that the control group would demonstrate extinction; however, healthy individuals have not been thoroughly studied on auditory extinction tasks. This suggests that further normative studies of extinction in ageing individuals be conducted to determine what aspects play a role in the significant extinction observed in non-brain-damaged controls. One possibility is that the performance of 100% accuracy on single trials created an inappropriate comparison for the DSS condition, therefore skewing the results. Due to the fact that non-brain-damaged control individuals have not been used in past literature, it is unknown if the patterns of performance are similar to other research. Future research should further explore the findings of the differential omission errors (right greater than left) as well as omission errors in general.

Using a control group of individuals who have suffered a left hemisphere stroke, but do not present with aphasia, would also be beneficial in determining if language loss plays a role in performance on this task. Two individuals with left hemisphere lesions and no aphasia completed these experiments post-hoc to determine if there was a potential difference in performance. It was observed that these two individuals had fewer omission errors than those individuals with aphasia (see Figure 7). Although only preliminary conclusions can be drawn due to the limited sample size, the data from these two individuals suggest that auditory attention deficits are not just due to stroke, but could be related to the aphasia. It could be argued that extinction deficits exist within a range from aphasia to normal, and potentially these deficits are revealed only under difficult conditions. Future studies will include a formalised group of individuals who have a left hemisphere lesion and no aphasia.

The current findings appear to support the theory that binding may play a role in performance on this task for individuals with aphasia. Alternatively, participants' aphasia may limit resources and the double presentations may use all available resources. Therefore, task requirements (counting as well as identification and localisation) may lead to decreased performance (i.e., an increase proportion of errors). That is, an error-free performance may not be possible even for the nonbinding task as previously predicted, due to the limited availability of resources (McNeil et al., 1991; Murray, 2000). It could be argued that persons with aphasia do not have a deficit in binding *per se*, and that auditory extinction could be due to inefficient or limited resources being available (McNeil et al., 1991; Tseng et al., 1993). For example, it could be argued that more resources are needed to process two stimuli versus one stimulus (DSS vs single presentations) and in a damaged attentional system resources are not allocated efficiently to allow this to occur. It could also be that there is a different amount of resources required for identifying and localising (two steps), whereas counting only requires a single step (note that both require storage, retrieval, speech planning, etc.). One potential way to test this hypothesis would be to combine two aspects that do not require binding (such as identification and counting) but still require a similar amount of resources. The binding theory would purport that if a task required two "things" to be pulled together that were unrelated to binding (i.e., identification and counting), and extinction was still observed, the deficits would not be due to a binding problem (and potentially due to resource allocation). The theory of "binding" would explain extinction as a failure to bind featural and location information together, and would predict that without such a requirement for binding of auditory identification and localisation pathway information, extinction may be greatly reduced. This view entails that the level of extinction is crucially dependent on the requirement to bind together information from

“what” and “where” auditory pathways, and not due to any simple perceptual competition between stimuli. While it is not possible to know how many aspects of the task are being processed, the proposed task would, at the very least, be an alternative test for the binding hypothesis.

Either explanation suggests that auditory attention deficits are present in patients with aphasia, which could lead to decreased performance. This would add supplementary support to Murray (2000) and McNeil et al. (1991), and would also suggest that further work in the area of aphasia assessment techniques could investigate processes in the auditory modality. Future research in the area of aphasia and auditory attention could potentially explore the assessment and rehabilitation of auditory attention.

Another aspect of this study that could be explored further is whether the stimuli used could still be considered low-level linguistic stimuli. It is possible that the letters (“O”, “T”) could interfere with aphasic participants' performance, whereas for RH patients this may not have been an issue, due to the lower level of linguistic involvement for RH lesions (see Shisler et al., 2004). Although omission errors for the letters task and the sex task was not significant (see Experiment 1), processing auditory linguistic information such as letters may be more disruptive to individuals with aphasia. In addition, the sex of the voices differed strikingly in their overall pitch (for ease of distinction between them). This may also contribute to the difficulties the participants had in attending and responding to the assigned stimuli, and it may be that both the letters and the sex of the voice were highly salient and not easy to ignore for these individuals, thus contributing to an increase in errors for both binding and nonbinding tasks. For example, due to the salience of the stimuli it may have been difficult for participants NOT to identify stimuli for the count-only task. This could confound the results of the study and possibly result in extinction for the nonbinding task. Anecdotally, several patients did find it difficult to ignore the letter when asked to name the sex of the voice in the second task.

Another explanation of the difference in performance for Experiment 1 versus Experiment 2 could arguably be temporal grouping. Vuilleumier and Rafal (2000) suggest that in visual attention, two stimuli presented simultaneously may be grouped together pre-attentively (i.e., prior to awareness), when patients are asked to count the information, therefore leading to decreased omission errors. It is possible that a similar phenomenon occurs in the auditory modality for participants with left hemisphere lesions and aphasia, and that the decrease in omission errors for Experiment 2 is due to temporal grouping when participants are asked to count only. Perhaps when participants are not required to bind (Experiment 2), information is grouped together and this then increases performance. If this were the case, however, it would be expected that the control group would have demonstrated differential performance on Experiment 1 versus Experiment 2.

The time post-stroke could also be a factor in the results of this study, since there was a wide range for these participants (from 1 day to 7 years). In general, it appeared that the less time that had elapsed since the stroke, the more extinction was demonstrated; however, analyses could not be completed due to the small *N* for each time period post-stroke. Future studies including more patients would be beneficial to allow an analysis of the effect of time post-stroke on extinction performance differentially. In addition, there were a variety of lesion locations, which could have affected performance. Again, further study including a greater number of participants will allow for an analysis of how lesion location influences performance for extinction. Moreover, aphasia type could contribute to the variable performance rates; however, it is important to note that all participants were identified with the same range of aphasia severity on the BDAE, even if aphasia type differed. Consideration of these factors is important for future research studies in extinction and aphasia. Regardless of the limitations of this study, the finding of extinction and decreased auditory attention in individuals with

aphasia is important to the study of the interplay of attention and language, and should be explored further.

If individuals with aphasia are deficient in the ability to bind together identification and localisation information, this could influence performance in auditory comprehension. Future studies should confirm these preliminary results as well as determine how rehabilitation may be of benefit. For example, working on auditory attention or the underlying system of binding in therapy may lead to increased auditory comprehension performance. These avenues could potentially enhance communication and perhaps, in the future, improve the lives of those individuals with aphasia.

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Figure 1. Equipment and participant placement and required responses. Required responses— Experiment 1a: “T on the left, O on the right”. Experiment 1b: “Male on the left, Female on the right”. Experiment 2: “Two”.

Figure 2.
The rates of omission of stimuli coming from the left speaker or right speaker under conditions of single presentation or DSS for the letter and sex naming and counting tasks.

Figure 3.
The mean number of omission errors for left and right stimuli under conditions of DSS for both letter and sex naming tasks.

Figure 4.
The rates of omission of relevant trials for same or different dichotic pairs.

Figure 5.
The rates of omission of irrelevant trials for same or different dichotic pairs.

Figure 6.
The rates of omission errors for DSS trials from Experiment 2 (Count) compared to Experiment 1 (Identification and Localisation). Note that the stimuli were the same in the two tasks, the only difference being the task instructions.

Figure 7.
The rates of omission of relevant trials for same or different dichotic pairs.

TABLE 1

Demographic and stroke-related data for the six participants with aphasia

Name	Age	Sex	Aphasia type ^a	Severity ^b	Time post-onset ^c	Aetiology ^c
GW	58	F	Broca's	4	7 years	L CVA
WR	42	M	Conduction	4	1 day	L CVA
HA	55	M	Wernicke's	4	39 days	L CVA
HF	74	F	Wernicke's	4	4 months	2 LCVAs
SC	52	F	Wernicke's	4	4 months	SAH
MS	72	F	Mixed	4	19 days	L CVA

^aDetermined by BDAE short form.^bBDAE short form severity score.^cDetermined by review of medical records.

TABLE 2

BDAE data for six participants with aphasia

Percentiles	GW	WR	HA	HF	SC	MS
Conversational speech	100	50	100	100	100	N/A
Auditory Comprehension						
Basic Word Discrimination	100	100	60	65	60	30
Commands	100	100	100	100	70	100
Complex ideational material	50	100	100	100	50	20
Oral expression						
Auto sequences	100	30	100	100	30	0
Repetition of sentences	100	60	100	60	100	0
Boston Naming Test	70	70	75	100	100	N/A
Reading						
Basic symbol recognition (Case & Script)	100	100	100	100	100	N/A
Word identification (Pic-Word Match)	40	100	40	100	20	N/A
Oral sentence reading	100	100	100	100	60	N/A
Reading comp (Sentence/Para)	100	100	100	100	100	N/A

TABLE 3
Percentage of mislocalised omitted stimuli in Experiment 1

		Task	
		Letter	Sex
Aphasia	GW	0	8.3
	WR	0	4.1
	SC	0	0
	HA	0	0
	HF	16.6	4.1
	MS	0	30.4

TABLE 4
Percentage of omissions on DSS trials for Experiments 1 and 2

Participant	Experiment 1		Experiment 2
	Letter	Sex	Count
GW	33.3	87.5	29.1
WR	100	95.8	54.1
SC	50	33.3	25
HA	45.8	37.5	29.1
HF	100	75	62.5
MS	100	100	100
Mean for aphasia group	71.5	71.5	49.96
Mean for control group	22.9	25.5	24.96