Introduction

Production of passive sentences is impaired in individuals with agrammatic aphasia. Some studies have attributed this difficulty to structural impairments (e.g., Friedmann & Grodzinsky, 1997; Schwartz et al., 1994), and others have attributed it to morphological impairments (e.g., Caplan & Hanna, 1989; Faroqi-Shah & Thompson, 2003). However, the source of this deficit remains unclear due to methodological issues present in previous off-line studies. Given that passive morphology is essential for identification of passive structures, it is challenging to examine impairments in passive structures when passive morphology is impaired. To overcome this problem, this study used syntactic priming to elicit active and passive sentence structures, and eye movements as well as speech onset latency were measured during production attempts to determine the source of passive production difficulty.

Methods

Participants. Nine healthy control speakers (age: 18-26) and nine individuals with agrammatic aphasia (age: 38-66, post-onset: 2-17 years) participated in the study. The control participants did not report any history of neurological and psychological disorders prior to the study. The aphasic participants were assessed by the Western Aphasia Battery (Kertesz, 1982, AQ: 71.2 - 82.4), and their aphasia resulted from a thrombo-embolic stroke in the left hemisphere. All were native speakers of English except one aphasic participant who was premorbidly Spanish-English bilingual. All (but one) were right handed, well-educated, and demonstrated good visual and hearing acuity.

Stimuli and Procedures. Forty prime sentences (20 passives, 20 actives), as in (1), were paired with pictures of target transitive events, which elicited target sentences. All sentences were semantically reversible and included two animate nouns (e.g., *boy*, *girl*) and a transitive verb (e.g., *chase*). For the primes, the nouns and verbs used in the active and passive conditions were matched for log lemma frequency (1.64 vs. 1.52, 1.5 vs. 1.64, respectively) using CELEX. For the targets, the nouns used in each condition were also matched for frequency (2.02 vs. 2.11) using CELEX, and the verbs used were the same as those in the primes. On experimental trials, participants listened to and viewed a prime sentence, and then repeated it aloud. Next, a picture appeared and participants were expected to describe it using the primed sentence structure. In order to avoid any delays in word retrieval, participants were familiarized with the nouns and verbs used in the pictures prior to the experiment. During the experiment, participants' eye movements were recorded by an ASL 6000 series remote eye tracker, and their speech was recorded by Praat. Production accuracy, speech onset latency, and eye movements were analyzed.

(1) The boy (N1) is kissing the girl (N2). (Active condition)
The driver (N1) is poked by the boy (N2). (Passive condition)

Results

The control group performed equally well in both active and passive conditions (97.77% vs. 98.88%, respectively) ($p > .05$); whereas, the aphasic group performed significantly more poorly in the passive condition than in the active condition (38.88% vs. 66.11%, respectively) ($p < .05$). Within both the control group and the aphasic group, speech onset latencies in the two conditions were not significantly different (p's > .05).

Both groups also gazed at entities-to-be-produced in the order of and prior to mentioning them, regardless of sentence type, albeit the gaze patterns for the aphasic group were slower than the control group in both conditions (p's < .05). However, when comparing the gaze duration to entities-to-be-produced in the two conditions, a group difference was revealed. Prior to speech onset, the control group gazed longer at the second entity-to-be-produced (i.e., N2 in a sentence) when producing passives than actives (p < .05), whereas the aphasic group did not show this pattern, indicating qualitatively different planning for the two sentence types for the control, but not the aphasic speakers.

Error analysis of the aphasic group revealed that the two most prevalent error types of the passive condition consisted of role reversals (e.g., *The boy is poked by the driver* for *The driver is poked by the boy*) and actives-for-passives (e.g., *The boy is poking the driver*) (42.2% vs. 37.61%, respectively). Errors in passive morphology (e.g., *The driver is poke the boy*) were rarely found (8.26%). Onset latency of passives with role reversal errors was significantly longer than that of actives-for-passives (6907 ms vs. 4219 ms) (p < .01), which was significantly shorter than that of correct passives (5773 ms) (p < .05).

Eye movements associated with incorrect responses were qualitatively different from those with correct responses. For passives produced with role reversal errors, the aphasic group spent a significantly longer time gazing at the first entity-to-be-produced before speech onset as compared to when correct passives were produced (p < .05), indicating difficulty processing passive sentence structure. For active-for-passive errors, however, this longer gaze duration was shown during speech (p < .05), but not before speech. That is, after producing the first entity, the aphasic group spent a longer time looking at it as compared to when correct passives were produced, indicating that this type of error resulted from unprepared sentence production before the speech onset. The eye movements associated with role reversal and active-for-passive errors indicated that these errors were qualitatively different with respect to their underlying nature.

Discussion

Despite relatively preserved production of passive morphology under conditions of syntactic priming, agrammatic speakers still made significant role reversals in passives. On-line data, eye movements as well as speech onset latencies, derived from this study provided evidence of real-time difficulty associated with constructing passive sentence structure, i.e., difficulty assigning correct thematic roles in non-canonical positions. In addition, on-line data indicated that the underlying nature of passives with role reversals is qualitatively different from that of actives-for-passives, which cannot be clearly differentiated with off-line testing methodology. The current findings suggest that impairments in sentence structure, rather than grammatical morphology, contribute to difficulty producing passive sentences in agrammatic aphasia.

Moreover, the eye movement data revealed differences between normal and aphasic speakers with regard to production of passive as compared to active sentences, though this was not the primary focus of this study. The eye data indicated that before speech onset, normal speakers planned both N1 and N2 for passive sentences whereas they only planned N1 for active sentences. Unlike normal speakers, aphasic speakers planned N1 for both sentence types. These data suggests that there are multiple mechanisms of sentence planning available for normal

speakers, and the most appropriate and efficient one can be chosen as needed. However, these choices may not be available for agrammatic aphasic speakers, resulting in using the same processing routines for both active and passive sentences.

References

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