Aphasia and Syntax

William Matchin[1] & Corianne Rogalsky[2]

[1]Department of Linguistics, University of California, San Diego
[2]Speech and Hearing Science, Arizona State University

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1 Introduction

The representations, operations, and principles of syntactic theories are generally held to be claims about how language is actually implemented in the human brain (Chomsky, 1965; 1995; Sprouse & Hornstein, 2016). For this reason, there is powerful potential for research on the nature of linguistic deficits due to brain damage, or *aphasia*, to inform syntactic theory. This is particularly so given that there exist disorders that appear to impair core aspects of language, such as *agrammatism*. Likewise, researchers and clinicians that seek to characterize the deficits in patients with aphasia and to develop assessment and treatment protocols can in principle greatly benefit from the insights into the nature of language provided by syntactic theory. However, there is currently little interaction between theoretical syntax and aphasiology. This is likely due to several reasons, including sociological ones such as the lack of researchers proficient in both fields and ineffective communication among researchers from these very different traditions. However, we suspect that there are deeper reasons for this disconnect. In particular, we suggest two fundamental obstacles: (i) a lack of insight into how grammatical operations apply to real-time sentence processing, and (ii) a focus by syntactic theories on grammatical operations, principles and modules that do not line up well with the currency of functional neuroimaging and neuropsychology: the cortical area. In addition, the assumption that 'agrammatism' is a syndrome caused by a single underlying cognitive source potentially related to a syntactic module is likely false, as is the assumption that damage to Broca's area is necessary and sufficient to cause agrammatism and/or Broca's aphasia. These are related to issues that have been raised by previous authors (Badecker & Caramazza, 1985; Embick & Poeppel, 2005; Embick & Poeppel, 2015; Mohr et al., 1978), and we reinforce them here.

In this chapter we will first outline the methods of research in aphasia and how they have been applied to syntax. Following this, we will review the history of the interaction of these two fields, particularly with respect to the putative syndrome of "agrammatism" that is most relevant to syntactic theory. We will make key observations about the successes and failures of this research. In light of these failures, we propose splitting agrammatism into at least two separate syndromes: one that is tied to deficits in domain-general verbal working memory resources, and another that is tied to a content-addressable memory (CAM) retrieval system operating over syntactic features (McElree et al., 2003; Lewis & Vasishth, 2005). This distinction allows us to capture aspects of agrammatism that appear to be domain-general as well as those that appear to be specific to language. We then suggest some helpful steps to reconnect syntactic theory to the study of aphasia.

2 Aphasia Definitions and Classifications

Aphasia is typically defined as language impairments that are acquired due to a brain injury. Impairments range in severity and can affect auditory speech perception, speech production, reading, and/or writing. Most aphasia research has historically focused on individuals who have experienced a stroke (disruption of blood flow in the brain) resulting in aphasia, but aphasia can result from almost any type of brain injury, including traumatic brain injury, tumor, surgical removal of brain tissue, or infection. Aphasia can also result from neurodegenerative diseases such as frontotemporal dementia, particularly one sub-type often termed primary progressive aphasia (Gorno-Tempini et al., 2011; Mesulam, 2014). While there are numerous ways to classify the subtypes of aphasia, the classifications most relevant to this chapter are discussed below.

2.1 Aphasia Assessments

Typical aphasia assessment measures range from five minute bedside assessments for patients with acute brain damage (i.e. typically within 24 hours of brain injury) to much more extensive test batteries, typically administered by speech-language pathologists in an outpatient setting to chronic patients in order to develop a long-term treatment plan. The details of these assessments can be found elsewhere (e.g. Patterson & Chapey, 2008), but here we will summarize the basic principles of aphasia assessments that are critical when interpreting the existing aphasia literature relevant to syntactic theory, and when designing new experiments to further collaboration between linguists and aphasia researchers.

Perhaps the two most common aphasia batteries referenced in the aphasia research literature are the Western Aphasia Battery (WAB; Kertesz, 2007) and the Boston Diagnostic Aphasia

Examination (BDAE; Goodglass & Kaplan, 1983). Both of these batteries are designed to assess individuals with brain damage on several dimensions of language, including multiple aspects of auditory comprehension (word, sentence and discourse), spontaneous speech production, speech repetition, naming, reading, and writing. Both the WAB and BDAE also contain non-verbal measures, including visual-spatial processing, manual gestures, and mathematical calculations to better understand the specificity of any language deficits present.

The WAB's scoring procedure provides an aphasia classification for each patient, with the possible aphasia classifications of: global, Broca's, transcortical motor, Wernicke's, transcortical sensory, mixed transcortical, conduction, and anomic. The BDAE does not provide criteria for aphasia classifications, but rather an approximate percentile ranking of performance in each language domain tested, with several sub-categories of possible error types within each domain. These percentiles can then be used to compute expressive and comprehension competency indices. Regarding overall severity, the WAB provides an aphasia quotient, which is essentially a composite score indicating the overall severity of speech production and comprehension deficits regardless of the type of aphasia, and the BDAE includes a subjective severity rating between 0-5 for combined speech production and comprehension abilities. Thus, patients designated as having, for example, "severe Broca's aphasia", may vary regarding the exact characteristics of their deficits.

In addition to overall performance in these domains, error types are also tabulated to gain a more precise description of a patient's deficits. For example, in tests of speech production, there are two main types of paraphasias (or word generation errors): phonemic (also known as literal) and verbal. Phonemic paraphasias are typically defined as words in which phoneme substitution errors are present (e.g. *blupt* for *blunt* or *tup* for *top*). Verbal paraphasias are word production errors in which an entire real word is substituted for the target word. If the produced word and the target word are highly semantically related (e.g. *mother* for *wife*), this type of verbal paraphasia is often described as a semantic paraphasia. An error can also be considered a mixed paraphasia if more than one type of error is made within the same word.

Two other types of speech production errors that are examined in aphasia assessments are agrammatic and paragrammatic speech. Agrammatic and paragrammatic speech are discussed in detail below, in relation to Broca's aphasia and Wernicke's aphasia. Briefly, agrammatic speech refers to a general lack of grammatical structure and closed class items, whereas paragrammatic speech refers to the presence of grammatical information, but an overall lack of coherent sentence structures. It is important to note there is no standard clinical definition of "agrammatic" aphasia and the WAB and BDAE do not include a cut-off for performance to be considered agrammatic or paragrammatic. Thus, studies of syntactic processing in aphasia that

examine “agrammatic” patients may potentially have very different participant inclusion criteria.

2.2 Broca’s aphasia

Broca’s aphasia is perhaps the most well-studied of the aphasias, and is most often the focus of testing predictions of syntactic theory in individuals with aphasia. Broca’s aphasia is characterized by effortful, error-filled speech (both spontaneous production and repetition), and relatively intact speech comprehension (Damasio, 1992; Goodglass & Kaplan, 1983) (exceptions to this description of “intact” speech comprehension will be discussed in this chapter as it relates to sentence comprehension impairments). Both phonemic and verbal paraphasias are often present in the speech production of patients with Broca’s aphasia, and patients are typically aware of their deficits and make attempts at error correction. (Goodglass & Kaplan, 1983). Another attribute of Broca’s aphasia that is of particular interest here is agrammatic production[1], i.e. speech production that frequently lacks closed class items, bound morphemes, and grammatical structure, resulting in single word or short phrase utterances (Jakobson, 1956; Goodglass 1968, 1976; Gleason et al. 1975; Kean, 1977). Here is an example of agrammatic speech production in an individual with Broca’s aphasia retelling the tale of Cinderella from Love & Brumm (2012):

> ...Happy. B- all- ballerina. I can’t say it. Uh, name. ... Sisters two. Mother evil. Mop- ing. Dress, bird, and, uh, mouse. One, two, three. Uh, angels? Fairy! Crying and uh, uh, mother uh, mother lock -ed it. … Yeah! Mommy, mommy, mommy! And uh, horse and dog. Wands. Uh, uh, muck lock lop moppins [muffins]. And uh, mouse and birds or? ... Oh well. Uh, bored. Curled. Pretty. And, uh, twelve. Shoe. Uh, run-ning. Yeah. And uh, sisters. Um, shoe? One. Shoe? Right there? Bigger. Uh, and uh, that’s right. … That’s right (motions putting on a shoe). ...Yeah. And affer [ever] and ever.
>
> ...
>
> (*excerpt from Love & Brumm, 2012, p. 207)*

Broca’s aphasia has traditionally been linked to damage in Broca’s area, which is typically defined as the posterior two-thirds of the left inferior frontal gyrus (Anwander et al. 2007;

[1] In existing literature the term *agrammatism* has been used to describe agrammatic production, agrammatic comprehension, or both. In this chapter for consistency and specificity we will use the terms *agrammatic production* and *agrammatic comprehension* to specify which language domain we are referring to, and use *agrammatism* only in regards to overarching theories or deficits related to both domains. Similarly, “agrammatic aphasia” is a frequent participant selection criteria in many of the studies we will be discussing, but it is not defined in a consistent manner; thus we will specify the characteristics of this group in each case as it arises.

Brodmann, 1909). Both the aphasia type and brain region are named after Paul Broca, a French scientist and physician in the mid 1800's who was among the first to relate speech production impairments with left frontal lobe damage. However, in the context of examining syntactic theory in aphasia patients, it is critical to not conflate the impairments of Broca's aphasia, particularly agrammatic production, with damage to Broca's area. Damage to Broca's area is not necessary for the presence of Broca's aphasia (Fridriksson, Bonilha & Rorden, 2007), and damage circumscribed to Broca's area is not sufficient to elicit Broca's aphasia (Mohr et al. 1978; Mohr 1976).

A recent large-scale neuroimaging study indicates that the most common pattern of brain damage associated with Broca's aphasia is damage that includes both the posterior portion of Broca's area (pars opercularis) as well as left posterior temporal regions (Fridriksson et al. 2015). Given the relatively long Euclidean distance between these two regions, it is not surprising that patients with Broca's aphasia often have large left hemisphere lesions that span portions of the frontal, temporal, and parietal lobes (Naeser & Hayward, 1978; Mohr et al. 1978). The only large-scale study of agrammatic production that we are aware of (Wilson et al., 2010) has linked syntactic production deficits with damage to the anterior portion of Broca's area (the pars triangularis), the supplementary motor area, and white matter underlying these structures. These results indicate that Broca's area is clearly implicated in Broca's aphasia and agrammatic production, but that dysfunction in Broca's area is not solely driving the linguistic deficits of these syndromes. This distinction between Broca's aphasia and Broca's area is critical to keep in mind when interpreting much of the existing literature examining various elements of syntactic theory in patients. Many studies have been aimed at better understanding the role of Broca's area in syntactic processing and thus select subjects based on the presence or absence of damage to Broca's area (but the Broca's area patients almost always also have damage in surrounding regions) (e.g. Linebarger et al., 1983, and see Grodzinsky, 2000). Other studies include subjects based on the diagnosis of Broca's aphasia or presence of agrammatic production (e.g. Gleason et al. 1975; Caramazza & Zurif, 1976). In these studies' subjects, the areas of brain damage, and therefore the affected mechanisms, may vary widely. These differences are important to consider when comparing findings across aphasia studies.

2.3 Wernicke's Aphasia

As a group, fluent aphasias are characterized by the relative ease of producing connected speech, yet the speech produced is often error-filled (Gordon, 1998). Wernicke's aphasia is perhaps the most well-known fluent aphasia, and is typically characterized by fluent (i.e. somewhat the opposite of effortful speech seen in Broca's aphasia) speech, but impaired speech comprehension, including single word comprehension. The speech of individuals with

Wernicke's aphasia typically contains both verbal and phonemic paraphasias, and is often described as paragrammatic, i.e. their speech often contains grammatical information, but grammatical, fully-formed sentences are rare (Goodglass & Kaplan, 1983). Individuals with Wernicke's aphasia are generally unaware of their deficits and thus attempts at error correction are minimal.

Love and Brumm (2012) provide a nice example of paragrammatic speech production in Wernicke's aphasia; the task was to retell the Cinderella story (prompted by a picture-only book of the story to reduce memory load and facilitate comprehension of the task):

> First I started with a s- little, small it was the lady's little which wa- was thing that I wanted before I could remember, but I can't do it now. This uh- I look carefully about what he he looked around but he couldn't really try it about there. At the same time, all these things, at least one, two, three people. Which were clever to the people. This, this and she supposed to do that. … I clevered what how much that little thing she went right here. Which is fine. I did as much as I could. At the same time, at the beginning, she started to look at the um, girl who is looking for all this stuff that was going through while he was there and I watched and watched that stuff that was going and through I looked at the mice doing that. …
>
> (*excerpt from Love & Brumm, 2012, p. 210)*

Similar to the relationship between Broca's aphasia and Broca's area, the relationship between Wernicke's aphasia and Wernicke's area is tenuous, at best. In fact, there even is no consensus amongst neuroscientists studying language as to the exact location of Wernicke's area (Tremblay & Dick 2016; Mesulam et al., 2015). Nonetheless, individuals diagnosed with Wernicke's aphasia often have large left temporal-parietal lobe damage (Damasio 1992).

Patients with Wernicke's aphasia have served as a valuable control group in many studies of agrammatic comprehension in Broca's aphasia (e.g. Caramazza & Zurif, 1976; Zurif et al.,1993; Grodzinsky & Finkel, 1998) because comprehension deficits in Wernicke's aphasia can typically be attributed to lexical-semantic or phonological deficits, and thus serve as a control for these types of impairments in sentence-level tasks often used to study agrammatic comprehension.

2.4 Conduction Aphasia

Conduction aphasia is also considered a fluent aphasia, characterized by largely intact auditory speech comprehension, repetition deficits, and phonemic paraphasias (often phoneme substitution errors) in speech production (Bartha & Benke, 2003; Goodglass, 1992; Baldo et al. 2008). Speech production in conduction aphasia is otherwise near normal; these patients do not exhibit the agrammatic production deficits characteristic of Broca's aphasia (Gleason et al., 1975; Goodglass et al., 1994). With respect to agrammatism, conduction aphasia is useful to compare with Broca's aphasia, because individuals with conduction aphasia present with some of the same agrammatic comprehension patterns as seen in Broca's aphasia (Caramazza & Zurif, 1976). Thus conduction aphasia provides an avenue to examine performance dissociations across sentence comprehension tasks and identify potential unique mechanisms underlying agrammatic production and comprehension.

Conduction aphasia has traditionally been framed as a "disconnection syndrome", thought to be due to damage to the arcuate fasciculus, a large white matter pathway that connects the posterior superior temporal lobe and Broca's area (Geschwind, 1965). The predominant theory was that a disconnection between auditory speech representations in the temporal lobe and motor speech representations in Broca's area resulted in the rather selective repetition deficits and phonemic speech errors in conduction aphasia (Wernicke, 1874; Lichtheim 1885). However, there now is strong evidence that conduction aphasia results from cortical damage, not white matter damage. The damage is most frequently in the vicinity of area Spt, a left hemisphere posterior superior temporal region near the end of Sylvian fissure where the temporal and inferior parietal lobes meet (Damasio & Damasio, 1980; Buchsbaum et al., 2011).

There certainly remains debate regarding the cortical (Spt) and white matter damage contributions to conduction aphasia (Fridriksson et al. 2010), largely in part because the arcuate fasciculus feeds into area Spt, and thus the two are quite frequently both damaged by the same brain injury. However, the functional properties of area Spt are now fairly well characterized, and tightly align with the deficits present in conduction aphasia: Spt is frequently implicated in phonological working memory and speech repetition (i.e. auditory-motor integration for speech) (Hickok et al., 2003; Buchsbaum et al. 2011; Rogalsky et al. 2015; Isenberg et al. 2012), and Spt has been shown to be more activated as a function of greater phonological load (Okada et al. 2003; Fegen, Buchsbaum & D'Esposito 2015).

It is generally well agreed upon that the primary characteristics of conduction aphasia (impaired repetition, phonemic paraphasias) result from impairments in phonological processing, which are particularly evident in phonological working memory tasks (Baldo, Klostermann & Dronkers, 2008). Patients with conduction aphasia may perform relatively well on single word or simple phrase repetition tasks, but exhibit significant declines in performance on non-word repetition

tasks, particularly for multi-syllabic non-words due to increased phonological processing demands (Goodglass, 1992). Similarly, repetition of sentences with abstract content is typically more impaired than repetition of more concrete items (Butterworth, Campbell & Howard, 1986). It also has been frequently noted that when patients with conduction aphasia make repetition errors, they often are still able to reproduce the main gist or idea, although it is not a verbatim reproduction (Baldo et al. 2008). The general consensus from these findings and others is that syntactic and semantic processing are largely intact in conduction aphasia, but that deficits arise in tasks where one must rely upon phonological information to be stored and/or retrieved (Baldo et al. 2008; Gvion & Friedmann, 2012). Later in this chapter we discuss how these phonological working memory deficits may be contributing to the selective agrammatic comprehension patterns seen in conduction aphasia, and how this might inform us regarding the underlying mechanisms of agrammatism more generally.

3 Research Methods

One potential roadblock between aphasia research and syntactic theory is a difficulty generating testable hypotheses, particularly regarding selecting the right method and aphasia population(s) to investigate. It also is critical to understand the limitations of current aphasia methods, and potential difficulties that arise due to the substantial individual variability present in aphasia populations (Jarso et al. 2013). These methodological topics are summarized in this section, to provide the linguist with an overview of potential avenues for collaboration.

3.1 Neuropsychology

Aphasia has been investigated by medical professionals for thousands of years (O'Neil, 1980). Early reports from Ancient Greece, Rome and Egypt, as well as much research today, can be classified as neuropsychology studies: patients are assessed on a behavioral task or battery of tasks to determine the selectivity of their linguistic deficits. Many of the initial neuropsychological studies that have provided insights into the neurobiology of sentence processing report single dissociations, i.e. a group of patients with the same diagnosis or symptoms all exhibit a deficit on task X, but not task Y, thereby suggesting that these patients have a selective deficit in the functions required by task X. For example, individuals with Broca's aphasia can perform at or near ceiling for sentence comprehension tasks with high semantic-processing demands, but often exhibit impairments in the comprehension of sentences with complex syntactic structures (example adapted from Van Orden, Pennington & Stone, 2001). This finding would suggest a selective impairment for syntactic processing. However, The "holy grail" of neuropsychological studies has traditionally been the double dissociation (Tuber, 1955;

Van Orden et al. 2001), where two patient groups are tested, and one group is found to be impaired on task X but not task Y, and the other group is impaired on task Y but not task X. A finding of a single dissociation does not rule out the possibility that all individuals with any type of aphasia may perform similarly on semantic versus syntactic comprehension tasks, not just those with Broca's aphasia (perhaps due to overall task difficulty or attentional fatigue). However, a double dissociation between Broca's and Wernicke's aphasia patients on the semantic and syntactic tasks would indicate a selective syntactic deficit in Broca's aphasia and a selective semantic deficit in Wernicke's aphasia. Single and double dissociations found within subsets of individuals with agrammatism (with subsets defined based on aphasia severity, location of brain damage, and/or symptomatology) on sentence comprehension tasks have proved particularly insightful regarding the neural mechanisms of syntactic processing, and are discussed throughout this chapter.

There are several common experimental designs that are used to investigate sentence comprehension abilities in aphasia. "Off-line" tasks such as sentence-picture matching, acceptability judgments, enactment (e.g. manipulating an object as described in the sentence), and memory probe tasks are often employed. Studies also have frequently incorporated "on-line" measures such as reading whole sentences presented all at once, self-paced reading or listening, and error detection tasks (Caplan, Michaud, Hufford & Makris, 2016). It is important to choose the type of task carefully: task-specific cognitive and linguistic demands can affect sentence comprehension performance in individuals with aphasia, particularly related to agrammatic comprehension patterns (Cupples & Inglis, 1993; Caplan, DeDe & Michaud, 2006; Caplan, Michaud & Hufford, 2013). In addition, the brain regions implicated in agrammatic comprehension can significantly vary depending on the task and types of sentence structures used (Gutman et al. 2010; Caplan et al. 2016; Tyler et al. 2011). Lastly, the modality of presentation may also affect sentence comprehension performance and the brain areas implicated: across studies of control subjects, Broca's area is more frequently implicated in visual (reading) tasks than auditory comprehension tasks (see Table 4 in Rogalsky et al. submitted). The more frequent involvement of frontal regions in reading versus auditory tasks may be related to greater involvement of subvocal articulation during reading compared to auditory tasks (Daneman & Newson, 1992; Slowiaczek & Clifton, 1980; Baddeley, Thomson & Buchanan, 1975).

In summary, neuropsychological approaches to studying sentence processing in aphasia have (and continue to) provide valuable insights into the nature of sentence processing deficits that can result from brain damage (discussed in detail in section 4 below). Nonetheless, task differences are important to consider when interpreting aphasia research findings, as the

relative cognitive and linguistic demands may reduce or exacerbate sentence comprehension deficits in individuals with aphasia.

3.2 Neuroimaging and Lesion-Symptom Mapping

For researchers interested in investigating the function(s) of a particular area of the brain, the advent of accessible neuroimaging techniques in the past 30 years has provided a wealth of resources to aphasia researchers to better describe and quantify the location of brain damage. However, not all structural MRI scans are created equal; there are several types of structural MRIs used in aphasia research, each providing different insights into the spatial extent, degree, and nature of the brain damage (for a review related to aphasia research, see Shahid et al. 2017 and Fisher, Prichard & Warach, 1995).

Lesion-symptom mapping is a term used to describe a group of prominent methods used by cognitive neuroscientists to identify what anatomical locations in the brain are critical for a given behavioral task. Lesion-symptom mapping is essentially the marriage of neuropsychological and neuroimaging techniques. The gold standard for lesion-symptom mapping studies also is the double dissociation (Teuber 1955): if damage to one area of the brain is related to a deficit in task X but not task Y, and damage to another brain area is related to a deficit in task Y but not X, then one can make meaningful conclusions regarding the specificity of functions supported by the two brain areas. While there are valid concerns regarding the assumption of double dissociations that the brain is organized in a modular fashion, there is a general consensus that a double dissociation reflects that the two tasks require distinct, although perhaps not independent, neural resources (Shallice, 1979; Van Orden, Pennington & Stone, 2001).

The first lesion-symptom mapping studies aimed at localizing specific linguistic functions to specific brain regions were region of interest (ROI) based. One approach was to identify participants according to the presence of a set of behavioral symptoms of interest, and to locate regions of brain damage that had high degrees of overlap in those participants. A second approach is to identify participants based on the presence of damage in a particular ROI, e.g. Broca's area, and compare their performance on a behavioral task (or tasks) to the performance of participants without damage to that ROI (either control subjects or patients with brain damage elsewhere). ROI studies continue to provide valuable information regarding the anatomical localization of language functions, much of which we discuss in subsequent sections of this chapter. However, it is important to note that while ROI studies can implicate a particular brain region in a particular function, they typically cannot determine if the function is

specific to the ROI, or if the function is supported by regions adjacent to the ROI that are also typically damaged simply due to the vascular structure of the brain including underlying white matter pathways of the ROI that connect distant brain regions.

Voxel-based lesion-symptom mapping (VLSM; Bates et al. 2003) currently is a popular technique to determine if patients with and without damage within each voxel of the brain perform significantly differently on a particular behavioral task. A voxel is the three-dimensional unit of data generated by an MRI scan; typical resolution of structural MRI scans used in VLSM studies is a voxel size of $1mm^3$ (Huettel, Song & McCarthy 2014, p. 13). There are several variations to VLSM, but the overall approach is to calculate a t-test or other appropriate statistic for each voxel in the brain, to determine if patients with damage in that voxel perform significantly differently on a behavioral task than patients without damage in that voxel (see Rorden, Kamath and Bonilha, 2007 for a discussion regarding the appropriate statistics to use in VLSM). Analyses of Covariance (Bates et al. 2003) and multivariate analyses (Caplan et al. 2007; Yourganov et al. 2016) also can be used in VLSM to identify critical regions for a task when several regions are implicated (Wilson, 2016). These post-hoc approaches allow researchers to better understand connectivity within language networks of the brain, which is critical given that there is mounting evidence from aphasia and typical language processing that single brain regions do not support a language task in isolation, but rather are part of complex, dynamic functional brain networks (Sebastian et al. 2017; Wilson 2016).

Similar techniques to VLSM also are used with known anatomical or functional regions of interest as the unit of measurement instead of voxels, such as sub-regions of Broca's area, primary auditory cortex, the superior temporal gyrus, etc. (e.g. Caplan et al. 2016). While this ROI lesion-symptom mapping approach may have reduced spatial resolution, it also is one way to reduce multiple comparison problems inherent to voxel-based approaches, may improve statistical power, and potentially better accommodates individual variability of the functional organization within an anatomical region.

Voxel-based morphometry (VBM; Ashburner & Friston, 2000) is another technique to investigate brain-behavior relationships. VBM has the same goal of VLSM, to identify voxels or brain regions in which abnormalities are associated with decreased performance on a particular task. An advantage to VBM is that it also can be used in patient populations that often have graded atrophy or more subtle brain damage than stroke patients, such as individuals with primary progressive aphasia or other neurodegenerative diseases. This is possible because VBM, uses a continuous measure, tissue density in each voxel, instead of the binary "damaged" or "not damaged" distinctions in VLSM. This difference allows for greater sensitivity to different degrees of tissue atrophy and eliminates some of the arbitrary decisions regarding the

threshold for considering a voxel to be "damaged." Gray matter and/or white matter density can be examined using VBM. Notably, the only large-scale lesion-symptom mapping study of agrammatic production (discussed later in the chapter, Wilson et al. 2010a) was conducted using VBM in individuals with primary progressive aphasia. For a detailed description and comparison of VLSM and VBM, please see Wilson (2016).

3.3 Individual Variability, Compensation, Functional Reorganization

No neuropsychology or lesion-symptom mapping method can lead one to predict with even close to 100% accuracy how any one patient will perform on any given language task. Individual variability is quite high amongst patients with the same aphasia diagnosis, as well as amongst patients with highly similar areas of brain damage (Pedersen, Vinter & Olsen, 2004; Kertesz & McCabe, 1977; Lazar & Antoniello, 2008; Lazar et al. 2008). For example, initial severity of deficits and lesion size explain only ~30-40% of variability in language abilities of chronic aphasia patients (Lazar et al. 2008). Jarso et al. (2013) identify four distinct neurobiological mechanisms that affect language abilities post-stroke other than the location and size of the stroke: "(a) reperfusion [return of blood flow into an area initially blocked by the stroke]; (b) recovery from diaschisis [i.e. disruption of function in healthy regions due to damage in functionally or structurally connected regions]; (c) recovery from structural disconnection; and (d) "reorganization" of language (p.454)." Reorganization in this case refers to the process of intact brain structures supporting functions previously supported by damaged regions.

One way to partially circumvent these largely unpredictable neurobiological factors is to examine only acute aphasia patients, i.e. test individuals within approximately 24-48 hours of experiencing a stroke so that reperfusion, recovery and reorganization are minimized (Hillis & Heidler, 2002; Jarso et al. 2013). There are of course many challenges to testing aphasia patients at their bedside immediately after a stroke, including difficulties with recruitment, having sufficient time available for testing, and the physical and mental fatigue that often are pronounced in the acute stages of stroke (Wilson, 2016). Nonetheless, some prominent aphasia researchers have successfully collected language data on acute aphasia patients, thereby providing valuable insights into a variety of language functions, including sentence processing, which are discussed in this chapter; the work of Argye Hillis and colleagues has been particularly pioneering in this area. Despite the potential benefits of testing acute aphasia patients, there also are additional factors that can affect sentence comprehension performance in even acute aphasia data, including motivation, overall health, and pre-stroke cognitive (e.g. working memory, attention), meta-linguistic and verbal abilities.

For lesion-symptom mapping studies, an additional source of variability is individual differences in pre-stroke laterality of language networks. In right-handed individuals, the vast majority of strokes that cause language deficits are in the left hemisphere. But, this does not mean that the left hemisphere alone supports language function, especially speech comprehension. Numerous functional MRI studies identify bilateral temporal regions to be activated by auditory speech stimuli (Binder et al. 1994; Hickok & Poeppel 2007) and epilepsy patients with their left hemisphere anesthetized during a presurgical procedure still perform significantly above chance on a speech comprehension task using only their right hemisphere (Hickok et al. 2008). Deficits for complex sentences or sentences with non-canonical word order in patients with right hemisphere damage has also been shown (Caplan et al., 1996), illustrating some contribution of the right hemisphere to higher aspects of language. Thus, it is not surprising that many reported "comprehension deficits" associated with left hemisphere damage are certainly declines in performance compared to a control group, but often the patients are not performing at chance or at floor level because of right hemisphere contributions to the task.

Together, these sources of potential individual variability in aphasia studies require careful consideration when designing and interpreting small case studies, and provide strong evidence for the need of large neuropsychological and lesion-symptom mapping studies with sufficient statistical power that include covariates for the sources of variability that can be quantified (see Shahid et al. 2017 for an in-depth discussion of power analyses for lesion-symptom mapping studies).

3.4 Cognitive Deficits in Aphasia

Although one may tend to think of an individual with aphasia as having specific deficits restricted to the language domain (speech production, comprehension, etc.), this is rarely the case. Domain-general cognitive functions such as working memory, attention, general alertness, cognitive control and inhibition have been found to be impaired in many individuals with aphasia; the nature and severity of these cognitive abilities widely varies both within and across types of aphasia (Caspari et al., 1998; Brownsett et al, 2014; Murray, Holland & Beeson, 1997, 1998; Erickson, Goldinger & LaPointe, 1996; Pettigrew & Hillis, 2014). Critically for the present chapter, verbal working memory deficits are frequently present in individuals with Broca's aphasia as well as in individuals with conduction aphasia (Caspari et al. 1998), which may explain some of the sentence comprehension patterns present in both of these aphasia populations.

The reason for the overlap of language and cognitive deficits in aphasia is two-fold: (i) the pattern of damage resulting from a stroke does not adhere to fine-grained functional

boundaries in the brain, but rather is determined by anatomical and vascular boundaries which can often group together distinct cognitive and linguistic processes (Fedorenko, Duncan & Kanwisher, 2012); thus brain tissues supporting multiple domains are often affected by the same brain injury and (ii) domain-general cognitive resources support language processing (e.g. Vaden et al, 2013), thus when they are damaged, language impairments can arise (Geranmayeh, Brownsett & Wise, 2014). As discussed earlier, common behavioral tasks used to measure sentence comprehension have been shown to tax linguistic and cognitive demands in different ways (Caplan et al. 2016). Therefore it is critical to use tasks in aphasia research that do not introduce unintended cognitive demands that may be unrelated to the study's hypotheses. The different cognitive demands present across tasks also can be used to a researcher's advantage to examine the role of different cognitive functions in syntactic processing or other linguistic functions. For example, performance differences on sentence comprehension tasks with varying levels of working memory demands can be compared to determine how performance may change when working memory resources are taxed and thus not available to support the syntactic processing demands.

The remainder of the chapter examines the previous interactions between aphasia research and syntactic theory, and proposes new directions for advancing our knowledge of the neural computations of sentence processing through collaboration between the two fields.

4 Aphasia and syntactic theory: a history[2]

The history of aphasia and syntax can be characterized by a long period of accumulating clinical observations about the grammatical abilities of patients with aphasia, a period of intense interaction between theoretical syntax and aphasia starting in the 1970s and lasting until the early-mid 2000s, and finally a return to a focus on more basic observations and classifications of linguistic deficits largely divorced from syntactic theory, albeit with more sophisticated research methods. The history of this interaction has proved quite informative about the relation between syntactic theory and the nature of linguistic deficits in aphasia. In particular, the most widely investigated syntactic theory in aphasia is the theory of Government and Binding (GB; Chomsky, 1981). Therefore, our review will chiefly concern GB and its successor within mainstream generative grammar, the Minimalist Program (MP; Chomsky, 1995; Adger, 2003; Hornstein, 2008). However, the conclusions we make here should be relevant to researchers in other frameworks, such as tree-adjoining grammar (TAG; Joshi, 1985; Frank, 2002), head-driven

[2] See Caplan & Hildebrandt (1988) and Avrutin (2001) for similar reviews of agrammatism that discuss aphasia in terms of syntactic theory.

phrase structure grammar (HPSG; Pollard & Sag, 1994), lexical-functional grammar (LFG; Bresnan, 2001), and construction grammar (CxG; Goldberg, 1995).

4.1 Agrammatism and the syntacto-topic conjecture

The period of close association between syntax and aphasiology began with the discovery that patients with Broca's aphasia not only had a disorder of speech production, but also problems with sentence comprehension (Heilman & Scholes, 1976; Caramazza & Zurif, 1976; Schwartz et al., 1980). Here we focus on the study by Caramazza & Zurif (1976), which is widely cited as a turning point in the understanding of the nature of agrammatism in Broca's aphasia that triggered the association of this clinical syndrome with syntactic theory. The key observation of Caramazza & Zurif (1976) was that patients with Broca's aphasia performed at chance at understanding object-relative constructions when they were semantically *reversible* (1), but not when they were *non-reversible* (2). That is, these patients had difficulty when the thematic relation among the arguments of the sentence could not be reconstructed based only on the identity of the arguments themselves without knowing their syntactic configurations. Importantly, the deficit is less severe for sentences with canonical word order, leading to the general pattern of better performance for e.g. reversible active sentences (3) compared to reversible passive (4) sentences, (e.g. Schwartz et al., 1980; see Grodzinsky et al., 1999 and Grodzinsky, 2000 for reviews). This pattern of comprehension breakdown, impaired performance for sentences with non-canonical word order and reversible thematic mapping, is called *agrammatic comprehension*.

(1) The apple that the boy is eating is red. (better)
(2) The boy that the girl is chasing is tall. (worse)

(3) The girl pushed the boy (better)
(4) The boy was pushed by the girl (worse)

The original hypothesis stemming from this research was that patients with agrammatic comprehension lacked the ability to generate a detailed structural representation of sentences. The idea was that they could guess the correct meaning of non-reversible sentences given the arguments and plausible thematic relations between them, but they could not rely on this strategy to correctly identify the meaning of semantically reversible sentences, which provided no such clues. Combined with a strategy that assigned an agent role to the first argument of the sentence in reversible sentences (Grodzinsky, 1986), the syndrome appeared to follow from such a structural deficit. The hypothesis that a central syntactic deficit underlies the syndrome

of agrammatism was called the *overarching agrammatism* hypothesis, with the associated claim that Broca's area is the locus of syntactic operations (Caramazza & Zurif, 1976; Berndt & Caramazza, 1980; Zurif, 1980; Schwartz et al, 1980). This proposal sought to unify agrammatic production and comprehension as resulting from damage to a common component of the normal linguistic system, namely syntax. This conclusion sparked the intensive study of aphasia in the context of theoretical syntax - that is, characterizing linguistic deficits as impairments to grammatical competence. These investigations were often performed in relation to Government and Binding theory (Chomsky, 1980), as well as computational and psycholinguistic approaches, making for strong integration of disciplines in the study of aphasia (see Caplan & Hildebrandt, 1988 for extensive discussion of syntactic theory and parsing theory in the context of aphasia and agrammatism). As we discuss later, this degree of integration among the disciplines is astonishing in light of the current state of affairs, which has little such integration.

The biggest development regarding the use of syntactic theory in the study of agrammatism in Broca's aphasia was the trace-deletion hypothesis (TDH; Grodzinsky, 1986; 2000). The TDH states that the Broca's aphasia patients' comprehension deficits are restricted to a subcomponent of the grammar, in particular, that the structural representations of sentences in patients with agrammatism lack traces of movement. This line of research identified a specific module of GB theory - the syntactic operation Move-α, or the transformational component of the grammar - as the functional role of Broca's area in language. Thus the TDH represented a major point of integration between the successful advances of syntactic theories in generative grammar and the clinical study of aphasia. More broadly, the success of this hypothesis led the way to a more general goal of integration among these fields called the *syntacto-topic conjecture* (STC; Grodzinsky, 2006; Grodzinsky & Friederici, 2006):

1. Major syntactic operations are neurologically individuated.
2. The organization of these operations in brain space is linguistically significant.

(1) states that there is a transparent mapping between grammatical operations or modules and the functions of brain areas - in other words, there are spots of brain that "do" particular grammatical operations. This was a central claim of the TDH - that the Movement module was localized to Broca's area. As we discuss in this chapter, much of the data presented in favor of the TDH addressed the localization claim by pointing to deficits in patients with assumed damage to Broca's area, and the eventual demise of the TDH resulted from contradictory empirical data in these patients. Neither the TDH or any related proposal of agrammatism based on syntactic theory attempted to address (2), the hypothesis that the locations of grammatical modules in the brain reflects some important principle of cognitive organization. It

is unclear exactly why Movement or any other particular syntactic module should be localized to Broca's area in particular - in our view, an understated general flaw of these theories. The failure of the TDH exemplifies the general failure of the STC, raising critical questions regarding the relation between syntactic theory and the brain and potentially informs the proper formulation of syntactic theory for the purposes of alignment to aphasiology. As such, we will focus our discussion mainly on the TDH.

There were several key pieces of evidence underlying the TDH as opposed to a more general syntactic deficit. Notably, patients with Broca's aphasia showed selective patterns of deficits - for instance, in many studies they performed well on sentences with canonical word order. As Grodzinsky (1986) pointed out, in order to interpret thematic relations some structural relation is necessary, as theta assignment occurs in particular syntactic configurations; this indicated that these patients did not lack syntactic competence entirely. According to Grodzinsky (1986), the agrammatic comprehension pattern could be accounted for by a restricted syntactic deficit contingent on two key elements: (i) deletion of traces from the structural representations of sentences, and (ii) an agent-first strategy that assigned the thematic role of Agent to arguments lacking a thematic role due to the inability of receiving one through a trace. Thus, patients were predicted to correctly understand simple active sentences because no traces were involved, and they were predicted to correctly understand subject-relative sentences and subject clefts (which presumably involve a Movement transformation) because the agent-first strategy usually assigned the same theta role to the argument that it would have received via a trace in the relative clause. By contrast, for sentences with non-canonical word order, an agent role would be assigned to the grammatical subject, leading to random interpretation because a competing argument was also assigned an agent role in its base-generated theta position. This was exactly the pattern that was seen - intact performance on actives, subject-relatives and subject clefts, with impaired performance on reversible passives, object-relatives and object clefts. The agent-first strategy was also independently proposed by Bever (1970) to account for behavioral data in normal subjects, supporting its use in explaining the behavioral pattern in agrammatism. This pattern of selective comprehension deficits was the strongest argument that Grodzinsky offered in favor of the TDH, although there was additional support from neuroimaging studies: increased activity related to the presence (Ben-Shachar et al., 2003) and distance (Santi & Grodzinsky, 2007a) of Movement transformations in Broca's area.

Other patterns of data in Broca's aphasia deepened the connections between aphasiology and syntactic theory. Hickok and colleagues highlighted several comprehension patterns that were problematic for the TDH: impaired comprehension of the matrix clause in subject-relative sentences (Caramazza & Zurif, 1976; Hickok, 1993) and impaired comprehension of *which*-N WH-questions with intact comprehension of bare *who* WH-questions (Hickok & Avrutin, 1996).

This led Hickok and colleague to develop the Revised Trace Deletion Hypothesis (RTDH), incorporating recent advances in syntactic theory concerning the VP-internal subject hypothesis (Kitagawa, 1986; Burton & Grimshaw, 1992), D-linking (Pesetsky, 1987), and the hypothesized distinction between Government and Binding chains (Cinque 1990). Additionally, patients with Broca's aphasia were argued to have difficulties comprehending sentences with phrasal movement but not head movement (Grodzinsky & Finkel, 1998, cf. Wilson & Saygin, 2004). These data were used by Chomsky (1999) to support the notion that head movement should be treated as a PF phenomenon rather than as a core syntactic operation. With respect to agrammatic production, Friedmann & Grodzinsky (1997; 2000) reviewed studies of production deficits in Broca's aphasia that indicated largely preserved agreement morphology with deficits in tense, which they explained through a grammatical deficit that pruned the upper nodes of the tree structure, leaving lower nodes intact. This converged with the split-INFL hypothesis that proposed separate structural positions for tense and agreement features (Pollock, 1989; Chomsky, 1991). More recently, there has been some work on explaining comprehension deficits in agrammatic Broca's aphasia through the lens of Relativized Minimality (RM; Rizzi, 1990), a locality constraint on syntactic operations. Grillo (2008; 2009) and Garraffa & Grillo (2008) have attempted to characterize agrammatic comprehension and production as resulting from impoverished syntactic representations that end up causing RM violations. This account assumes previous hypotheses of reduced syntactic processing resources in agrammatism (Kolk, 1995; Zurif et al., 1993), and extends these hypotheses particularly to syntactic representations at *phase edges* (Chomsky, 2001), namely nominal (DP), verbal (vP), and clausal (CP) projections. Thus, developments in syntactic theory and aphasiology of agrammatism mutually reinforced each other, appearing to make good on the mentalistic commitments of syntactic theory.

4.2 The failure of the Trace-Deletion Hypothesis and the Syntacto-Topic Conjecture

While the TDH and related proposals derived from syntactic theory were useful in driving research and promoting interaction between cognitive neuroscience and syntactic theory, it is clear that the essence of these proposals is incorrect. The main reason for this is that Broca's aphasia patients with agrammatic comprehension have behavioral patterns that are clearly contradictory and/or mysterious under these proposals. The conclusions of those well-versed in linguistic theory who study aphasia and neuroscience are that these patients generally have *intact grammatical knowledge*, and that their deficits stem from problems in the *use* of this knowledge (Edwards & Lightfoot, 2000; Hickok & Avrutin, 1995; Zurif et al., 1993). Additionally, the TDH and related proposals suffer from problems with explanatory adequacy: why should brain damage selectively target *these* syntactic representations and/or operations, and not others? The failure of the TDH, the most successful example of the STC, leaves the field of

aphasiology without a clear understanding of how syntactic theory relates to language deficits due to brain damage - clearly an undesirable state of affairs.

An important set of data regarding agrammatism and its relation to syntactic theory are the careful studies of acceptability judgments in aphasia. With respect to the TDH, Broca's aphasia patients have shown dissociations between their "agrammatic" comprehension patterns and their intact ability to make subtle acceptability judgments about a wide range of grammatical structures, including those with phrasal movement (Linebarger et al., 1983; Wilson & Saygin, 2004). We will discuss some of these findings in detail to illustrate the problem that these studies raise for the TDH and STC as well as helping define the set of data to be explained by hypotheses about the deficits underlying agrammatic comprehension and production.

Linebarger et al. (1983) tested four patients with Broca's aphasia, confirmed to have brain lesions that included damage to Broca's area, on an acceptability judgment test. All four patients had notable sentence comprehension deficits characteristic of agrammatic comprehension, including worse performance on reversible passive than reversible active sentences[3]. The task consisted of ten conditions designed to test a variety of facets of grammatical knowledge, listed below along with examples from the grammatical and ungrammatical conditions from each condition.

1. Strict subcategorization
    a. *He came my house at six o'clock.
    b. He came to me house at six o'clock.
    c. *I hope you to go to the store now.
    d. I want you to go to the store now.
2. Particle movement
    a. *She went the stairs up in a hurry.
    b. She went up the stairs in a hurry.
    c. She rolled the carpet up in a hurry.
3. Subject-aux inversion
    a. *Is the boy is having a good time?
    b. Is the boy having a good time?
4. Empty elements
    a. *This job was expected Frank to get.
    b. Which job did you expect Alfred to get?
    c. Frank was expected to get the job.
    d. *The workmen were expected would finish by noon.

[3] All four patients had some deficits on reversible active sentences, an important point to which we return later.

5. Tag question: subject copying
    a. *The little boy fell down, didn't it?
    b. The little boy fell down, didn't he?
6. Left branch condition
    a. *How many did you see birds in the park?
    b. How many birds did you see in the park?
7. Gapless relative clauses
    a. *Mary ate the bread that I baked a cake.
    b. Mary ate the bread that I baked.
8. Phrase structure rules
    a. *The gift my mother is very nice.
    b. The gift my mother got is very nice.
    c. The gift for my mother is very nice.
9. Reflexives
    a. *I helped themselves to the birthday cake.
    b. I helped myself to the birthday cake.
    c. *The famous man itself attended the ceremony.
    d. The famous man himself attended the ceremony.
10. Tag questions: aux copying
    a. *John is very tall, doesn't he?
    b. John is very tall, isn't he?

The stimuli were designed in order to prevent the patients from being able to determine well-formedness based on local information alone - both ungrammatical and grammatical sentences contained linear word sequences that appeared in grammatical sentences. Approximately 40 trials from each condition were presented to each patient (20 grammatical, 20 ungrammatical). The results (Figure 1) indicate that these patients performed remarkably well - most conditions were well above chance for all four subjects, with notable decrements of performance for the three conditions that involve agreement: tag questions (subject and auxiliary) and reflexives, an interesting data point to which we return later. These results indicate that these 'agrammatic' patients in fact have largely intact grammatical knowledge, with the possible exception of agreement, and that sentence comprehension deficits of reversible active and passive sentences likely originate from non-grammatical deficits.

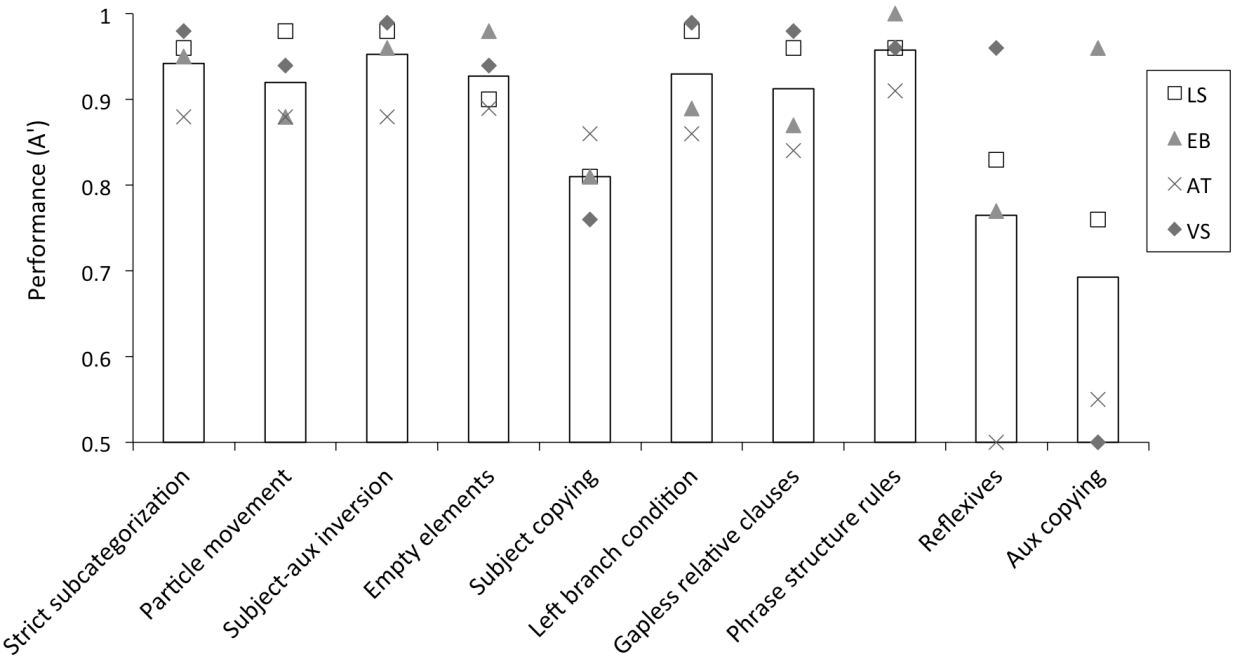

Figure 1. Acceptability judgment data reproduced from Linebarger et al. (1983). Y-axis reflects the A' value for each experimental condition. White bars indicate the average across subjects, and individual characters mark each subject's performance. An A' value of .5 indicates chance performance on the task. X-axis indicates the experimental condition. The key on the right indicates the patient that corresponds to each icon. Two null A' values reported in Linebarger have been included here as .5, as these two values resulted from either zero "yes" or zero "no" responses across the grammatical or ungrammatical examples, respectively, from that condition.

Wilson & Saygin (2004) performed a similar study to identify the patterns of brain damage associated with deficits of grammatical knowledge, with a focus on comparing constructions requiring intact phrasal movement chains to a variety of other constructions not involving phrasal movement (Table 1), in addition to performing VLSM analyses to identify brain areas associated with deficits of grammatical knowledge. They divided their stimuli into those that they intuitively found more or less difficult within each condition. Age-matched control subjects performed well, although not at ceiling. Patients with damage including Broca's area (the pIFG) showed similar deficits for sentences with and without phrasal movement (Figure 2), and patients without damage to Broca's area showed the same pattern. Patients with damage to the posterior temporal lobe showed the most severe deficits, and whole-brain VLSM analyses showed little evidence of IFG involvement in deficits on constructions with or without phrasal movement. These results are consistent with similar large-scale analyses of sentence comprehension (Dick et al., 2001; Caplan et al., 1996; Thothathiri et al. 2012; Pillay et al. 2017; Magnusdottir et al. 2013; Rogalsky et al., submitted) suggesting no particular association of Broca's area with grammatical knowledge.[4]

[4] Some lesion studies have shown involvement of the Broca's area in sentence comprehension, particularly for complex sentences with non-canonical word order (e.g., Mesulam et al., 2015), but the general pattern across

Condition	Grammatical	Ungrammatical
Trace/Hard	David seems likely to win	*John seems that it is likely to win.
	Which woman did David think saw Pete?	*Which woman did John think that saw Tony?
Trace/Easy	The dog which bit me was black.	*Me the dog which bit was black.
	What did Bill buy besides apples?	*What did Bill buy oranges and?
Other/Hard	Could they have left without me?	*Could have they left without us?
	He donated the books to the library.	*She donated the library the books.
Other/Easy	The children threw the football over the fence.	*The children sang the football over the fence.
	Could they have left town?	*Have they could left the city?

Table 1 (from Wilson & Saygin, 2004). Examples of stimuli from each condition

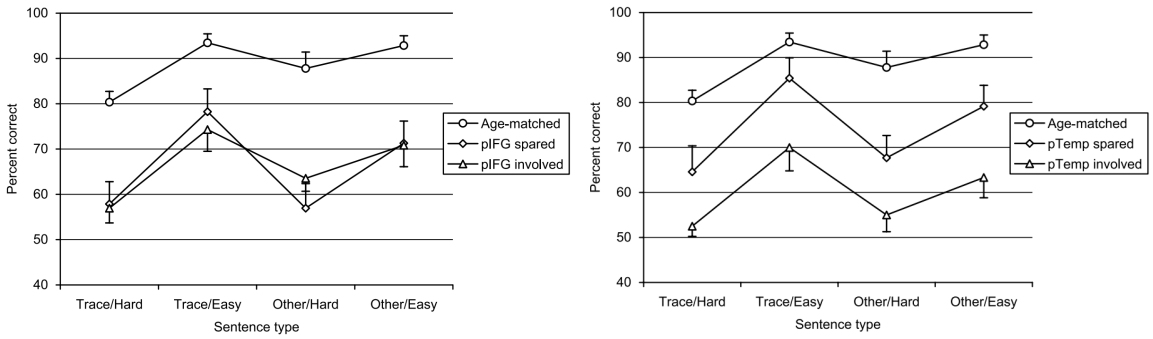

Figure 2 (adapted from Wilson & Saygin, 2004). Performance broken down by lesion location. pIFG = posterior inferior frontal gyrus (Broca's area), pTemp = posterior temporal lobe.

The results of these acceptability judgment studies demonstrate that patients with agrammatic Broca's aphasia and/or damage to Broca's area have a surprisingly good ability to judge syntactic well-formedness (with the possible exception of agreement), and that there is no particular association of Broca's aphasia or damage to Broca's area with syntactic Movement. Another problem for the TDH and related proposals is that even sentence comprehension

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studies, particularly with respect to basic sentence comprehension, suggests limited involvement of this region in grammatical knowledge.

deficits do not follow straightforwardly from these theories. For instance, comprehension of pronouns and reflexives (Hickok, 1993; Grodzinsky, 1993; Santi & Grodzinsky, 2007b) and reversible simple active and locative constructions (Grodzinsky et al., 1999; Schwartz et al., 1980) are impaired in agrammatic patients to varying extents, none of which depend on intact traces. These problematic patterns of behavioral data are coupled with neuroimaging data that indicate no selectivity in Broca's area for linear distance in Movement dependencies when compared to anaphoric dependencies (Matchin et al., 2014), illustrating that activity in this region is not tied to Movement operations but rather the processing of long-distance dependencies that place demands on memory resources.

Additionally, it is not clear how the TDH would account for agrammatic production deficits in Broca's aphasia, which seem unrelated to syntactic movement. The RM approach to agrammatism (Garraffa & Grillo, 2008; Grillo, 2008) fares better, as it predicts problems with all of these representations levels due to a general problem with syntactic features at phase edges that underlies both their comprehension and production deficits. However, the agrammatic production deficits of Broca's aphasia cannot be well described as a categorical loss of certain morphological categories or syntactic features, as deficits in production of functional morphology are dependent on the position of the item within a sentence. Gleason et al. (1975) showed that the omission of determiners and pronouns in 8 agrammatic Broca's aphasic patients occurred largely in sentence initial position, with significantly improved performance in sentence medial position. Additionally, Dutch-speaking patients with agrammatic Broca's aphasia do not produce inflected verb forms in incorrect syntactic positions (Bastiannse & van Zonneveld, 1998), suggesting problems in production functional morphology but not a lack of grammatical knowledge regarding the distribution of these morphemes. Such data are difficult to explain with a theory that makes categorical cuts across grammatical representations, whether specific to Movement, phase edge features, or otherwise. Across the whole profile of sentence comprehension, production, and acceptability judgment studies, a coherent theory of the underlying deficits in agrammatism derived from syntactic theory appears impossible.

An important point in this context is that individuals with Broca's aphasia often have cognitive deficits that are suggestive of non-linguistic processing explanations for their sentence comprehension deficits. Chief among these is working memory. Performance on standard batteries for aphasia correlate with measures of verbal working memory capacity (Caspari et al., 1998), and damage to Broca's area is correlated with impaired verbal working memory capacity (Pettigrew & Hillis, 2014). In fact, the seminal study that spawned the association of Broca's aphasia with a syntactic deficit, Caramazza & Zurif (1976), found the same 'agrammatic' sentence comprehension pattern (deficits on reversible sentences with non-canonical word order) in patients with conduction aphasia. Patients with conduction aphasia in general do not

exhibit agrammatic production, and do not have damage to Broca's area (Damasio & Damasio, 1980; Buchsbaum et al., 2011). These patients *do* have impaired phonological working memory capacity that appears tied to their 'agrammatic' sentence comprehension deficits (Friedmann & Gvion, 2003; Gvion & Friedmann, 2012), however. This suggests that the underlying cause of the sentence comprehension deficits in agrammatism in both Broca's and conduction aphasia lies in reductions in the working memory resources required to process sentences rather than grammatical knowledge itself.

In support of this idea, studies that attempt to limit the working memory capacities of healthy adults using attention-demanding concurrent secondary tasks or degradation of intelligibility of stimulus materials have shown similar 'agrammatic' comprehension patterns as some patients with aphasia (Dick et al., 2001; Rogalsky et al., 2008). Similarly, the behavior of children mirrors that of patients with agrammatism. Nakayama (1987) found that children made more errors on non-canonical as compared to canonical word order in their elicited sentence production, and Crain & Nakayama (1987) found that children's errors were related to increased length of dependencies. This similarity is plausibly due to the fact that children have limited working memory capacities (Cowan et al., 2006). Additionally, it is clear that Broca's aphasia patients do not process linguistic material as quickly as healthy adults. Evidence for this is that these patients show delayed lexical priming compared to age-matched control subjects (Prather et al., 1992), and when the rate of sentence presentation is slowed, their comprehension of sentences with non-canonical word order significantly improves (Love et al., 2008). Zurif et al. (1993) found that Broca's aphasia patients did not show predictive lexical priming effects for filler-gap dependencies, while Wernicke's aphasia patients and healthy controls do show these effects (Crain & Fodor, 1995; Stowe, 1986; Frazier & Flores d'Arcais, 1989) - this suggests that patients with Broca's aphasia do not use an active gap-filling procedure typical of intact grammatical processing. Regarding Broca's area, ERP data from Jakuszeit et al. (2013), shows that patients with damage in and around Broca's area do not show an early negativity associated with agreement violations that was present in healthy control subjects (although these patients did show an early negativity to phrase structure violations). These data are difficult to explain via categorical deficits in grammatical operations or representations, and are much more compatible with processing resource limitations.

In general, many scientists who once advocated an explanation of agrammatism in Broca's aphasia through a syntactic deficit have changed their positions in light of the data reviewed above, including Caramazza (Caramazza & Berndt, 1985; Badecker & Caramazza, 1985), Schwartz (Linebarger et al., 1983), Hickok (Hickok & Avrutin, 1995), and Zurif (Zurif et al., 1993). Zurif et al. (1993) suggested that damage to Broca's area reduces certain processing resources that are necessary for building structures, which in turn leads to incomplete syntactic

representations in comprehension and production, an approach that is similar to other proposals (Kolk, 1995; Hagiwara, 1995; Grillo, 2008). It is not clear how these theories account for the deficits in verbal working memory and the similarity of performance for noncanonical sentence comprehension between agrammatic Broca's aphasia and conduction aphasia; however, these proposals all share the assumption that grammatical knowledge is intact, with the use of this knowledge impaired. Interestingly, Grodzinsky's (1986) original position regarding the TDH was that a disrupted processor was the root of the comprehension disorder:

> It is very likely that some kind of memory (either dedicated to language processing or not), or perhaps some sort of temporary store, which relates positions in sentences during comprehension (i.e., is essential for the execution of the coindexing algorithm necessary for chain formation), is disrupted, and the result is the comprehension deficit in agrammatism … Also, it is possible that the temporary store is crucial for other tasks during sentence comprehension, namely, not only for relating positions, but also for a different type of linking, namely, agreement … (pg. 156)

While Grodzinsky's position eventually shifted to focus specifically on syntactic operations and the STC, this earlier position is quite similar to the working memory hypothesis we provide in the next section.

Altogether, these developments force us to acknowledge that while syntactic theory provides useful descriptions for the capacities of normal and agrammatic sentence processing, agrammatism (among aphasic syndromes more generally) is not a singular behavioral profile (Badecker & Caramazza, 1985) and is not explainable via deficits in a particular grammatical module or syntactic operation. These observations have broader implications for the relation between syntactic theory and our understanding of linguistic deficits due to brain damage, as there is not a single successful case of an impaired syntactic operation or grammatical module explaining deficits in aphasia syndromes. As a consequence, the integration of research on aphasia with syntactic theory is now quite limited. By contrast, there has been substantial progress in identifying selective deficits in domains that have not been clearly tied to grammatical modules, such as word comprehension, sentence comprehension, speech production, and speech perception (Dronkers et al., 2004; Dronkers, 1996; Hickok & Poeppel, 2004; Mesulam, 2015; Thothathiri et al., 2012), illustrating that there is not a general failure of the localizationist approach to aphasia. Thus a re-evaluation of the approach to the study of aphasia from the aspect of syntactic theory is needed.

The failure of the STC is reminiscent of the (purported) failure of the derivational theory of complexity (DTC). The DTC posited a transparent relation between grammatical operations and online processing measures (Miller & Chomsky, 1963). There were many experiments testing

the DTC that represented a golden age of close interaction between psycholinguistics and syntactic theory (see Miller, 1962, Fodor et al., 1974, and Phillips, 1996 for reviews). However, there were clearly cases where the grammatical theory predicted increased processing complexity that were not borne out in experimental data, and often times the details of syntactic theory appeared to be in difficult to reconcile with a direct implementation in online sentence processing (Fodor et al., 1974). Following this, the period of close interaction between syntactic theory and psycholinguistics ended, and psycholinguists and syntacticians began to pursue separate interests.

However, as with the DTC, the failure of the STC critically depends on the model of grammar, as Fodor et al. (1974) pointed out. If the grammatical model changes, then the data must also be re-evaluated. As Phillips (1996) argues, a different model of grammar can potentially be reconciled effectively with online processing measures, and much recent research suggests that grammatical operations are in fact directly reflected in online processing (see Lewis & Phillips, 2015, for a review), indicating that the DTC may have been prematurely abandoned. It strikes us that this may also be the case for the relation between syntactic theory and aphasiology. In particular, we think that grammatical frameworks such as the Minimalist program (Chomsky, 1995; Adger, 2003; Hornstein, 2008) and tree-adjoining grammar (TAG; Joshi, 1985; Frank, 2002), which aim to reduce language-specific cognitive machinery and principles to a minimum, (Sprouse & Hornstein, 2016), are the right avenues of approach for linking syntactic theory with aphasia, and more specifically agrammatic comprehension and production. In the following section, we outline a hypothesis about how linguistic deficits in agrammatism are linked to new developments in working memory that operate over syntactic representations. If this proposal is successful, the goal for understanding the link between syntactic theory and agrammatism would then become understanding how postulates of syntactic theory relate to working memory, part of the larger goal of connecting syntactic theory to online sentence processing (Fodor et al., 1974; Phillips, 1996; Lewis & Phillips, 2015).

5 Content-addressable memory (CAM) retrieval as a possible link between syntactic theory and agrammatism

The main challenge for any working memory theory of agrammatic production and comprehension deficits is to show how the disparate and often seemingly conflicting behavioral profile of Broca's aphasia patients falls under such an account. In order to clarify this issue, we have compiled a list of deficits found in patients defined as having Broca's aphasia and agrammatic comprehension:

1. Sentence comprehension deficits:
    a. Classic agrammatic comprehension pattern: deficits on comprehension of sentences with non-canonical word order (Caramazza & Zurif, 1976; Schwartz et al., 1980)
    b. Better comprehension of object-extracted WH-phrases with bare *who* as opposed to *which*-N phrases (Hickok & Avrutin, 1996; Sheppard et al. 2015; cf. Thompson et al. 1999)
    c. Deficits on comprehension deficits of the main clause of subject-relatives (Hickok et al., 1993; Hickok & Avrutin, 1995)
    d. Comprehension deficits for locatives (Schwartz et al., 1980)
    e. Comprehension deficits for reversible active sentences (Schwartz et al., 1980)
    f. Deficits on comprehension of inflection, including case and agreement (Luria, 1975)
2. Sentence production deficits:
    a. Deficits in generating properly formed phrases/sentences given sets of words (Zurif et al., 1972; Caramazza et al., 1981)
    b. Deficits in production of closed-class words and inflectional morphology (Goodglass et al., 1972; Gleason et al., 1975)
    c. Increased difficulty for functional words in sentence onset position relative to sentence medial position (Gleason et al., 1975)
    d. Potential dissociation between production of tense (impaired) and agreement (intact) (Friedmann & Grodzinsky, 1997; 2000)
    e. Deficits in production of verbs with complex argument structure, as well as the arguments associated with these verbs (Thompson et al., 1997)
3. Acceptability judgment deficits:
    a. Acceptability judgment deficits of anaphora: reflexives, pronouns, auxiliary copying (Blumstein et al., 1983; Linebarger et al., 1983; Grodzinsky et al., 1993; Santi & Grodzinsky, 2007b; Wulfeck, 1988)
    b. Acceptability judgment deficits of number agreement (Wulfeck & Bates, 1991)
    c. Acceptability judgment deficits of movement (Grodzinsky & Finkel, 1998; Santi & Grodzinsky, 2007b)
    d. Miscellaneous “difficult” acceptability judgment deficits (Wilson & Saygin, 2004)
4. Processing differences:
    a. Slowed lexical processing (Prather et al., 1992)
    b. Improvement of sentence comprehension with slowed presentation rate (Love et al., 2008)
    c. Slowed prediction of syntactic dependencies (Zurif et al., 1993; Jakuszeit et al., 2013)

d. Verbal working memory deficits (Caspari et al., 1998; Pettigrew & Hillis, 2014)

This list is certainly not exhaustive; however, it covers a large set of data that clearly illustrate the problems with extant theories of agrammatic comprehension and production, and generalizations from this set of data suggest alternative explanations of the underlying deficits. We will shortly suggest that there are in fact two syndromes underlying "agrammatism", with distinct underlying source deficits.

It is plausible that some of these deficits derive from verbal working memory deficits. However, clearly the whole set does not follow from such deficits alone, particularly for agrammatic production. There are additional empirical problems with a verbal working memory-centric view of agrammatism. Even if impaired verbal working memory could explain the full range of sentence processing deficits in Broca's aphasia, there are patients with impaired verbal working memory but not the same linguistic deficits as in Broca's aphasia patients with agrammatic comprehension, namely, patients with conduction aphasia. The existence of conduction aphasia poses problems for both grammatical and verbal working memory theories of agrammatism. While patients with conduction aphasia show many of the same 'agrammatic' sentence comprehension patterns as patients with Broca's aphasia (Caramazza et al., 1976; Goodglass et al., 1993), and both groups of patients have phonological working memory deficits, conduction aphasia patients do *not* have agrammatic production. Their spontaneous production is grammatically near normal (Gleason et al., 1975), and they do not seem to share all of the grammatical comprehension deficits of agrammatic Broca's aphasia, such as an inability to use morphological features to aid sentence comprehension (Blumstein et al., 1983). Similarly, Caramazza et al. (1981) showed clear dissociations between a patient with conduction aphasia and a patient with Broca's aphasia who both had the classic agrammatic comprehension pattern on a task requiring subjects to freely generate a well-formed sentence given a set of words, including both content and function words. The conduction aphasia patient was able to effortfully perform the task, while the Broca's aphasia patient showed severe deficits even with strong cueing from the experimenters. The conduction aphasia patient was quite sensitive to the function of closed-class items and the grammatical status of resulting strings words while performing the task, while the Broca's patient was not.

Even if the similarities between these two groups of patients with respect to agrammatic comprehension can be explained via phonological working memory deficits, there are plenty of differences that need to be explained by something else, and this "something" appears to make reference to syntax. This holds for at least the classic production deficits in Broca's aphasia. It is also possible that some of the comprehension and acceptability judgment deficits listed above are unique to Broca's aphasia, such as problems with anaphora, agreement, and interpretation

of long-distance subject-verb agreement. However, it must be noted that the comprehension and acceptability judgment abilities of conduction aphasia have not been investigated nearly as thoroughly as Broca's aphasia; we consider more detailed testing of patients with conduction aphasia on these tasks to be of prime importance to identify any unique deficits of agrammatism that are not also present in conduction aphasia.

5.1 Splitting agrammatism in two

Since Caramazza & Zurif (1976), a main focus of the study of Broca's aphasia with respect to syntactic theory has been agrammatic comprehension. Given that this comprehension pattern is paralleled in patients with conduction aphasia who do not have agrammatic production (and presumably do not share other acceptability judgment and sentence comprehension deficits exhibited in Broca's aphasia, although this needs to be tested), it appears that this focus may have been misplaced. We propose that there are actually two distinct behavioral syndromes that often co-occur in patients with Broca's aphasia and agrammatic comprehension and production. The first syndrome is comprised of a deficit in phonological working memory that underlies the comprehension asymmetry between the canonical and noncanonical sentences - this deficit is shared with conduction aphasia. The second syndrome is comprised of deficits to a content-addressable memory (CAM) system operating over syntactic features - this deficit is absent in conduction aphasia. Our proposal is similar to Caplan & Waters (1999; 2013), who reviewed psycholinguistic and neuropsychological studies showing dissociations between basic sentence comprehension and measures of verbal working memory resources. They suggested that the verbal working memory system is relevant to language processing only for post-interpretive demands, that is, for performing particular judgments or tasks beyond the basics of sentence comprehension, and that a separate pool of language-specific working memory resources exists to support basic sentence comprehension. We suggest that both pools are often damaged in Broca's aphasia. We first discuss the shared deficit in phonological working memory that explains the comprehension asymmetry between canonical and noncanonical word order; following this, we outline the architecture of the language-specific working memory system and its application to agrammatism.

5.2 Agrammatic comprehension - verbal working memory deficits

Our explanation regarding asymmetrical comprehension deficits for reversible noncanonical compared to canonical sentences is that noncanonical sentences require verbal working memory resources that are impaired in both Broca's aphasia as well as conduction aphasia.

Why should noncanonical sentences require greater verbal working resources than canonical ones? One possibility is that the increased distance between filler and gap requires additional memory resources, which has been proposed to explain comprehension deficits in aphasia as well as patterns of data in healthy subjects (Gibson, 1998; 2000). However, this analysis does not well account for comprehension impairments on passives, as there is not a clear dependency distance distinction between active and passive sentences. Alternatively, we suggest that sentences with noncanonical word order routinely require structural revision. Central to this analysis is the hypothesis that both individuals with and without aphasia predict the structural and/or thematic roles of initial NPs during sentence comprehension. This hypothesis was a core aspect of Grodzinsky's movement proposal, which relied on an agent-first strategy in patients with Broca's aphasia (Grodzinsky, 1986), and appears to be a reasonably well-supported strategy in typical sentence processing (Bever, 1970; Lewis & Vasishth, 2005; Demberg & Keller, 2008). If so, then non-canonical sentences will routinely require revision similar to garden-path sentences, as the initial NP is predicted to be the subject. It is in cases of revision that we assume verbal working memory resources are required in order to effectively re-parse the sentence.

Regardless of whether distance or revision causes increased memory demands, there is substantial evidence that the processing of non-canonical sentences requires verbal working memory resources. Several studies have shown behavioral disruption for comprehension of object-relative sentences compared to subject-relative sentences in the presence of distracting secondary tasks that require verbal working memory resources (Fedorenko et al., 2007; Rogalsky et al., 2008). Additionally, Kush et al. (2015) performed an experiment examining the interfering effects of phonological similarity on sentence comprehension. They found that phonological similarity only affected initial encoding of words, and did not interfere with later memory retrieval during long-distance dependency resolution, suggesting that phonological information is only used in situations of re-analysis or repair, such as non-canonical or garden-path sentences. These studies predict that we should see a tight correlation between performance on verbal working memory tasks and comprehension of sentences with non-canonical word order or garden-paths, which is the observed pattern across studies of patients with Broca's and conduction aphasia (Caramazza & Zurif, 1976; Pettigrew & Hillis, 2014; Friedmann & Gvion, 2003).

Why are verbal working memory resources damaged in both Broca's and conduction aphasia? Neuroimaging research suggests that the verbal working memory system is distributed across frontal, parietal, and temporal cortex, allowing for distinct lesions to disrupt this system. As discussed in Section 2, patients with conduction aphasia most frequently have left hemisphere damage left hemisphere damage affecting area Spt, a region frequently implicated in

phonological working memory by both functional neuroimaging and lesion studies (Hickok et al., 2003; Buchsbaum et al. 2011; Rogalsky et al. 2015b). Broca's aphasia is associated with large perisylvian lesions that typically include the pars opercularis of Broca's area[5]. Broca's area is thought to be involved in phonological processing in general (Hagoort, 2005) and specifically in verbal working memory, in conjunction with area Spt (Hickok et al., 2003). Functional neuroimaging experiments have shown that the posterior portion of Broca's area, the pars opercularis, shows increased activity to object-relative sentences compared to subject-relative sentences, in support of this view (Stromswold et al., 1996; Just et al., 1996), and brain activation for syntactic complexity overlaps well with activation related to verbal working memory demands (Stowe et al., 2005; Rogalsky et al., 2008). Thus, while Broca's aphasia and conduction aphasia are distinct syndromes with distinct lesion patterns, they both involve damage to the distributed verbal working memory system.

Differences between Broca's aphasia and conduction aphasia arise for tasks that do not tax phonological working memory capacity but rather target the CAM system operating over syntactic representations, which we posit is impaired in patients with agrammatic production, but not necessarily impaired in all patients with agrammatic comprehension (i.e., conduction aphasia).

5.3 Deficits in canonical sentence comprehension, acceptability judgment deficits, and agrammatic production - impaired syntactic content-addressable memory

The second syndrome of agrammatism is comprised of deficits due to damage to language-specific working memory (Caplan & Waters, 1999). Recent behavioral research suggests that the working memory system underlying core aspects of sentence comprehension is a content-addressable memory (CAM) retrieval system operating over syntactic features (McElree et al., 2003; Lewis & Vasishth, 2005; Lewis et al., 2006; Caplan & Waters, 2013). It is damage to this system that we believe underlies the classic syndrome of agrammatism that separates Broca's aphasia from conduction aphasia - Broca's aphasia often includes damage to both verbal and language-specific working memory resources. The anterior portion of Broca's area (the pars triangularis) is more frequently associated with syntax and morphology in neuroimaging studies (Pallier et al., 2011; Nelson et al., 2017; Sahin et al., 2009; Moro et al., 2001). We posit here that the pars triangularis is the location of this CAM operating over syntactic features. Our proposal is similar to Fiebach et al., (2005), who suggested that the function of Broca's area is in

[5] The extent to which damage to the anterior portions of Broca's area, namely BA45, or to the white matter connecting this region to other areas, is sufficient to cause Broca's aphasia or agrammatic symptoms generally is an important issue to resolve with respect to our hypotheses here.

syntactic working memory rather than structure-building operations *per se*. Wilson et al. (2010; 2011) showed that in primary progressive aphasia, damage to both gray matter in the IFG as well as dorsal white matter pathways connecting the IFG to the temporal lobe contributed to both agrammatic production and comprehension. Therefore, we posit that impairments to CAM can result from direct damage to the pars triangularis or disconnection of this region from the temporal lobe.

The two subregions of Broca's area, the pars opercularis and the pars triangularis, are adjacent and are both supplied by the superior system of the middle cerebral artery. This means that both cortical regions are likely affected by the same stroke or degenerative disorder, resulting in deficits in both verbal working memory and syntactic CAM. Consistent with this idea, fMRI studies by Fedorenko et al. (2012) and Rogalsky et al. (2015a) indicate that there are subregions of Broca's area (mostly in pars triangularis) that are specific to sentences (and do not activate during verbal working memory tasks) adjacent to subregions (mostly in pars opercularis) that activate to both sentences and verbal working memory tasks. Thus, we posit that Broca's aphasia, often resulting from a stroke of the MCA, typically involves damage to (or disconnection of) two distinct systems.

It is beyond the scope of this chapter to review the evidence motivating CAM or to detail all the properties of this system (see Lewis & Vasishth, 2005, Lewis et al., 2006, and Caplan & Waters, 2013), or to provide an exhaustive account of the many data points concerning Broca's aphasia that are explained by appeal to CAM. However, our goal is to illustrate the promise of understanding aphasia from the vantage point of CAM, and to underscore the importance of relating syntactic theory to such a memory retrieval system.

CAM differs substantially from the prominent verbal working memory storage model originally proposed by Baddeley & Hitch (1974). The core differences between these models are the format of the representations in memory and how information is stored, maintained and accessed. In the Baddeley & Hitch model, information relevant to sentence processing is stored in a verbal code in a short-term memory buffer. This allows it to be rehearsed and maintained by subvocal articulation, and limits the representational format of memory items for sentence processing to phonological representations; linguistic distinctions, e.g. for syntactic category or phi features, cannot be made. By contrast, in the CAM model of sentence processing developed by McElree et al. (2003) and Lewis et al. (2006), memory retrieval access operates over syntactic features stored in long-term memory. Lewis & Vasishth (2005) and Lewis et al. (2006) posit that syntactic representations in memory consist of phrasal maximal projections comprised of bundles of features; features include structural relations (e.g., head, complement, specifier), agreement and case (Figure 3). In this model, information is not simply rehearsed

and maintained in the serial order it is presented, as in the Baddeley model. Rather, representation chunks in long-term memory are activated by the perception of words. Long distance dependencies are established when retrieval cues of the dependent element initiate a parallel access search for matching features in memory. For instance, a verb with agreement morphology such as singular would trigger a search for singular nouns. The key property of the CAM model is that syntactic representations are the targets of memory retrieval access; damage to this retrieval system can thereby be expected to impair the use of syntactic representations, which we posit underlies the classical agrammatic production syndrome.

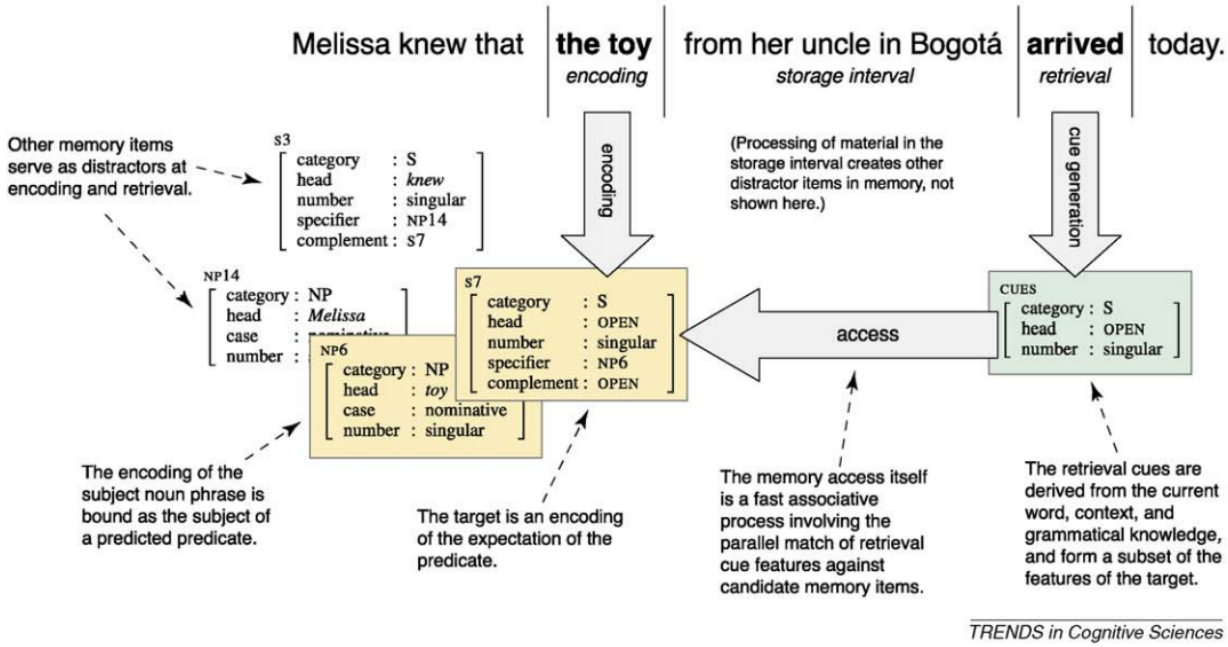

Figure 3 (from Lewis et al., 2006). An illustration of how syntactic content-addressable memory operates during sentence processing. See Lewis et al. (2006) for details and explication.

There are relatively few modern theories of agrammatic production. One theory (Kolk, 1995) assumes that these production deficits result from slowed processing that results in morphological errors, particularly for complex syntactic structures. The theory of Grillo (2009) extends this notion to suggest that functional elements at the ends of the computational domains of phases (DP, vP, CP) will be most impaired by slowed processing. However, these proposals do not satisfactorily address the fact that patients with Broca's aphasia can make acceptability judgments on sentences that appear to require intact access to these features, nor do these theories well explain the full range of comprehension and acceptability judgment deficits observed in Broca's aphasia.

By contrast, the CAM approach that we advocate here posits that these problems derive from an inability of these patients to perform top-down access (i.e., to access these features in the

absence of perceptual input) of words/morphemes that lack rich conceptual-semantic features during production and certain contexts of sentence comprehension. During standard comprehension of local dependencies, the relevant words and morphemes are activated by sensory input. However, during sentence production and for the computation of long-distance dependences in comprehension, perceptual input is not available to activate these items - thus, they must be accessed through top-down processing mechanisms[6]. We suggest that the forms of content words can be accessed through conceptual-semantic features associated with these words. Function words and morphemes, on the other hand, lack this rich content, and thus *must* be accessed based on syntactic features. For instance, we posit that the word "the" is retrieved by top-down activation of abstract syntactic features such as "+singular" and "+Determiner". However, damage to a system that uses syntactic features to retrieve word forms will leave a patient without the ability to activate functional words and morphemes in the absence of perceptual input. This explains the dramatic dissociation between content and function words/morphemes, and comports well with meta-linguistic reports of patients with Broca's aphasia that function words lack meaning, while content words have rich meaning that makes them easy to use.[7]

The deficits seen across comprehension and acceptability judgment studies on anaphora, including pronouns, reflexives, and auxiliary copying (Linebarger et al., 1983; Grodzinsky, 1993) are directly interpreted as problems in accessing the antecedent via CAM. We suggest that syntactic representations are activated during sentence comprehension, including the syntactic features of the retrieval cues. However, the deficit arises from an inability of these retrieval cues to access features in memory, preventing the re-activation of the memory chunks and impairing comprehension of these long-distance dependencies. This extends to general comprehension or acceptability judgments deficits linked to inflection, including tense, case, and agreement (Luria, 1975; Linebarger et al., 1983; Grodzinsky et al., 1993), as the subject must access a syntactic form previously encountered in order to assess the appropriateness of the presented form.

Deficits in CAM can also potentially explain processing speed differences between patients with Broca's aphasia and normal subjects. If we assume that top-down memory retrieval access can operate both in forward and in reverse, then CAM can be used to predictively activate future

[6] It is possible that there is sensory input during production that can help trigger the activation of grammatical representations, given that subjects perceive their own utterances. This might explain the reported dissociation between production of functional items at the beginning of sentences (severely impaired) and the middle of sentences (less impaired) (Gleason et al., 1975). It may also address the reported intact production of agreement in Broca's aphasia (Friedmann & Grodzinsky, 1997; 2000), as these studies cued subjects with a sentence prompt and local subject-verb agreement.

[7] E.g., http://www.aphasiathemovie.com/Aphasia_Project/Carl_in_the_Classroom_2009.html.

linguistic material, which presumably speeds up processing. Damage to the memory system thus predicts slower processing of syntactic information (Zurif et al., 1993; Jakuszeit et al., 2013), and slowing the presentation of linguistic material should facilitate comprehension when predictive resources are not available (Love et al., 2008).

There are plenty of open questions here, and we have not attempted to exhaustively describe how damage to syntactic CAM apply to the list of deficits attributed to Broca's aphasia above. At least some of the salient deficits in agrammatism not clearly explained by verbal working memory problems can be coherently addressed through the CAM framework, particularly agrammatic production.

5.4 Incorporating syntactic theory into CAM

One of the strengths of the present proposal is its use of an independently proposed memory/ processing architecture. This architecture is fully computationally implemented and is motivated from robust observations from psycholinguistic sentence processing experiments. Thus, approaching agrammatic comprehension and production in Broca's aphasia from the aspect of CAM furthers the goal of an interdisciplinary approach to the study of language. Assuming that the linguistic deficits of agrammatism not covered by verbal working memory are successfully explained by the proposed deficits to the CAM architecture, then where does that leave the connection between syntactic theory and aphasia? An important point here is that our review highlights the general failure of the STC. This suggests that syntactic theories that propose elaborate grammatical machinery have not been successful in explaining patterns of language deficits in aphasia. Alternatively, theories which aim to reduce the degree of domain-specific operations and principles to a minimum, such as Chomsky's Minimalist Program (1995) and Tree-adjoining Grammar (Joshi, 1985; Frank, 2002) are preferred as they do not suggest large portions of cortex dedicated to grammatical processing that are vulnerable to brain damage. At present, it is unclear to us how syntactic theories of this sort might relate to CAM, and it is beyond the scope of our review to present and explore hypotheses of this relation. However, we point out that because of the increasing success of CAM as an explanation for various linguistic and psycholinguistic phenomena, our analysis here with respect to aphasia provides additional motivation for establishing links between syntactic theories and the CAM architecture. The challenge changes from connecting syntactic theory directly to behavior (including aphasia), but rather connecting syntactic theory to CAM. This is part of a broader question of how syntactic theory connects to real-time sentence comprehension (Fodor et al., 1974; Berwick & Weinberg, 1984; Phillips, 1996; Lewis & Phillips,

2015), which we believe is critical for there to be effective interaction between syntactic theory and aphasia research.

6 The future of experimental syntax and aphasiology

In order for aphasia research and syntactic theory to mutually benefit each other, we suggest addressing two specific issues that would allow them to potentially line up better with aphasia data.

1. Connecting syntactic theory with real-time sentence processing

Language assessment tasks in aphasia research require patients to process sentences using their grammatical knowledge, non-grammatical processing resources like verbal working memory, and cognitive systems unrelated to language that are required to make task responses. Thus, observed differences between patients and healthy subjects may lie at any of these levels. In order to evaluate the relevance of syntactic theory to aphasia, it is important to have clear ideas about how grammatical operations and principles relate to real-time processing and the tasks patients are asked to perform so that the effects of these various components can be clearly separated. This is particularly important as syntactic operations are often formulated in ways to that do not translate transparently to real-time sentence processing, such as bottom-up syntactic derivations.

2. Aligning the granularity of linguistics and aphasiology at the level of the cortical area

The currency of aphasia is the brain area - some piece of tissue that can be damaged due to stroke, neurodegenerative disease, or other brain injury and can be quantified and compared across subjects. This means that aligning syntactic theory with aphasiology requires us to identify a level of linguistic granularity that aligns with relatively large-scale neural organization (see Poeppel & Embick, 2005 and Embick & Poeppel, 2015 for discussion of this general point with respect to linguistics and neuroscience). It may be that grammatical operations do not correspond to that level of granularity - for instance, they might correspond to micro-level neural circuitry or even sub-cellular chemical properties (Gallistel & King, 2011). We believe it is helpful to identify what properties of language *do* match up well with the cortical area or a network of areas. There are successful cases of functional localization in the visual domain that can inform this search, such as the fusiform face area (Kanwisher, 2010) and the visual word form area (Dehaene & Cohen, 2011). If these successful cases of functional localization are to be a model for syntax, then we should identify generalizations stemming from syntactic theory

or psycholinguistics that can be examined in a similar fashion. This is not to say that we should forgo investigating how the brain instantiates basic grammatical operations, but rather that we should make progress in understanding what we can about the brain and syntax given the methods currently available to us. Our hope is that this will form a useful precursor to future investigations, in which hypotheses about the neural implementation of grammatical operations can be formulated and tested using finer-grained methods, as is starting to be the case (Ding et al., 2016; Nelson et al., 2017).

Annotated References

Caramazza & Zurif, 1976

This study was the first to clearly demonstrate sentence comprehension deficits in Broca's aphasia, namely the contrast between reversible (worse performance) and non-reversible (better performance) object-relative sentences. This paper is marked as the origin of the notion of that a central syntactic deficit underlies agrammatism. In addition, a lesser-known observation of this study was that the conduction aphasia patients (without classic production agrammatism) showed the same comprehension profile as the Broca's aphasia patients, a fact that we think has gotten not nearly enough attention in the aphasia literature.

Caplan & Hildebrandt, 1988

A systematic review of agrammatism in light of syntactic theory, psycholinguistics, and computational parsing theory, one that is quite rare today.

Caplan & Waters, 1999

This study presented the clearest case for two separate pools of working memory resources: domain-general and language-specific, and that these two pools can dissociate.

Fedorenko et al., 2012

This study clearly demonstrated that there are distinct subregions within Broca's area: one region that responds to both sentences and a variety of cognitively demanding tasks (including verbal working memory), and one region that responds to sentences but not do these demanding tasks. These distinct subregions may map onto the distinct pools of resources hypothesized by Caplan & Waters (1999).

Grodzinsky, 1986

This paper was the first to lay out the logic for and evidence behind the influential TDH, the idea that problems with syntactic movement underlie agrammatic comprehension in Broca's aphasia, and the association between Broca's area and syntactic movement.

Lewis & Vasishth, 2005

This paper introduces the CAM model in computational detail, as well as reviewing conceptual and empirical evidence for this model of memory. It marks a departure in psycholinguistic from the dominance of a verbal working memory storage model to a model of memory based on limited storage and re-activation of items in long-term memory. The model makes explicit that the memory representations and retrieval cues are syntactic features, which opens the path for a connection to language-specific deficits in aphasia and agrammatism more specifically.

Lewis & Phillips, 2014

This paper discusses the relation between the grammar (from the perspective of syntactic theory) and real-time sentence comprehension and production, reviewing experimental evidence bearing on this issue and arguing in favor of a one-system view in which the grammar is directly implemented in online processing.

Linebarger et al., 1983

This paper clearly demonstrated the asymmetry between agrammatic sentence comprehension in Broca's aphasia and the capacity for these patients to make subtle acceptability judgments. This paper is widely cited, yet is not cited enough, regarding claims about the function of Broca's area, Broca's aphasia, and syntactic processing. It remains one of the most important desiderata for any theory to address.

Mohr et al. (1978)

This paper examined several cases of Broca's aphasia and demonstrated that damage to Broca's area is neither necessary nor sufficient to cause Broca's aphasia. This paper is important because it means that we cannot assume that a patient with agrammatic Broca's aphasia has a lesion to Broca's area, and many papers on agrammatism have made that assumption.

Poeppel & Embick, 2005

This paper clearly laid out the serious challenges of integrating linguistics with cognitive neuroscience, challenges that we think have been under-appreciated by linguists looking to bridge these gaps.

Stromswold et al., 1996

This paper was one of the first and clearest papers to demonstrate that sentences with noncanonical word order activate the posterior portion of Broca's area (pars opercularis) more than sentences with canonical word order.

Wilson & Saygin, 2004

This study represents a careful and systematic assessment of the TDH and the role of Broca's area in syntax more generally. It clearly showed that while Broca's area damage appears somewhat related to sentence processing, it is not more or less involved in Movement than sentences without Movement, and there are other brain areas that are much strongly related to syntactic knowledge, namely posterior temporal areas. This study has been very under-cited, in our view, in discussions of syntactic theory and aphasia.

Schwartz et al. 1980

This study found that patients with agrammatic Broca's aphasia often struggle with sentences that do not involve Movement: reversible active and locative sentences. In our view, there has never been a serious attempt to account for these data.

References

Adger, D. (2003). *Core syntax: A minimalist approach* (Vol. 33). Oxford: Oxford University Press.

Anwander, A., Tittgemeyer, M., von Cramon, D. Y., Friederici, A. D., & Knosche, T. R. (2007). Connectivity-based parcellation of Broca!s area. *Cerebral Cortex, 17*, 816–825.

Avrutin, S. (2001). Linguistics and agrammatism. *Glot International*, *5*(3), 1-11.

Baddeley, A. D., & Hitch, G. (1974). Working memory. *Psychology of learning and motivation*, *8*, 47-89.

Baddeley, A.D., Thomson, N. & Buchanan, M. (1975). Word length and the structure of short-term memory. *Journal of Verbal Learning and Verbal Behavior, 14,* 575-589.

Badecker, W., & Caramazza, A. (1985). On considerations of method and theory governing the use of clinical categories in neurolinguistics and cognitive neuropsychology: The case against agrammatism. *Cognition*, *20*(2), 97-125.

Badecker, W., & Kuminiak, F. (2007). Morphology, agreement and working memory retrieval in sentence production: Evidence from gender and case in Slovak. *Journal of Memory and Language*, *56*(1), 65-85.

Baldo, J.V., Klostermann, E.C., Dronkers, N.F. (2008). It's either a cook or a baker: patients with conduction aphasia get the gist but lose the trace. *Brain and Language, 105*(2), 134-140.

Bartha, L. & Benke, T. (2003). Acute conduction aphasia: an analysis of 20 cases. *Brain and Language, 85*, 93–108.

Bastiaanse, R., & Van Zonneveld, R. (1998). On the relation between verb inflection and verb position in Dutch agrammatic aphasics. *Brain and Language*, *64*(2), 165-181.

Bates, E., Wilson, S.M., Saygin, A.P., Dick, F., Sereno, M.I., Knight, R.T. & Dronkers, N.F. (2003). Voxel-based lesion-symptom mapping. *Nature Neuroscience, 6*(5), 448-50.

Ben-Shachar, M., Hendler, T., Kahn, I., Ben-Bashat, D., & Grodzinsky, Y. (2003). The neural reality of syntactic transformations: Evidence from functional magnetic resonance imaging. *Psychological science*, *14*(5), 433-440.

Berndt, R. S., & Caramazza, A. (1980). A redefinition of the syndrome of Broca's aphasia: Implications for a neuropsychological model of language. *Applied psycholinguistics*, *1*(3), 225-278.

Berwick, R. C., & Weinberg, A. S. (1986). *The grammatical basis of linguistic performance: Language use and acquisition*. MIT press.

Bever, T. G. (1970). The cognitive basis for linguistic structures. *Cognition and the development of language*.

Binder, J.R., Rao, S.M., Hammeke, T.A., Yetkin, F.Z., Jesmanowicz, A., Bandetti, P.A., et al. (1994). Functional magnetic resonance imaging of human auditory cortex. *Annuals of Neurology, 35*(6), 662-672,

Blumstein, S. E., Goodglass, H., Statlender, S., & Biber, C. (1983). Comprehension strategies determining reference in aphasia: A study of reflexivization. *Brain and Language*, *18*(1), 115-127.

Bresnan, J. (2001). Lexical-Functional Syntax. Blackwell Publishers, Oxford.

Brodmann, K. (1909). Vergleichende Lokalisationslehre der Grobhirnrinde in ihren Prinzipien dargestellt aufgrund des Zellenbaues. Leipzig: Johann Ambrosius Barth Verlag.

Brownsett, S.L.E., Warren, J.E., Geranmayeh, F., Woodland, Z., Leech, R. & Wise, R.J.S. (2014). Cognitive control and its impact on recovery from aphasic stroke. *Brain, 137*(1), 242-254.

Buchsbaum, B.R., Baldo, J., Okada, K., Berman, K.F., Dronkers, N., D'Esposito, M. & Hickok, G. (2011). Conduction aphasia, sensory-motor integration, and phonological short-term memory-an aggregate analysis of lesion and fMRI data. *Brain and Language, 119*(3), 119-128.

Burton, S., & Grimshaw, J. (1992). Coordination and VP-internal subjects. *Linguistic Inquiry*, 305-313.

Butterworth, B., Campbell, R. & Howard, D. (1986). The uses of short-term memory: A case study
Quarterly Journal of Experimental Psychology, 38A, 705-737.

Caplan, D., DeDe, G. & Michaud, J. (2006). Task-independent and task-specific syntactic deficits in aphasic comprehension. *Aphasiology, 20,* 893-920.

Caplan, D., & Hildebrandt, N. (1988). *Disorders of syntactic comprehension*. MIT Press.

Caplan, D., Hildebrandt, N., & Makris, N. (1996). Location of lesions in stroke patients with deficits in syntactic processing in sentence comprehension. *Brain*, *119*(3), 933-949.

Caplan, D., Michaud, J. & Hufford, R. (2013). Dissociations and associations of performance in syntactic comprehension in aphasia and their implications for the nature of aphasic deficits. *Brain and Language 127*(1), 21-33.

Caplan, D., Michaud, J., Hufford, R. & Makris, N. (2016). Deficit-lesion correlations in syntactic comprehension in aphasia. *Brain and Language, 152*, 14-27.

Caplan, D., & Waters, G. S. (1999). Verbal working memory and sentence comprehension. *Behavioral and brain Sciences*, *22*(1), 77-94.

Caplan, D., & Waters, G. (2013). Memory mechanisms supporting syntactic comprehension. *Psychonomic bulletin & review*, *20*(2), 243.

Caplan, D., Waters, G., Kennedy, D., Alpert, N., Makris, N., Dede, G., … Reddy, A. (2007). A study of syntactic processing in aphasia II: Neurological aspects. *Brain and Language, 101*, 151–177.

Caramazza, A., & Zurif, E. B. (1976). Dissociation of algorithmic and heuristic processes in language comprehension: Evidence from aphasia. *Brain and language*, *3*(4), 572-582.

Caramazza, A., Basili, A. G., Koller, J. J., & Berndt, R. S. (1981). An investigation of repetition and language processing in a case of conduction aphasia. *Brain and language*, *14*(2), 235-271.

Caspari, I., Parkinson, S. R., LaPointe, L. L., & Katz, R. C. (1998). Working memory and aphasia. *Brain and cognition*, *37*(2), 205-223.

Chomsky, N. (2014). *Aspects of the Theory of Syntax* (Vol. 11). MIT press.

Chomsky, N. (1993). *Lectures on government and binding: The Pisa lectures* (No. 9). Walter de Gruyter.

Chomsky, N. (2014). *The minimalist program*. MIT press.

Chomsky, N. (1999). *Derivation by phase* (No. 18). MIT, Department of Linguistics.

Cinque, G. (1990). Types of A'-dependencies. Cambridge, MA: MIT Press.

Crain, S., & Fodor, J. D. (1985). How can grammars help parsers. In D. Dowty, D. Kartunnen, & A. M. Zwicky (Eds.), *Natural language parsing: Psycholinguistics, computational, and theoretical perspectives* (pp. 94–129). Cambridge University Press.

Crain, S., & Nakayama, M. (1987). Structure dependence in grammar formation. *Language*, 522-543.

Cupples, L. & Inglis, A.L. (1993). When task demands induce “asyntactic” sentence comprehension: a study of sentence interpretation in aphasia. *Cognitive Neuropsychology, 10*, 201-234.

Damasio, H., & Damasio, A. R. (1980). The anatomical basis of conduction aphasia. *Brain*, *103*(2), 337-350.

Damasio, A. R. (1992). Aphasia. *New England Journal of Medicine*, *326*(8), 531-539.

Daneman, M. & Newson, M. (1992). Assessing the importance of subvocalization during normal silent reading. *Reading and Writing, 4(*1), 55-77.

Demberg, V. & Keller, F. (2008). Data from eye-tracking corpora as evidence for theories of syntactic processing complexity. *Cognition, 109*(2),193-210.

Dick, F., Bates, E., Wulfeck, B., Utman, J. A., Dronkers, N., & Gernsbacher, M. A. (2001). Language deficits, localization, and grammar: Evidence for a distributive model of language breakdown in aphasic patients and neurologically intact individuals. *Psychological review*, *108*(4), 759.

Dronkers, N. F., Wilkins, D. P., Van Valin, R. D., Redfern, B. B., & Jaeger, J. J. (2004). Lesion analysis of the brain areas involved in language comprehension. *Cognition*, *92*(1), 145-177.

Embick, D., & Poeppel, D. (2015). Towards a computational (ist) neurobiology of language: correlational, integrated and explanatory neurolinguistics. *Language, cognition and neuroscience*, *30*(4), 357-366.

Erickson, R.J., Goldinger, S.D. & LaPointe, L.L. (1996). Auditory vigilance in aphasic individuals: detecting nonlinguistic stimuli with full or divided attention. *Brain and Cognition, 30*(2), 244-253.

Fedorenko, E., Gibson, E., & Rohde, D. (2007). The nature of working memory in linguistic, arithmetic and spatial integration processes. *Journal of Memory and Language*, *56*(2), 246-269.

Fedorenko, E., Duncan, J., & Kanwisher, N. (2012). Language-selective and domain-general regions lie side by side within Broca's area. *Current Biology*, *22*(21), 2059-2062.

Fegen, D., Buchsbaum, B.R. & D'Esposito, M. (2015). The effect of rehearsal rate and memory load on verbal working memory. *Neuroimage, 105*, 120-131.

Fiebach, C., Schlesewsky, M., Lohmann, G., Cramon, D. V., & Friederici, A. (2005). Revisiting the role of Broca's area in sentence processing: Syntactic integration versus syntactic working memory. *Human Brain Mapping, 24*(2), 79-91. doi:10.1002/hbm.20070

Fisher, M., Prichard, J.W. & Warach, S. (1995). New magnetic resonance techniques for acute ischemic stroke. *Journal of the American Medical Association, 274*(11), 908-11.

Fodor, J., Bever, A., & Garrett, T. G. (1974). The psychology of language: An introduction to psycholinguistics and generative grammar.

Frank, R. (2004). *Phrase structure composition and syntactic dependencies* (Vol. 38). Mit Press.

Frazier, L., & Flores D'Arcais, G. B. (1989). Filler driven parsing: A study of gap filling in Dutch. *Journal of Memory and Language*, 28(3), 331–344.

Fridriksson, J., Bonilha, L. & Rorden, C. (2007). Severe Broca's aphasia without Broca's area damage. *Behavioral Neurology, 1*8(4), 237-238.

Fridriksson, J,, Fillmore, P., Guo, D, & Rorden, C. (2015). Chronic Broca's aphasia is caused byi damage to Broca's and Wernicke's areas. *Cerebral Cortex, 25*(12), 4689-96.

Fridriksson, J., Kjartansson O., Morgan, P.S., Hjaltason, H., Magnusdottir, S., Bonilha, L., & Rorden, C. (2010). Impaired speech repetition and left parietal lobe damage. *Journal of Neuroscience, 30*(33), 11057-11061.

Friedmann, N., & Gvion, A. (2003). Sentence comprehension and working memory limitation in aphasia: A dissociation between semantic-syntactic and phonological reactivation. *Brain and Language*, *86*(1), 23-39.

Friedmann, N. A., & Grodzinsky, Y. (1997). Tense and agreement in agrammatic production: Pruning the syntactic tree. *Brain and language*, *56*(3), 397-425.

Friedmann, N. A., & Grodzinsky, Y. (2000). Split inflection in neurolinguistics. *The acquisition of syntax: Studies in comparative developmental linguistics*, 84-104.

Gallistel, C. R., & King, A. P. (2011). *Memory and the computational brain: Why cognitive science will transform neuroscience* (Vol. 6). John Wiley & Sons.

Garraffa, M., & Grillo, N. (2008). Canonicity effects as grammatical phenomena. *Journal of neurolinguistics*, *21*(2), 177-197.

Geranmayeh, F., Brownsett, S.L.E. & Wise, R.J.S. (2014). Task-induced brain activity in aphasic stroke patients: what is driving recovery? Brain 137(10), 2632-2648. PMID: 24974382

Geschwind, N. (1965). Disconnexion syndromes in animals and man. *Brain*, *88*(3), 585-585.

Gleason, J. B., Goodglass, H., Green, E., Ackerman, N., & Hyde, M. R. (1975). The retrieval of syntax in Broca's aphasia. *Brain and Language*, *2*, 451-471.

Goodglass, H. (1968). Studies in the grammar of aphasics. In S. Rosenberg & J. Koplin (Eds.), Developments in applied psycholinguistic research. New York: MacMillan.

Goodglass, H. (1976). Agrammatism. In H. Whitaker & H. A. Whitaker (Eds.), Studies in neurolinguistics (Vol. 1). New York: Academic Press.

Goodglass H. (1992). Diagnosis of conduction aphasia. In: Kohn SE, editor. Conduction aphasia. Hillsdale, N.J: Lawrence Erlbaum Associates; pp. 39–49.

Goodglass, H., Gleason, J. B., Bernholtz, N. A., & Hyde, M. R. (1972). Some linguistic structures in the speech of a Broca's aphasic. *Cortex*, *8*(2), 191-212.

Goodglass, H. & Kaplan, E. (1983). The Assessment of Aphasia and Related Disorders (2nd ed.). Philadelphia: Lea & Febiger.

Gordon, J.K. (1998). The fluency dimension in aphasia. *Aphasiology, 12*(7-8), 673-688.

Gorno-Tempini, M. L., Hillis, A. E., Weintraub, S., Kertesz, A., Mendez, M., Cappa, S. E. E. A., ... & Manes, F. (2011). Classification of primary progressive aphasia and its variants. *Neurology*, *76*(11), 1006-1014.

Grillo, N. (2008). *Generalized minimality: Syntactic underspecification in Broca's aphasia*. Doctoral Dissertation, University of Utrecht. LOT.

Goldberg, A. E. (1995). *Constructions: A construction grammar approach to argument structure*. University of Chicago Press.

Grodzinsky, Y. (1986). Language deficits and the theory of syntax. *Brain and language*, *27*(1), 135-159.

Grodzinsky, Y., Wexler, K., Chien, Y. C., Marakovitz, S., & Solomon, J. (1993). The breakdown of binding relations. *Brain and Language*, *45*(3), 396-422.

Grodzinsky, Y. (2000). The neurology of syntax: Language use without Broca's area. *Behavioral and brain sciences*, *23*(1), 1-21.

Grodzinsky, Y., & Finkel, L. (1998). The neurology of empty categories: Aphasics' failure to detect ungrammaticality. *Journal of cognitive neuroscience*, *10*(2), 281-292.

Grodzinsky, Y. (2006). A blueprint for a brain map of syntax. *Broca's region*, 83-107.

Grodzinsky, Y., & Friederici, A. D. (2006). Neuroimaging of syntax and syntactic processing. *Current opinion in neurobiology*, *16*(2), 240-246.

Grodzinsky, Y., Pinango, M. M., Zurif, E., & Drai, D. (1999). The critical role of group studies in neuropsychology: Comprehension regularities in Broca's aphasia. *Brain and language*, *67*(2), 134-147.

Gutman, R., DeDe, G., Michaud, J., Liu, J. & Caplan, D. (2010). Rasch models of aphasic performance on syntactic comprehension. *Cognitive Neuropsychology, 27*(3), 230-244.

Gvion, A., & Friedmann, N. (2012). Does phonological working memory impairment affect sentence comprehension? A study of conduction aphasia. *Aphasiology*, *26*(3-4), 494-535.

Hagiwara, H. (1995). The breakdown of functional categories and the economy of derivation. *Brain and Language*, *50*(1), 92-116.

Hagoort, P. (2005). On Broca, brain, and binding: a new framework. *Trends in cognitive sciences*, *9*(9), 416-423.

Heilman, K. M., & Scholes, R. J. (1976). The nature of comprehension errors in Broca's, conduction and Wernicke's aphasics. *Cortex*, *12*(3), 258-265.

Hickok, G., Zurif, E., & Canseco-gonzalez, E. (1993). Structural description of agrammatic comprehension. *Brain and Language*, *45*(3), 371-395.

Hickok, G., & Avrutin, S. (1995). Representation, referentiality, and processing in agrammatic comprehension: Two case studies. *Brain and Language*, *50*(1), 10-26.

Hickok, G., & Avrutin, S. (1996). Comprehension of wh-questions in two Broca's aphasics. *Brain and language*, *52*(2), 314-327.

Hickok, G., Buchsbaum, B., Humphries, C., & Muftuler, T. (2003). Auditory–motor interaction revealed by fMRI: speech, music, and working memory in area Spt. *Journal of Cognitive Neuroscience*, *15*(5), 673-682.

Hickok, G., Okada, K., Barr, W., Pa, J., Rogalsky, C., Donnelly, K., Barde, L., & Grant, A. (2008). Bilateral capacity for speech sound processing in auditory comprehension: evidence from Wada procedures. Brain and Language, 107(3), 179-84.

Hickok, G. & Poeppel, D. (2007). The cortical organization of speech processing. *Nature Reviews Neuroscience, 8*, 393-402.

Hillis, A.E. & Heidler, J. (2002). Mechanisms of early aphasia recovery. *Aphasiology, 16* (9), 885-895.

Hornstein, N. (2009). *A theory of syntax*. Cambridge: CUP.

Isenberg, A.L., Vaden, K.I. Jr., Saberi, K., Muftuler, L.T. & Hickok, G. (2012). Functionally distinct regions for spatial processing and sensory motor integration in the planum temporale. *Human Brain Mapping, 33*(10), 2453-2463.

Jakobson, R. (1956). *Two aspects of language and two types of aphasic disturbances*. In R. Jakobson and M. Halle (Eds.), Fundamentals of Language. The Hague: Houton.

Jakuszeit, M., Kotz, S. A., & Hasting, A. S. (2013). Generating predictions: Lesion evidence on the role of left inferior frontal cortex in rapid syntactic analysis. *Cortex*, *49*(10), 2861-2874.

Jarso, S., Li, M., Faria, A., Davis, C., Leigh, R., Sebastian, R., Tsapkini, K., Mori, S. & Hillis, A.E.. (2013). Distinct mechanisms and timing of language recovery after stroke. *Cognitive Neuropsychology, 30*(7-8), 454-475.

Joshi, A. K. (1985). How much context-sensitivity is required to provide reasonable structural descriptions: tree adjoining grammars. In David Dowty, Lauri Karttunen, and Arnold Zwicky,

(Eds), *Natural Language Parsing: Psychological, Computational and Theoretical Perspectives*. Cambridge University Press, Cambridge, 206–250.

Kean, M. L. (1977). The linguistic interpretation of aphasic syndromes: Agrammatism in Broca's aphasia, an example. *Cognition*, *5*(1), 9-46.

Kertesz, A. (2007). *The Western Aphasia Battery-Revised*. New York: Grune & Stratton.

Kertesz, A. & McCabe, P. (1977). Recovery patterns and prognosis in aphasia. *Brain, 100* Pt 1, 1-18.

Kitigawa, Y. (1986). Subjects in Japanese and English. Unpublished Ph.D dissertation. University of Massachusetts, Amherst, MA.

Kolk, H. (1995). A time-based approach to agrammatic production. *Brain and language*, *50*(3), 282-303.

Kush, D., Johns, C. L., & Van Dyke, J. A. (2015). Identifying the role of phonology in sentence-level reading. *Journal of Memory and Language*, *79*, 18-29.

Lazar, R.M. & Antoniello, D. (2008). Variability in recovery from aphasia. *Current Neurology and Neuroscience Reports, 8*(6), 497-502.

Lazar, R.M., Speizer, A.E., Festa, J.R., Krakeur, J.W. & Marshall, R.S. (2008). Variability in language recovery after first-time stroke. *Journal of Neurology Neurosurgery & Psychiatry, 79*(5), 530-534.

Lewis, R. L., & Vasishth, S. (2005). An activation-based model of sentence processing as skilled memory retrieval. *Cognitive science*, *29*(3), 375-419.

Lewis, S., & Phillips, C. (2015). Aligning grammatical theories and language processing models. *Journal of Psycholinguistic Research*, *44*(1), 27.

Lichtheim, L. (1885). On aphasia. *Brain, 7,* 433–484.

Linebarger, M. C., Schwartz, M. F., & Saffran, E. M. (1983). Sensitivity to grammatical structure in so-called agrammatic aphasics. *Cognition*, *13*(3), 361-392.

Love, T. & Brumm, K. (2012). *Language Processing Disorders.* In Cognitive and Acquired Language Disorders. Elsevier.

Love, T., Swinney, D., Walenski, M., & Zurif, E. (2008). How left inferior frontal cortex participates in syntactic processing: Evidence from aphasia. *Brain and Language*, *107*(3), 203-219.

Luria, A. R. (1975). Two kinds of disorders in the comprehension of grammatical constructions. *Linguistics*, *13*(154-155), 47-56.

Magnusdottir, S., Fillmore, P., Den Ouden, D. B., Hjaltason, H., Rorden, C., Kjartansson, O., ... & Fridriksson, J. (2013). Damage to left anterior temporal cortex predicts impairment of complex syntactic processing: A lesion-symptom mapping study. *Human brain mapping*, *34*(10), 2715-2723.

Matchin, W., Sprouse, J., & Hickok, G. (2014). A structural distance effect for backward anaphora in Broca's area: An fMRI study. *Brain and language*, *138*, 1-11.

Mesulam, M. M., Rogalski, E. J., Wieneke, C., Hurley, R. S., Geula, C., Bigio, E. H., ... & Weintraub, S. (2014). Primary progressive aphasia and the evolving neurology of the language network. *Nature Reviews Neurology*, *10*(10), 554-569.

Mesulam, M. M., Thompson, C. K., Weintraub, S., & Rogalski, E. J. (2015). The Wernicke conundrum and the anatomy of language comprehension in primary progressive aphasia. *Brain*, *138*(8), 2423-2437.

McElree, B., Foraker, S., & Dyer, L. (2003). Memory structures that subserve sentence comprehension. *Journal of Memory and Language*, *48*(1), 67-91.

Miller, G. A. (1962). Some psychological studies of grammar. *American psychologist*, *17*(11), 748.

Miller, G.A., Chomsky, N., (1963). Finitary models of language users. In: Luce, R.D., Bush, R.R., Galanter, E. (Eds.), *Handbook of Mathematical Psychology*, volume 2. Wiley, New York, pp. 419–491.

Mohr, J.P. (1976). Broca's area and Broca's aphasia. In: H. Whitaker & H.A. Whitaker, editors. *Studies in Neurolinguistics, volume 1*. New York: Academic Press.

Mohr, J. P., Pessin, M. S., Finkelstein, S., Funkenstein, H. H., Duncan, G. W., & Davis, K. R. (1978). Broca aphasia Pathologic and clinical. *Neurology*, *28*(4), 311-311.

Moro, A., Tettamanti, M., Perani, D., Donati, C., Cappa, S. F., & Fazio, F. (2001). Syntax and the brain: disentangling grammar by selective anomalies. *Neuroimage*, *13*(1), 110-118.

Murray, L.L., Holland, A.L. & Beeson, P.M. (1997). Auditory processing in individuals with mild aphasia: a study of resource allocation. J Speech Lang Hear Res 40(4), 792-808.

Murray, L.L., Holland, A.L. & Beeson, P.M. (1998). Spoken language of individuals with mild fluent aphasia under focused and divided-attention conditions. J Speech Lang Hear Res 41(4), 213-227.

Naeser, M. A. & Hayward, R.W. (1978). Lesion localization in aphasia with cranial computed tomography and the Boston Diagnostic Aphasia Exam. *Neurology, 28,* 545–545.

Nakayama, M. (1987). Performance factors in subject-aux inversion by children. *Journal of Child Language*, 14, 113-125

Okada, K., Smith, K.R., Humphries, C. & Hickok, G. (2003). Word length modulates neural activity in auditory cortex during covert object naming. *Neuroreport, 14*(18), 2323-2326.

O'Neill, Y.V. (1980). *Speech and speech disorders in Western thought before 1600*. Westport, CT: Greenwood Press.

Patterson, J.P. & Chapey, R. (2008). *Assessment of language disorders in adults*. In Language intervention strategies in aphasia and related neurogenic communication disorders (5th ed.) ed. Chapey, R.

Pedersen, P.M., Vinter, K. & Olsen, T.S. (2004). Aphasia after stroke: type, severity and prognosis. The Copenhagen aphasia study. *Cerebrovascular Disorders, 17*(1), 35-43.

Pesetsky, D. (1987). Wh-in-situ: Movement and unselective binding. In E. J. Reuland & A. G. B. ter Meulan (Eds.), The representation of (in)definiteness. Cambridge, MA: MIT Press

Pettigrew, C., & Hillis, A. E. (2014). Role for memory capacity in sentence comprehension: evidence from acute stroke. *Aphasiology*, *28*(10), 1258-1280.

Phillips, C. (1996). *Order and structure* (Doctoral dissertation, Massachusetts Institute of Technology).

Pillay, S. B., Binder, J. R., Humphries, C., Gross, W. L., & Book, D. S. (2017). Lesion localization of speech comprehension deficits in chronic aphasia. *Neurology*, *88*(10), 970-975.

Poeppel, D., & Embick, D. (2005). Defining the relation between linguistics and neuroscience. *Twenty-first century psycholinguistics: Four cornerstones*, 103-118.

Pollard, C., & Sag, I. A. (1994). *Head-driven phrase structure grammar*. University of Chicago Press.

Prather, P., Zurif, E., Stern, C., & Rosen, T. J. (1992). Slowed lexical access in nonfluent aphasia: A case study. *Brain and Language*, *43*(2), 336-348.

Rogalsky, C., & Hickok, G. (2011). The role of Broca's area in sentence comprehension. *Journal of Cognitive Neuroscience*, *23*(7), 1664-1680.

Rogalsky, C., Matchin, W., & Hickok, G. (2008). Broca's area, sentence comprehension, and working memory: an fMRI study. *Frontiers in Human Neuroscience*, *2*.

Rogalsky, C., Love, T., Driscoll, D., Anderson, S. W., & Hickok, G. (2011). Are mirror neurons the basis of speech perception? Evidence from five cases with damage to the purported human mirror system. *Neurocase*, *17*(2), 178-187.

Rogalsky, C., Almeida, D., Sprouse, J., & Hickok, G. (2015a). Sentence processing selectivity in Broca's area: evidence for structure but not syntactic movement. *Language, Cognition and Neuroscience*, *30*(10), 1326-1338.

Rogalsky, C., Poppa, T., Chen, K., Anderson, S.W., Damasio, H., Love, T. & Hickok, G. (2015b). Speech repetition as a window on the neurobiology of auditory-motor integration for speech: a voxel-based lesion symptom mapping study. *Neuropsychologia, 71,* 18-27.

Santi, A., & Grodzinsky, Y. (2007a). Working memory and syntax interact in Broca's area. *Neuroimage*, *37*(1), 8-17.

Santi, A., & Grodzinsky, Y. (2007b). Taxing working memory with syntax: Bihemispheric modulations. *Human brain mapping*, *28*(11), 1089-1097.

Sebastian, R., Long, C., Purcell, J.J., Faria, A.V., Lindquist, M., Jarso, S., Race, D., Davis, C., Posner, J., Wright, A. & Hillis, A.E. (2016). Imaging network level language recovery after left PCA stroke. Restor Neurol Neurosci, 34(4), 473-89.

Shahid, H., Sebastian, R., Schnur, T.T., Hanayik, T., Wright, A., Tippett, D.C., Fridriksson, J., Rorden, C. & Hillis, A.E. (2017). Important considerations in lesion-symptom mapping: Illustrations from studies of word comprehension. *Human Brain Mapping, 38*(6), 2990-3000.

Shallice, T. (1979). Case study approach in neuropsychological research. *Journal of Clinical Neuropsychology, 1,* 183–211.

Schwartz, M. F., Saffran, E. M., & Marin, O. S. (1980). The word order problem in agrammatism: I. Comprehension. *Brain and language*, *10*(2), 249-262.

Shaywitz, B.A., Shaywitz, S.E., Pugh, K.R., Constable, R.T., Skudlarski, P., Fulbright, R.K., Bronen, R.A., Fletcher, J.M., Shankweiler, D.P., Katz, L., et al. (1995). Sex differences in the functional organization of the brain for language. *Nature, 373* (6515), 607-609.

Sheppard, S. M., Walenski, M., Love, T., & Shapiro, L. P. (2015). The auditory comprehension of Wh-questions in aphasia: support for the intervener hypothesis. *Journal of Speech, Language, and Hearing Research*, *58*(3), 781-797.

Slowiaczek, M.L. & Clifton, C. (1980). Subvocalization and reading for meaning. *Journal of Verbal Learning and Verbal Behavior, 19,* 573-582.

Sprouse, J., & Hornstein, N. (2016). Syntax and the Cognitive Neuroscience of Syntactic Structure Building. In G. Hickok, & S. A. Small (Eds.), *The Neurobiology of Language*. Elsevier.

Stowe, L. A. (1986). Parsing WH-constructions: Evidence for on-line gap location. *Language and Cognitive Processes*, 1(3), 227–245.

Stromswold, K., Caplan, D., Alpert, N., & Rauch, S. (1996). Localization of syntactic comprehension by positron emission tomography. *Brain and language*, *52*(3), 452-473.

Teuber, H. L. (1955). Physiological psychology. *Annual Review of Psychology, 6,* 267–296.

Thompson, C. K., Lange, K. L., Schneider, S. L., & Shapiro, L. P. (1997). Agrammatic and non-brain-damaged subjects' verb and verb argument structure production. *Aphasiology*, *11*(4-5), 473-490.

Thompson, C. K., Tait, M. E., Ballard, K. J., & Fix, S. C. (1999). Agrammatic Aphasic Subjects' Comprehension of Subject and Object Extracted Wh-Questions. *Brain and Language*, *67*(3), 169-187.

Thothathiri, M., Kimberg, D. Y., & Schwartz, M. F. (2012). The neural basis of reversible sentence comprehension: evidence from voxel-based lesion symptom mapping in aphasia. *Journal of cognitive neuroscience*, *24*(1), 212-222.

Tremblay. P. & Dick, A.S. (2016). Broca and Wernicke are dead, or moving past the classic model of language neurobiology. *Brain and Language, 162,* 60-71.

Tyler, L.K., Wright, P., Randall, B., Marslen-Wilson, W.D. & Stamatakis, E.A. (2010). Reorganisation of syntactic processing following LH damage: Does RH activity preserve function. *Brain, 133*, 3396-3408.

Vaden, K.I. Jr, Kuchinsky, S.E., Cute, S.L., Ahlstrom, J.B., Dubno, J.R. & Eckert, M.A. (2013). The cingulo-opercular network provides word-recognition benefit. *Journal of Neuroscience, 33*(48), 18979-86.

Van Orden, G.C., Pennington, B.F. & Stone, G.O. (2001). What do double dissociations prove? *Cognitive Science 25*, 111-172.

Wagers, M. W., Lau, E. F., & Phillips, C. (2009). Agreement attraction in comprehension: Representations and processes. *Journal of Memory and Language*, *61*(2), 206-237.

Wernicke, C. (1874). Der aphasische Symptomencomplex: eine psychologische Studie auf anatomischer Basis. Cohn & Weigert.

Wilson, S.M. (2016). Lesion-symptom mapping in the study of spoken language understanding. *Language, Cognition and Neuroscience*, doi: 10.1080/23273798.2016.1248984.

Wilson, S. M., & Saygın, A. P. (2004). Grammaticality judgment in aphasia: Deficits are not specific to syntactic structures, aphasic syndromes, or lesion sites. *Journal of Cognitive Neuroscience*, *16*(2), 238-252.

Wilson, S. M., Henry, M. L., Besbris, M., Ogar, J. M., Dronkers, N. F., Jarrold, W., ... & Gorno-Tempini, M. L. (2010). Connected speech production in three variants of primary progressive aphasia. *Brain*, *133*(7), 2069-2088.

Wilson, S. M., Dronkers, N. F., Ogar, J. M., Jang, J., Growdon, M. E., Agosta, F., ... & Gorno-Tempini, M. L. (2010). Neural correlates of syntactic processing in the nonfluent variant of primary progressive aphasia. *Journal of Neuroscience*, *30*(50), 16845-16854.

Wilson, S. M., Galantucci, S., Tartaglia, M. C., Rising, K., Patterson, D. K., Henry, M. L., ... & Gorno-Tempini, M. L. (2011). Syntactic processing depends on dorsal language tracts. *Neuron*, *72*(2), 397-403.

Wulfeck, B. (1988). Grammaticality judgments and sentence comprehension in agrammatic aphasia. *Journal of Speech and Hearing Research*, *31*(1), 72-81.

Wulfeck, B., & Bates, E. (1991). Differential sensitivity to errors of agreement and word order in Broca's aphasia. *Journal of Cognitive Neuroscience*, *3*(3), 258-272.

Yourganov, G., Fridriksson, J., Rorden, C., Gleichgerrcht, E.& Bonilha, L. (2016). Multivariate Connectome-Based Symptom Mapping in Post-Stroke Patients: Networks Supporting Language and Speech. *Journal of Neuroscience, 33*(25), 6668-6679.

Zurif, E., Swinney, D., Prather, P., Solomon, J., & Bushell, C. (1993). An on-line analysis of syntactic processing in Broca's and Wernicke's aphasia. *Brain and language*, *45*(3), 448-464.

Zurif, E. B. (1980). Language Mechanisms: A Neuropsychological Perspective: The effects of focal brain damage on the processing of syntactic elements may provide an important clue to the manner in which language is organized in the brain. *American Scientist*, *68*(3), 305-311.