CHAPTER

74

Psycholinguistic Approaches to the Study of Syndromes and Symptoms of Aphasia

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74.1 INTRODUCTION

One of the fascinating aspects of aphasia is that language breakdown is not unitary. Not all aspects of language are affected in the same way and, crucially, depending on the site and extent of the lesion, brain injury to the left hemisphere results in a clinically diverse set of impairments and abilities. Persons with aphasia may show a constellation of impaired and spared abilities in auditory comprehension, speech output, repetition, naming, reading, and writing that together comprise a particular syndrome.

There is a long history of the characterization of the aphasia syndromes (Geschwind, 1965; Benson, 1979 for review), and although there are differences in nomenclature, there is general consensus on the clinical features that comprise a particular syndrome. However, syndromes provide a description of clinical behaviors. They do not provide an explanation of the language deficits that give rise to these behaviors. For example, a failure to understand language could be due to any number of factors—among them, impairment in processing the sound structure of language, impairment in mapping sounds to word meaning, and/or failure to process the syntactic structure or the semantic structure of words. To understand the nature of language deficits in aphasia requires taking a different approach, one that takes into consideration the potential language processes and mechanisms underlying these deficits. Roman Jakobson (1956) was probably the first linguist to take such a psycholinguistic approach to aphasia. Since then, there has been a history and tradition of using the theoretical framework and experimental tools provided by psycholinguistics to examine the nature of deficits among the different types of aphasia (Goodglass, 1993). As discussed, such a study has provided the framework for much of the recent work using functional neuroimaging and electrophysiological approaches to the neurobiology of language.

This chapter reviews the contributions that this approach has made to our understanding of the neurobiology of language. We begin by describing those classical aphasia syndromes and their associated lesion profiles that have served as the focus of much of the research on language impairments in aphasia. The psycholinguistic studies that were conducted were designed to understand the basis of the underlying deficits giving rise to particular clinical features of these syndromes and the functional role of the brain areas involved. The findings have also been used to provide insight into the functional architecture of language, that is, how the system fractionates provides a window into its structural properties and the mechanisms and processes involved in normal language use.

74.2 THE APHASIA SYNDROMES

The aphasia syndromes typically result from damage to perisylvian areas of the left hemisphere. Although there have been many syndromes described in the literature (see Geschwind, 1965 for a review), the three that have probably been studied in the most detail and have served as the foundation for psycholinguistic studies of aphasia are Broca's, conduction, and Wernicke's aphasia. These syndromes provide a rich tapestry of impaired and spared language abilities. Of importance, they are defined in terms of the *relative* performance of patients among a set of language functions, including speaking, auditory comprehension, repetition, naming,

and comprehension and production in reading and writing. Thus, an absolute score on a particular language function cannot be used to classify a patient. Rather, it is performance on a particular language function in relation to the other clinical characteristics that defines a patient's syndrome.

Focusing solely on a particular language function will result in grouping patients together who have different syndromes and potentially different underlying deficits. For example, there are a number of aphasia syndromes that are characterized by poor auditory comprehension, including Wernicke's aphasia, transcortical sensory aphasia, and global aphasia (Goodglass & Kaplan, 1972). Similarly, there are a number of syndromes that are characterized by fluent speech output, including Wernicke's aphasia, conduction aphasia, transcortical sensory aphasia, and anomic aphasia. It is the relationship between these various language functions that differentiate between and among the aphasia types.

Broca's aphasia is characterized by impairment in language expression in the face of relatively good auditory comprehension. Speech output of these patients is typically nonfluent: production is slow, labored, with many speech errors, and is often dysarthric, (i.e., characterized by motor weakness that affects speech articulation). Additionally, these patients often display agrammatism in their speech output, with a tendency to omit freestanding function words such as *the* and *is* and to either delete or substitute grammatical endings. Repetition is usually similar to or a little better than spontaneous speech output. Lesions associated with Broca's aphasia typically involve the frontal operculum (Broca's area, i.e., BA44 and 45) and premotor and motor regions posterior and superior to the frontal operculum, and extend to the white matter structures including the basal ganglia and insula (Damasio, 1998).

Wernicke's aphasics show a very different clinical picture. They have fluent well-articulated speech in the context of impairment in auditory language comprehension. These patients often produce paraphasias, which are errors in their output that are either phonologically based (phonemic paraphasias, e.g., *top* → *dop*) or semantically based (verbal paraphasia, e.g., *wife* → *sister*). Some Wernicke's aphasics produce jargon or neologisms, which are productions that are phonologically possible but are not words in the language (e.g., *tufbei*). Although speech output is fluent, containing grammatical words and endings, sentences are often described as paragrammatic, characterized by the inappropriate juxtaposition of words often rendering the sentence ungrammatical. Additionally, the content of the discourse is typically empty semantically, partly because of the overuse of semantically empty high-frequency words such as *thing*, *is*, *this*. Wernicke's patients also have repetition impairment as well as a naming deficit. The lesions associated with Wernicke's aphasia include the posterior superior temporal gyrus (BA22), often extending to the middle temporal, supramarginal, and angular gyri (Damasio, 1998; Dronkers, Redfern, & Ludy, 1995; Dronkers, Wilkins, Van Valin, Redfern, & Jaeger, 2004).

In conduction aphasia, repetition is the presenting deficit in the context of fluent, well-articulated speech and relatively good auditory language comprehension. Speech output contains phonemic paraphasias and relatively few semantic paraphasias. The patient appears to be aware of these errors because they often attempt to correct them, producing *conduite d'approche* or successive approximations to the target word. The lesions associated with conduction aphasia include the supramarginal gyrus and the white matter structures deep to it (the arcuate fasciculus). Importantly, the posterior portion of the superior temporal gyrus (Wernicke's area) is typically spared (Damasio, 1998; but see Hickok et al., 2000).

These clinical characteristics and associated lesion loci have raised a series of questions about the functional and neural architecture of language. With respect to the functional architecture of language, it is generally assumed that both expressive and receptive language functions comprise different linguistic domains involving multiple stages of processing. These domains include phonological/phonetic, lexical, syntactic, and conceptual/semantic. It is also generally assumed that information flows from one stage of processing to the other and that this information flow is interactive, with activation at one stage of processing influencing activation at other stages of processing both upstream and downstream from it (Dell, 1986; Marslen-Wilson & Warren, 1994). For example, phonetic/phonological information affects lexical processing (bottom-up processing), and lexical processing, in turn, can affect phonetic/phonological processing (top-down processing).

The clinical characteristics of Broca's, conduction, and Wernicke's aphasia suggest potential deficits in the representations and processes involved in the reception and/or expression of speech, words, and syntax. And it is these domains that we review here. At the same time, they also provide potential insight into the neural systems underlying these domains, allowing for an examination of whether the neural areas are functionally autonomous.

74.3 SOME CAVEATS AND CHALLENGES

The use of the aphasia syndromes as the framework to investigate the neurobiology of language has been met with challenges in the literature that are

worthwhile to briefly review and consider. In particular, there have been challenges to the assumption that clinical syndromes can be used to reliably classify and study participants with aphasia (Caramazza, 1984), and there have been challenges to the assumption that there is one-to-one mapping between clinical syndrome and underlying neuropathology (Willmes & Poeck, 1993). This has led to two other approaches over the past 30 years in the investigation of language deficits in aphasia. The first has focused on detailed case studies of patients as a means of informing current theories of language processing (Caramazza, 1986; see Rapp & Goldrick, 2006 for a review). The single case methodology has been used throughout the history of neuropsychology and has provided many insights into not only how language may fractionate but also what neural areas give rise to the deficit (Geschwind, 1965 discussion of Dejerine). However, the more recent case study approach has largely been agnostic with respect to the underlying neuropathology of the patient (Rapp & Goldrick, 2006). Thus, a downside of this approach is that it cannot provide insight into the neural architecture underlying the patient's deficit, nor can it provide *predictions* of patterns of deficits to new patients. In the second approach, a psycholinguistic question is raised and then studied in a group of patients unselected for either syndrome or lesion (see Schuell & Jenkins, 1959). The assumption here is that the underlying deficit is the same irrespective of lesion localization. Under these assumptions, it is impossible to determine whether there are distinct deficits that arise as a function of a particular area of damage and whether the basis of the deficit differs as a function of lesion site.

In the end, it is lesions that produce language impairments in aphasia. And the rationale for focusing on syndromes is that they not only present with a constellation of impairments but also are the result of lesions to particular neural areas. With advances in neuroimaging techniques over the past 20 years, we know that lesions of patients are rarely focal and typically include both cortical and subcortical structures. No individual with aphasia has exactly the same lesion profile, and there are differences not only in the extent of the lesion but also in the degree of damage to a particular area. With regard to behavioral effects of lesions, we know that within a syndrome there are differences in severity and that not all patients can be classified into a particular syndrome, in both cases presumably because of the extent and location of the lesion. We also have learned that damage in one area can result in hypometabolism in areas distant from it, even in the absence of overt structural damage (Metter, Hanson, Jackson, & Kempler, 1990). Such findings suggest that deficits may reflect neural systems, rather than solely being due to local pathology.

Taken together, such observations identify the complexities of this research endeavor. That the picture is more complex than what the classical aphasiologists from the 1920s to the 1960s had proposed in terms of clinical diagnosis and lesion locus is not surprising. However, these facts do not obviate the existence of the aphasia syndromes or that lesions are localizable and ultimately produce different patterns of language impairment broadly in line with the classical aphasia syndromes (Kreisler et al., 2000; Richardson, Fillmore, Rorden, LaPointe, & Fridriksson, 2012). The results of this approach converge with recent neuroimaging findings focusing on the neural substrates of language.

74.4 LANGUAGE DEFICITS UNDERLYING APHASIA SYNDROMES

It is beyond the scope of this article to review the extensive literature on psycholinguistic investigations of aphasia. Rather, we examine two main classes of findings that have been shown in each of the domains of phonetics/phonology, the lexicon, and syntax. The first class of findings has shown similarities in patterns of deficits across patients presenting with different aphasia syndromes. Such results provide insight into the functional architecture of language, typically showing integrity of the structural properties of language. They also suggest that such processing recruits a broadly distributed neural system. The second class of findings has shown deficits in patients presenting with different aphasia syndromes; however, of interest and importance, the patterns of deficits differ as a function of clinical syndrome. Such findings suggest that the nature of the deficit giving rise to the pathological performance is due to a different functional impairment presumably reflecting the neural locus of the lesion.

74.4.1 Phonetic/Phonological Impairments

74.4.1.1 Speech Production

Although all aphasic patients make speech production errors across a number of language tasks, based on the clinical picture of the patients, it was generally assumed that the source of those errors differed. In particular, given that the lesions of Broca's aphasics involved frontal structures typically including motor areas, they were considered to have phonetic impairments reflecting articulatory planning and articulatory implementation deficits. In contrast, given that Wernicke's and conduction aphasics' lesions involved posterior areas, these patients were considered to have phonological impairments reflecting selection deficits. Experimental results showed that these

characterizations were both correct and wrong. In particular, acoustic analysis of the patterns of production of a number of parameters of speech showed clear-cut phonetic impairments for Broca's aphasics that were not present in either Wernicke's or conduction aphasics. Broca's aphasics showed deficits in the timing relations required for voicing in stop consonants (voice-onset time) (Blumstein, Cooper, Goodglass, Statlender, & Gottlieb, 1980; Gandour & Dardarananda, 1984; Kent & Rosenbek, 1983), timing of voicing onset and amplitude properties required for voicing in fricative consonants (Kurowski, Hazen, & Blumstein, 2003), and duration and amplitude measures required for the production manner of articulation for nasal consonants (Kurowski, Blumstein, Palumbo, Waldstein, & Burton, 2007). Although the two posterior aphasic groups showed normal articulatory implementation, they did show more variability in their productions than normal individuals. Taken together, these findings support the view that Broca's aphasics have articulatory planning and implementation deficits, and that frontal brain structures including Broca's area, premotor and motor regions posterior and superior to the frontal operculum, and white matter structures deep to them are functionally involved in these stages of production. That the posterior patients only showed variability in their productions in the context of normal articulatory implementation suggests that speech production not only recruits frontal structures but also invokes posterior structures, presumably as part of a feedback mechanism for error detection, monitoring, and sensorimotor integration from auditory (temporal cortex) and somatosensory (inferior parietal cortex) areas (Guenther, Ghosh, & Tourville, 2006; Hickok, 2012; Hickok, Houde, & Rong, 2011; Houde & Nagarajan, 2011; see Blumstein & Baum for discussion of neuroimaging studies supporting this view, Chapter 55, this volume).

In contrast to phonetic deficits, studies of phonological analysis of the speech of Broca's, conduction, and Wernicke's aphasia failed to show different patterns of errors. Analyses of speech output errors showed that patients from all three groups made similar phonological errors, including phoneme substitutions (phonemic paraphasias), addition or deletions of sounds, transposition of sounds either within a word or between words, and contextual errors based on the phonological context of the word (Blumstein, 1973). For all patients, errors reflected structural principles of the language. In particular, sound substitutions were more likely to occur between sounds that were distinguished by a single phonetic feature, and both addition and deletion errors were more likely to result in the canonical syllable structure CV (e.g., segments were typically added if a word began with a vowel, e.g., "elevator" → /kɛləvetr/, sound segments were added to produce a word with a CV onset, e.g., "cloudy" → /kəlawdi/, and consonant clusters were simplified, "French" → /fɛnč/). The similar pattern of errors irrespective of clinical syndrome suggests that whether the error occurred at selection, articulatory planning, or implementation stages of production, the basic structure of words and the phonological principles underlying them are preserved. It is not surprising that when errors occur, they are more likely to be manifest among phonologically similar sounds and to result in "simpler" phonological structures.

74.4.1.2 Speech Perception

As described, one of the distinguishing clinical features for Wernicke's aphasics is poor auditory comprehension. The question is, what is the basis or bases of this impairment? Because the primary auditory areas surface in the temporal lobe and the superior temporal gyrus has been implicated in speech perception (Binder, 2000; Hickok & Poeppel, 2007), one likely candidate deficit that has been studied in detail is an impairment in the processing of the sounds of speech. Luria (1966) proposed that Wernicke's aphasics have a deficit in phonemic hearing. In this view, an inability to correctly perceive the phonological properties of speech sounds leads to poor auditory comprehension and results in what appears to be semantic impairments. For example, it is not uncommon for such patients to select the incorrect picture of a word such as "pea" if it is presented in an array of phonologically similar items such as "bee," "T," or "key."

A series of studies was conducted investigating discrimination and identification of both naturally produced and synthetically constructed stimuli (Csepe, Osman-Sagi, Molnar, & Gosy, 2001; Leeper, Shewan, & Booth, 1986). Results showed that Wernicke's aphasics displayed severe speech perception deficits (Basso, Casati, & Vignolo, 1977; Blumstein, Baker, & Goodglass, 1977; Caplan, Gow, & Makris, 1995; Gow & Caplan, 1996; Robson, Keidel, Lambon Ralph, & Sage, 2012; see Hickok, 2009 for an alternative view). In addition, however, Broca's and conduction aphasics also showed deficits, although they were milder (see Hickok, Costanzo, Capasso, & Miceli, 2011 for an alternative view). Of interest, the predictive relationship between performance on these tasks and comprehension ability was inconsistent across studies; some failed to show a relationship (Basso et al., 1977; Blumstein et al., 1977), whereas others did show a relationship (Miceli, Gainotti, Caltagirone, & Masullo, 1980; Robson et al., 2012). These findings suggest that Wernicke's aphasics do have a speech perception deficit, but that other aspects of language, particularly semantic processing, may also be impaired (Baker, Blumstein, &

Goodglass, 1981; Basso et al., 1977; Robson, Sage, & Lambon Ralph, 2012; but see Walker et al., 2011). The possibility that Wernicke's aphasics have not only speech perception impairment but also a deficit in semantic processing is also supported by the neuroimaging literature. As described, it is not uncommon for the lesion profile of Wernicke's aphasics to extend into the middle temporal gyrus, an area that is involved in semantic processing and appears to be recruited in accessing stored semantic representations (see Binder, Desai, Graves, & Conant, 2009 for a review).

The findings that speech perception impairments emerge not only in Wernicke's aphasics but also in milder forms in Broca's and conduction aphasics are consistent with neuroimaging results showing that multiple neural areas are recruited in the processing of speech. Studies examining phonological contrasts (Burton, Small, & Blumstein, 2000) and acoustic phonetic properties of speech (Blumstein, Myers, & Rissman, 2005; Joanisse, Zevin, & McCandliss, 2007; Liebenthal et al., 2010) have shown activation in a neural network, including temporal (superior temporal gyrus), parietal (supramarginal gyrus), and frontal (inferior frontal gyrus) areas. It has been argued that the functional role of these areas differs with superior temporal areas (potentially bilaterally) recruited in earlier stages of speech processing, temporoparietal areas (posterior superior temporal and supramarginal gyri) involved in phonological processing (Buchsbaum, Hickok, & Humphries, 2001; Hickok et al., 2008; Hickok, 2009), and frontal areas engaged in executive processes related to phonetic category decisions (Burton et al., 2000; Myers, Blumstein, Walsh, & Eliassen, 2009; see also Venezia, Saberi, Chubb, & Hickok, 2012).

Nonetheless, of interest, despite differences in overall performance of the patients in speech perception tasks, their patterns of impairment were similar. Namely, all patients were more likely to make discrimination errors on stimulus pairs that were distinguished by a single phonetic feature than several features, and they were more likely to make errors discriminating stimulus pairs contrasting in the features corresponding to place of articulation than for the feature voicing (Blumstein et al., 1977). Finally, all patients displayed more deficits in perceiving the sound structure of nonsense syllables compared with real words. That Wernicke's aphasics showed a systematic pattern of impairment similar to other aphasic patients indicates that their behavior is not random and does not reflect a loss of sensitivity to the phonetic/phonological properties of speech.

The patterns of performance displayed by the patients reflect the integrity of the structural properties of the sound structure of language. Sounds distinguished by a single phonetic feature not only share more phonological features but also are more similar acoustically than are sounds distinguished by multiple features. Thus, it is not surprising that they are more difficult to discriminate. Nonetheless, despite the difficulty that all patients have in either discriminating or categorizing acoustic cues associated with either voicing or place of articulation, the locus and shape of the phonetic boundary are similar to those of normal individuals. The superiority of performance for real words compared with nonsense syllables is consistent with current models of the functional architecture of language (Dell, 1986; McClelland & Elman, 1986). Here, information flow is interactive; namely, information flows from phonetic/phonological analysis stages to activate potential lexical candidates. These candidates, in turn, boost the activation of phonological units downstream from them. Because nonwords do not match any words in the lexicon, they may only weakly activate phonologically similar words (Milberg, Blumstein, & Dworetzky, 1988). In such a case, they do not have the same degree of support of the lexical-semantic network; hence, they are more vulnerable in tasks that focus on phonological/phonetic properties.

74.5 LEXICAL IMPAIRMENTS

Models of the cognitive architecture of language have proposed that the words of a language (the mental lexicon) are organized in terms of a network-like architecture of shared or partially overlapping sound structure or semantic properties (Gaskell & Marslen-Wilson, 1999; Plaut, 1995). In this view, a word not only activates its phonological and semantic representations but also partially activates words that share sound structure and semantic properties with it. As a consequence, accessing a word for either spoken production or auditory word recognition requires selecting the target word from this set of activated competitors. Thus, both the production and recognition of words require a multistage process including access to the mental lexicon, activation of a network of potential word candidates, and, ultimately, the selection of the target word from the set of semantically related and phonologically related competitors.

One of the most common and least localizing clinical features in aphasia is a word retrieval deficit. This may be shown either in spoken word production or in auditory word recognition. In spoken word production, patients may fail to come up with a word either in spontaneous speech output or when presented with a picture or verbal description of a word (naming) (Goodglass & Kaplan, 1972). Typically, naming errors include phonemic paraphasias, where the patient

makes a sound error on the target word, or verbal (semantic) paraphasias, where the patient produces an incorrect word, often semantically or associatively related to the target word. Clinically, Wernicke's aphasics tend to make more semantic paraphasias, and conduction aphasics make more phonemic paraphasias. In auditory word recognition, Broca's, conduction, and Wernicke's aphasics may fail to select a picture of a word from an array of pictures whose names are either phonemically or semantically related to the target (Baker et al., 1981).

Evidence suggests that although Broca's, conduction, and Wernicke's aphasics display word processing deficits, it is not because they have lost either the "concept" of a word or its phonological representation. Rather, they appear to have difficulty accessing or retrieving the word. Improved naming occurs for these patients with contextual support provided by either a phonological cue (e.g., producing [bə] for the word "bear") or a semantic cue ("Smoky, the _.").

The relative preservation of semantic/conceptual representations has been shown in studies examining semantic priming. Both Broca's and Wernicke's aphasics show semantic priming in a lexical decision task (Blumstein, Milberg, & Shrier, 1982; de Salles, Holderbaum, Parente, Mansur, & Ansaldo, 2012; Hagoort, 1997; Milberg & Blumstein, 1981). That is, they display shorter reaction time latencies to target words preceded by semantically related ("dog–cat") compared with semantically unrelated ("ring-cat") words. These findings support the integrity of the lexical-semantic network in these patients. In contrast to the classical aphasias, however, recent research suggests that degradation of semantic structure does occur, but in patients with semantic dementia or aphasic patients with lesions extending to the anterior temporal lobe (an area not included in the lesion profile of Wernicke's aphasics) (Jefferies & Lambon Ralph, 2006; Walker et al., 2011).

The relative preservation of phonological representations has been shown in studies examining the tip-of-the-tongue state in aphasic patients. The tip-of-the-tongue state is a phenomenon in which subjects are unable to come up with a word but "feel" that they know what the word is and, in fact, that the word is on the "tip of their tongue." Brown and McNeil (1966) showed that normal individuals retain the sound structure properties of words that they failed to retrieve; they can identify its first letter, the number of syllables it has, and words that are semantically related to it. Although aphasics show a similar sensitivity to the sound structure of words they cannot name (Barton, 1971), Goodglass, Kaplan, Weintraub, and Ackerman (1976) showed that conduction aphasics were better able to recall the sound properties of words compared with both Broca's and Wernicke's aphasics. Thus, they suggested that the differences in the pattern of performance reflected different stages in word retrieval.

Despite the relative preservation of the lexical-semantic and phonological structure of words in Broca's, conduction, and Wernicke's aphasics, these patients do show deficits in the various stages involved in lexical access. In particular, a series of studies have shown that Wernicke's aphasics are able to activate lexical candidates; however, the lexical competitors remain active longer (or fail to get inhibited). In contrast, Broca's aphasics also activate lexical candidates; however, they are unable to resolve competition in selecting the target word from among competing lexical candidates. These findings have been shown using a variety of paradigms, including lexical decision (Janse, 2006; Milberg, Blumstein, & Dworetzky, 1987) and eyetracking (Yee, Blumstein, & Sedivy, 2008). Additional support for selection deficits comes from both verb generation (Thompson-Schill et al., 1998) and lexical decision studies (Bedny, Hulbert, & Thompson-Schill, 2007) that focused on lesion location, in this case a portion of the inferior frontal gyrus (BA45), and not clinical diagnosis of aphasia.

The lexical processing deficits of Broca's and Wernicke's aphasics emerge whether the source of the competition is semantic or phonological. For example, in the semantic case, the patient must select a word from competing meanings of ambiguous words presented in congruent and incongruent contexts (e.g., the subject is required to make a lexical decision on the third word of a triplet such as "coin-bank-money" versus "river-bank-money") (Bedny et al., 2007; Milberg et al., 1987). Another study required the patient to select words that have high versus low selection demands (e.g., the subject is asked to generate a verb for the word "scissors" versus "ball") (Thompson-Schill et al., 1998).

Several paradigms have been used to investigate the effects of resolving phonological competitors. One set of experiments used eyetracking and examined the potential effects of onset competitors in selecting a target word (Yee et al., 2008). Here, the subject was asked to point to a picture given the auditory presentation of a word from an array that included the picture of the target word, a word with an onset competitor, and two semantically and phonologically unrelated foils (e.g., the target word is "hammock" and the pictures include "hammock," "hammer," "monkey," and "chocolate"). Another series of experiments examined the effects of acoustically degraded prime stimuli on the magnitude of semantic priming (Misiurski, Blumstein, Rissman, & Berman, 2005; Utman, Blumstein, & Sullivan, 2001). Stimulus pairs included semantically related stimuli

with and without voicing competitors, "time-clock" (with a voiced competitor "dime") and "cat–dog" (with no voiced competitor, "gat" is not a word). Similar to normal individuals, Broca's aphasics showed semantic priming for phonologically clear, semantically related pairs, and reduced semantic priming for degraded prime stimuli without a voiced competitor. In contrast to normal individuals, Broca's aphasics lost priming only when the degraded prime had a voiced competitor.

Taken together, these findings indicate that aphasic patients have lexical processing impairments. In particular, aphasics retain the underlying semantic and phonological representations of words, but they show impairments in *accessing* them. Moreover, different patterns of performance emerge between Broca's and Wernicke's aphasics, suggesting that the basis of their deficit differs (see Blumstein, 2009, for review; Janse, 2006). For Wernicke's aphasics, word candidates stay active longer either due to an inability to inhibit word competitors or due to their overactivation. In contrast, Broca's aphasics show a deficit in selection processes and an inability to select among competing semantic and phonological competitors.

Neuroimaging findings support the view that both spoken word production and word recognition processes engage a temporoparietal and frontal network. Semantic processing recruits both temporal and frontal structures (see Binder et al., 2009 for a review); selection among competing semantic alternatives recruits the inferior frontal gyrus (Thompson-Schill, D'Esposito, Aguirre, & Farah, 1997); and selection among competing phonological alternatives also recruits the inferior frontal gyrus as well as the posterior superior temporal and supramarginal gyri (Righi, Blumstein, Mertus, & Worden, 2010).

74.6 SYNTACTIC IMPAIRMENTS

As described, one of the clinical characteristics of some Broca's aphasics is agrammatism in speech output in the context of generally good auditory comprehension. There is a long and controversial history of the potential basis of the grammatical deficit in these patients. Early hypotheses (Kolk & Heeschen, 1990; Kolk & Van Grunsven, 1985) suggested that the deficit reflected a compensatory mechanism of the patient to provide the most semantic content with the least amount of speech. In this view, the output disorder reflects an "economy of effort" and thus is secondary to nonfluent output and difficulty in producing and articulating speech. Evidence in support of this view came from analyses of the error patterns in production. Results showed that there was not only a tendency to omit function words but also a tendency to simplify morphological structures, particularly in contexts where the morphological ending was redundant (e.g., "two books" → "two book") (Dick, Bates, Wulfeck, Utman, & Gernsbacher, 2001). As shown by analyses of inflected languages, morphological errors produced by agrammatic aphasics were in fact substitutions of one morphological ending for another, not a "loss" of endings (Grodzinksy, 1990; Menn & Obler, 1990). Analyses showed that there was a tendency to produce a linguistically less marked structure such as a verb in the present tense or in infinitival form rather than a verb with a past tense or future tense inflection.

A series of seminal studies by Zurif and colleagues (Caramazza & Zurif, 1976; Goodenough, Zurif, & Weintraub, 1977; Zurif, Caramazza, & Myerson, 1972), however, suggested that the agrammatic deficit of Broca's aphasics was not limited to speech production, but rather was a "central" impairment affecting not only speech production but also comprehension. This was originally shown using a hierarchical clustering paradigm in which subjects were presented the written form of a sentence such as "the dog chased a cat" (Zurif et al., 1972). With the sentence always in display, subjects were given a random selection of three cards, each containing one of the words in the sentence. They were asked to put "the two words that went best together." Results showed that Broca's aphasics did not know where/how to cluster the function words. They were as likely to cluster "the" and "a" with each other than within their associated noun phrases. Thus, Broca's aphasics showed impairment in their linguistic "intuitions" about the syntactic structure of sentences.

From there, a plethora of studies examined sentence comprehension in aphasia focusing on syntactic structures. Results have shown that Broca's aphasics displayed impairments in comprehending sentences when the only cue to comprehension was syntax (e.g., "the lion chased the tiger" versus "the boy ate the hamburger") (Caramazza & Zurif, 1976). They had difficulty in understanding noncanonical syntactic structures such as passive sentences compared with active sentences ("the girl is liked by the boy" versus "the boy likes the girl"), syntactically complex compared with simple sentences ("the boy who sees the man likes the girl" versus "the boy likes the girl") object-embedded compared with subject-embedded sentences ("the boy the girl likes reads a book" versus "the girl likes the boy who reads the book") and sentences that did and did not contain traces (Caplan, Baker, & Dehaut, 1985).

These findings gave rise to a large number of hypotheses to characterize the underlying impairment, the details of which are beyond the scope of this article. The proposals are far-reaching,

invoking either representational (Grodzinsky, 1986, 2000; Mauner, Fromkin, & Cornell, 1993) or processing deficits (Grodzinsky & Friederici, 2006; Love, Swinney, Walenski, & Zurif, 2008) involving potential impairments in syntactic structures governing movement and/or binding (Choy & Thompson, 2010; Swinney & Zurif, 1995), thematic role assignment (Saffran, Schwartz, & Linebarger, 1998), working memory or resource limitations (Caplan & Waters, 1995; Carpenter, Miyake, & Just, 1995), and time-course delay of processing (Ferrill, Love, Walenski, & Shapiro, 2012; Love, Swinney, & Zurif, 2001). See Chapter 47 (Rogalsky) of this volume for further discussion.

Beyond the debate concerning the underlying deficit in Broca's aphasics, there is a more critical issue—namely, is it truly the case that only Broca's aphasics display syntactic comprehension impairments? Unfortunately, much of the literature examining the basis of syntactic impairments has tested only Broca's aphasics. Thus, it is not clear whether other types of patients also show impairments. Those studies that have looked at other patient groups or patients with different lesion sites show similar patterns of impairment as those of Broca's aphasics (Caplan et al., 1995; Caplan, Hildebrandt, & Makris, 1996; Dick et al., 2001; Zurif & Caramazza, 1976). It is not surprising to find that structurally complex sentences are more difficult to understand not only for aphasic patients but also for neurologically intact subjects tested under adverse listening conditions (Dick et al., 2001; cf. also Obleser, Meyer, & Friederici, 2011).

What is not clear from these studies is whether the *basis* of the impairment is different across aphasia syndromes. Although some hypotheses have been proposed (Friederici, 2011), no studies have yet been conducted that distinguish behavioral performance of patients based on some operational measure of the purported functional deficit. One challenge inherent in this research is assessing syntactic comprehension independent of meaning.

The neuroimaging literature has shown similar conflicting findings. Some studies have shown selective activation of the inferior frontal gyrus in auditory processing of syntactic structure (Moro et al., 2001; Stromswold, Caplan, Alpert, & Rauch, 1996), and others have shown a broad fronto-temporo-parietal network (Fedorenko, Nieto-Castañon, & Kanwisher, 2012; Friederici, Meyer, & von Cramon, 2000; Just, Carpenter, Keller, Eddy, & Thulborn, 1996; see Kaan & Swaab, 2002 for a review).

Although it remains unclear whether there are functional distinctions in the auditory processing of syntactic structure as a function of clinical syndrome and/or lesion site, the original observation that only Broca's aphasics display agrammatism in production remains. This leaves open the possibility that these patients do have a selective syntactic impairment, but it is restricted to spoken language production. It is for future research to determine whether this is the case and what the underlying basis of this impairment may be.

74.7 CONCLUSION

Psycholinguistic studies of the clinical syndromes of aphasia have provided a unique window into the neurobiology of language. Such studies offer insights that behavioral and neuroimaging studies alone cannot. Behavioral studies do not provide evidence of the neural systems underlying a particular deficit. Neuroimaging studies are unable to determine whether activation of a neural area indicates that it is *necessary* for a particular linguistic function. Coupled with these approaches, technological advances now available for detailed mapping of lesion profiles coupled with careful clinical examination and classification of patients hold the promise of not only gaining a deeper understanding of the functional and neural architecture of language but also providing critical insights into the bases of language deficits that can be used in developing rehabilitation programs for patients with aphasia.

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