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LANGUAGE PROCESSING: Functional Organization and Neuroanatomical Basis

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■ **Abstract** Earlier formulations of the relation of language and the brain provided oversimplified accounts of the nature of language disorders, classifying patients into syndromes characterized by the disruption of sensory or motor word representations or by the disruption of syntax or semantics. More recent neuropsychological findings, drawn mainly from case studies, provide evidence regarding the various levels of representations and processes involved in single-word and sentence processing. Lesion data and neuroimaging findings are converging to some extent in providing localization of these components of language processing, particularly at the single-word level. Much work remains to be done in developing precise theoretical accounts of sentence processing that can accommodate the observed patterns of breakdown. Such theoretical developments may provide a means of accommodating the seemingly contradictory findings regarding the neural organization of sentence processing.

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INTRODUCTION

The traditional view of language and the brain, instantiated in the Wernicke-Lichtheim model (Wernicke 1874, Lichtheim 1885), held that auditory word representations were localized in a posterior temporal brain region, now known as Wernicke's area, and motor word representations were localized in a frontal region, now known as Broca's area (see also Geschwind 1970). Damage to the motor word representations in Broca's area would result in a disruption of language production with a sparing of comprehension. Damage to sensory word images in Wernicke's area would impair comprehension but would not produce the mirror image syndrome to Broca's aphasia, as production would also be affected. That is, Wernicke argued that input from sensory word images was needed to select the appropriate motor word representations.

The description above is oversimplified, but even in a more elaborated form, the Wernicke-Lichtheim model falls far short of explaining the complexities of language processing (see Caplan 1987, Dronkers & Larsen 2001). The model does capture a striking difference between patients who have anterior damage and those who do not in that those with anterior damage produce nonfluent speech characterized by slow, labored articulation, whereas those with posterior damage typically produce fluent, rapidly articulated speech (though marked by phonological, grammatical, and semantic errors). (See Table 1, examples 1 and 2.) One salient limitation of this theory is that it is a theory of single-word processing and thus says nothing about the means by which words are combined into sentences. Patients who produce nonfluent speech tend to produce speech that is "agrammatic," that is, having reduced syntactic complexity and an absence of function words (e.g., prepositions and auxiliary verbs) and inflectional markers (e.g., plural markers on nouns and past tense markers on verbs). This agrammatism is not obviously accounted for by a disruption to motor word representations (but see Lenneberg 1973).

Studies during the 1970s and early 1980s uncovered surprising findings that challenged the traditional model. These findings demonstrated that although Broca's aphasics seemed to have good comprehension on clinical assessment, they showed poor comprehension when comprehension depended on understanding the syntactic information in a sentence (e.g., Caramazza & Zurif 1976, Schwartz et al. 1980). For example, when asked to match a sentence such as "The dog was chased by the cat" to a picture, these patients had difficulty choosing between a correct picture and one that reversed the roles of agent and object (i.e., a picture of a dog chasing a cat). They did well, however, if the incorrect picture substituted a different noun or verb. Berndt & Caramazza (1980) argued that the syndrome of Broca's aphasia should be redefined as reflecting a disruption of syntax, which was the underlying cause of agrammatic speech and the sentence comprehension deficit. The labored articulation of the Broca's aphasics was thought to be a co-occurring deficit resulting from damage to motor areas for speech adjacent to Broca's area. Caramazza & Berndt (1978) presented evidence that aphasic patients with posterior damage had

TABLE 1 Examples of aphasic language production

1. Nonfluent aphasics telling Cinderella story
   a. Severely nonfluent (left frontal/parietal/temporal CVA)
      JS: girl . . . three . . . bad . . . wish . . . pump . . . poor girl . . .
   b. Moderately nonfluent (left frontal parietal CVA)
      ML: Always working . . . Doesn't do . . . Sunrise and sunset, work, work, work . . .
      Three ladies will attend . . . uh . . . All the fineries and . . . uh . . . all the . . .
2. Fluent aphasic conversation with speech therapist (left temporal parietal CVA)
   Therapist: What did you have (to eat)?
   PH: Today I haven't touched a /maiwa/ /dˆ/ David. He had beastly tomorrow.
   Therapist: Was the food good?
   PH: Yes, it was fine.
3. Anomic aphasic attempting to name a picture of a vest (herpes encephalitis, predominantly left temporal damage)
   MS: This is something that . . . depends on what it looks like. Sometimes women wear it, sometimes men wear it. And you put underneath before you . . . You put a shirt on. Then you put this on. Then you put a coat on . . .

a disruption of semantic representations, which again affected both comprehension and production. They cited findings from Blumstein et al. (1977) as indicating that these patients' word comprehension deficits could not be attributed to difficulty perceiving phonological information. Thus, according to these authors, the theoretical distinction between Broca's and Wernicke's aphasia was more appropriately thought of as damage to syntax versus semantics, respectively, rather than damage to motor versus sensory word representations.

In the past 25 years neuroanatomical and behavioral findings have caused difficulties for this more modern synthesis as well as the traditional approach. Mohr et al. (1978) concluded that a circumscribed lesion to Broca's area gave rise to only temporary disruptions of motor speech and other language functions, with the deficits lasting only a few days to a few months. Agrammatism was not a feature of the acute or chronic state for these patients. In order for permanent disruptions of language output and agrammatism to occur, a larger lesion was required involving the insula and other areas of the frontal and parietal opercula (with such lesions often extending well posteriorly, sometimes including the supramarginal gyrus). More recently, in a study of lesion overlap, Dronkers (1996) concluded that the insula was the critical region involved in apraxia of speech, that is, in a disruption of speech motor planning.

Similar problematic findings have been uncovered with regard to the functional role of Wernicke's area. One difficulty is that the definition of Wernicke's area is not so agreed upon as Broca's area (see Wise et al. 2001 for discussion). Assuming the most common definition—the posterior third of the superior temporal gyrus (Damasio 1998)—there is evidence that a lesion restricted to this area does not

give rise to the long-lasting symptom complex termed Wernicke's aphasia and that a wider lesion is needed (Selnes et al. 1983). Also, patients have been reported who have lesions outside of Wernicke's area yet who show the symptoms of Wernicke's aphasia (e.g., Murdoch et al. 1986, Dronkers et al. 2000).

Both the traditional and more recent syndrome-based approaches suffer from the heterogeneity of the deficits exhibited by patients assigned to one of the clinical classifications. For example, the poor comprehension of patients classified as Wernicke's aphasics may arise from a disruption of phoneme identification, a disruption of phonological word forms, or a disruption of semantics (see Howard & Franklin 1988 for discussion). Among Broca's aphasics with agrammatic speech, a substantial proportion does not show syntactic comprehension deficits (Berndt et al. 1996). Group studies that average results across members of the group and contrast these averages across clinical groups or with normal subjects are thus unlikely to provide a solid basis for determining the functional components of language and their organization in the brain. What is needed is a more fine-grained, theory-based analysis of the nature of the language deficits in individual cases (Caramazza & McCloskey 1988, McCloskey 1993). With such a functional analysis, one is in a much better position to determine whether consistent brain/language relationships can be identified.

Consequently, the discussion of neuropsychological data in this chapter focuses on individual case studies in which the precise nature of the patients' deficit has been identified. Lesion localization from a series of cases with the same deficits provides better information than group studies concerning the relation between language function and brain areas. Of course, we now have other sources of information regarding the localization of language function in the brain from neuroimaging studies carried out with intact normal subjects. Results from these studies may provide much more precise information than lesion studies, given the accidental nature of the size and extent of lesions in humans. It remains quite difficult, however, to design imaging studies to isolate specific cognitive functions (see Norris & Wise 2000 for discussion in the domain of speech perception). Patient data provide crucial corroborating evidence regarding the necessity of a given brain region for a particular cognitive function. In some cases unexpected results from neuroimaging have led to studies of patients with lesions to the activated region that have confirmed the localization that had not previously been uncovered in lesion studies (Thompson-Schill et al. 1997, 1998).

WORD PROCESSING

Data from normal subjects and brain-damaged patients have led to the overall model of word processing shown in Figure 1. (The model includes the processes involved in picture naming, as this task is often used to test word production.) The model assumes a separation between the lexical forms involved in spoken and written word processing, but a single semantic system that is accessed from

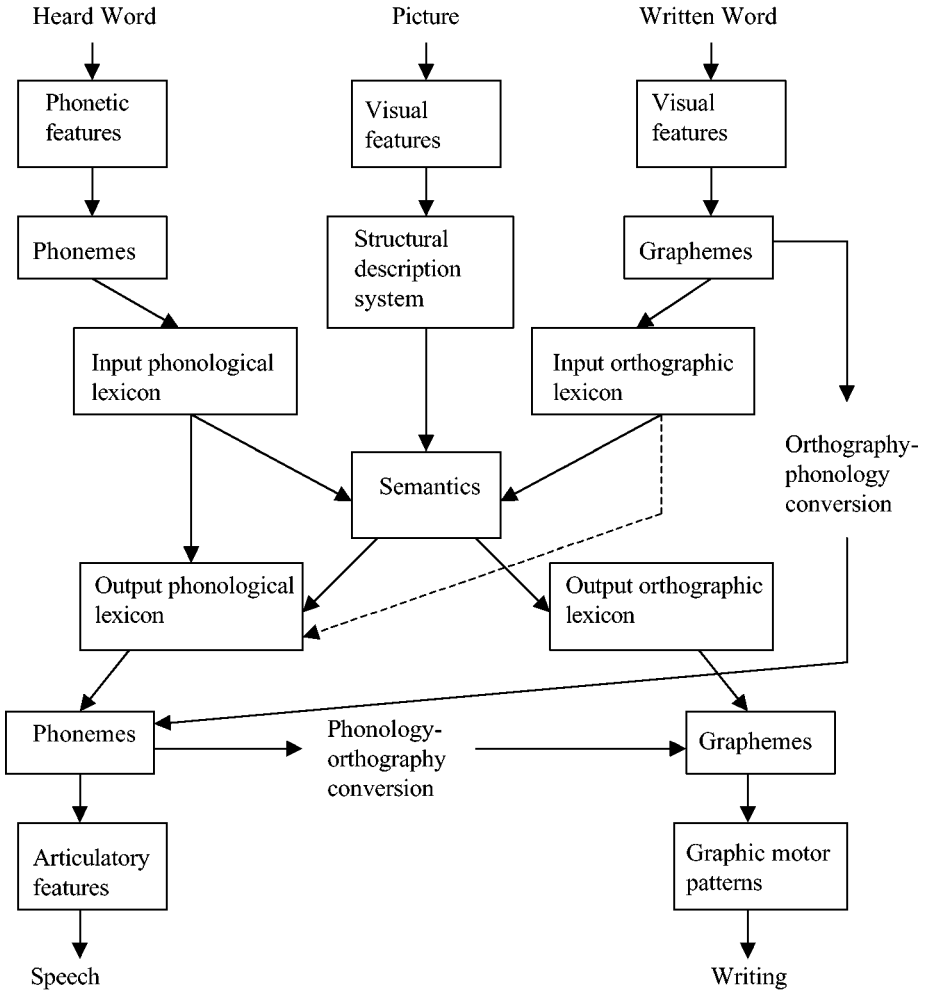

Figure 1 Model of single-word processing for auditory and written word perception and production. Also included are processes involved in picture naming.

both modalities. On both the input and output sides there are peripheral processing components involved in auditory and visual perception and the motor control of speaking and writing. The discussion focuses on the more central aspects of word processing, concentrating mainly on spoken language.

Spoken Word Recognition

NEUROPSYCHOLOGICAL STUDIES As shown in the model, phonetic, phonemic, lexical-phonological, and semantic representations are assumed to be involved in

word recognition, and disruptions at any of these levels should lead to deficits in word comprehension. Disruptions of semantic representations are discussed separately; this section concentrates on phonological aspects of word recognition.

Patients labeled as "pure word deaf" would seem to provide evidence that different systems are involved in speech and nonspeech auditory perception, as they have difficulty perceiving speech but do better with music or environmental sounds. (See Griffiths et al. 1999, Poeppel 2001 for recent reviews). Several researchers have presented evidence that, contrary to the word deaf label, these patients' deficit is not at a lexical or even phonemic level, but rather at the level of extracting the acoustic cues to speech. That is, they have difficulty perceiving rapid changes in complex pitch patterns, which affect the perception of speech more than other common sounds. Consistent with this view, these patients have difficulty discriminating nonspeech sounds that depend on perceiving rapid temporal changes (e.g., Albert & Bear 1957, Auerbach et al. 1982, Tanaka et al. 1987, Wang et al. 2000).

Pure word deaf patients typically have bilateral lesions of the superior temporal lobe, though some have unilateral lesions that, in all but one case, have been localized to the left hemisphere (Griffiths et al. 1999, Poeppel 2001). The bilateral lesions suggest that both hemispheres are involved in extracting phonetic cues to speech (Praamstra et al. 1991). The unilateral lesions have also been argued to be consistent with bilateral processing as these lesions typically include deep subcortical structures and thus could damage both phonetic processing on the left and disconnect the results of such processing on the right from lexical phonological representations on the left (Geschwind 1965, Takahasi et al. 1992, Poeppel 2001).

According to the model in Figure 1, phonetic cues are mapped onto phonemes. Some researchers have argued, however, that phonetic features are mapped directly onto lexical representations, and thus a phonemic level is not needed (Marslen-Wilson & Warren 1994). In fact, it appears that no cases have been reported that show the pattern predicted from a deficit to the phonemic level, that is, impaired vowel and consonant perception but preserved perception of nonspeech stimuli with acoustic features similar to those in speech. In contrast to the lack of evidence for a phonemic level, there is evidence supporting a lexical phonological level separate from semantics. That is, some patients have been reported who show preserved phoneme discrimination and identification but impaired performance on spoken word recognition tasks tapping lexical and semantic levels such as auditory lexical decision and synonymy judgments (Howard & Franklin 1988, Martin & Saffran 1992, Hillis et al. 1999). As with the pure word deaf patients, these patients show much better semantic processing for written words, again ruling out a semantic deficit per se. All of these cases have had unilateral left hemisphere damage. For all, the damage included temporal or parietal regions, but for two the damage was quite widespread and included other left hemisphere regions (Howard & Franklin 1988, Hillis et al. 1999).

Other patients show a preservation of lexical phonological information but disrupted access from this information to semantics. Hall & Riddoch (1997) reported a particularly striking example of this pattern. Their patient, KW, with a left parietal

lesion, performed well on auditory and written lexical decisions. On semantic tasks he performed very poorly with auditory presentation but was within the normal range with written presentation. Franklin et al. (1994, 1996) and Kohn & Friedman (1986) have reported similar cases. Two of these (Franklin et al. 1994, 1996) were reported to have left middle cerebral artery infarcts, which could implicate frontal, parietal, and temporal regions. The case of Kohn & Friedman had damage to Wernicke's area.

In sum, patients with damage either to lexical phonological representations or to the connections between these representations and semantics all have left hemisphere damage, with temporal or parietal regions most often involved. Thus, whereas phonetic feature extraction may be carried out bilaterally, the results of this feature analysis appear to be mapped onto lexical phonological representations in the posterior left hemisphere, which then provide access to semantic representations. Consistent with this conclusion are findings from a study by Hillis et al. (2001), which examined the effects of reperfusion of the left superior temporal lobe in six stroke patients who had word comprehension difficulties and showed hypoperfusion of this region. Using a pharmacological agent to increase blood pressure they demonstrated increases in word comprehension that were directly related to the extent of reperfusion of this region as measured through magnetic resonance perfusion weighted imaging.

Contrary to the tenets of the traditional Wernicke-Lichtheim model, some findings from lesion studies suggest a role for the frontal lobes, specifically Broca's area, in speech perception (Blumstein 1998, 2001; Martin et al. 1999a). Blumstein (1998) reviewed studies showing that aphasic patients of all clinical categories, including Broca's aphasics, have speech perception deficits when tested on phoneme and word discrimination and identification. Functional neuroimaging of neurally intact individuals have also implicated a role for frontal regions in speech perception (see neuroimaging section for discussion).

NEUROIMAGING STUDIES The findings from neuorimaging studies are consistent with the lesion data with regard to bilateral temporal lobe involvement in the extraction of phonetic cues from speech. When passive listening to speech sounds has been compared with rest or listening to simple auditory signals like tones, bilateral superior-temporal lobe activation has been obtained (e.g., Petersen et al. 1988; Zatorre et al. 1992; Binder et al. 1994, 1996, 1997; see Cabeza & Nyberg 2000 for an overview).

Left-lateralized activation would be predicted for phonological processing beyond the level of phonetic feature extraction. In order to reveal such processing, speech stimuli need to be compared to a control condition (or conditions) that employs stimuli with acoustic features like those in speech. The results of such studies have varied depending on the nature of the control condition and the subjects' task. Some studies have contrasted an active speech task, such as phoneme monitoring, with passive listening to speech (Zatorre et al. 1992, Poeppel et al. 1996). Others have contrasted two active listening conditions in which attention

is directed either towards phonological or semantic properties of speech stimuli (Démonet et al. 1992, 1994). More left-lateralized activation is obtained in active conditions. The interpretation of such findings is open to debate, however, as some argue that passive listening or performing a semantic task automatically activates all regions normally engaged in speech processing, and thus, such subtractions would have eliminated relevant cortical areas (Norris & Wise 2000), leaving only task- or strategy-specific activation. Others argue, however, that performing an active task simply boosts activation in the most critical regions because attention is directed to speech-specific processing (Fiez et al. 1995).

Other studies have compared passive listening to speech to passive listening to stimuli with speech-like features (e.g., Binder et al. 2000, Howard et al. 1992). Binder et al. (2000) contrasted the activation resulting from passive listening to words, pseudowords (e.g., splin), reversed words (i.e., recorded speech played backwards), tones, and noise. No areas were found to be more activated in the word condition than the pseudoword or reversed word conditions. All three stimuli produced greater activation bilaterally in the mid–superior temporal gyrus and adjacent superior temporal sulcus than the nonspeech stimuli. There was only weak evidence of more extensive activation in the left than right hemisphere for the speech stimuli.

Wise and colleagues (Scott et al. 2000, Wise et al. 2001), however, have suggested that considerable lexical activation may arise from processing pseudowords (see Gaskell & Marslen-Wilson 1997, McClelland & Elman 1986) or even reversed words, as subjects consistently identify a fairly large proportion of phonemes in reversed speech (Binder et al. 2000). Scott et al. (2000) used as stimuli spoken sentences and altered versions of these sentences that varied in intelligibility and the presence of phonetic features. They reported a region of the left superior temporal sulcus that responded more to the conditions with phonetic information (whether or not they were intelligible) and also found that an anterior portion of this sulcus responded only to intelligible speech. The interpretation of the role of this more anterior region is unclear, however, as the intelligible sentences engaged sentential semantic and syntactic processes in addition to lexical processes.

Activation in frontal regions has also been uncovered in neuroimaging studies of speech perception. Price et al. (1996) reported a complex relation between task factors and inferior frontal activation [Brodmann's area (BA) 45] during speech perception (e.g., the activation was greater for slower rates of presentation of the words). Frontal areas are routinely activated, however, when the neuroimaging study employs a speech perception task that involves active manipulation of phonological information such as phoneme monitoring or phoneme discrimination (e.g., Démonet et al. 1992, 1994; Zatorre et al. 1992). Burton et al. (2000) showed that in two closely matched syllable discrimination tasks, inferior frontal activation was obtained only when the task required segmentation of the initial phoneme from the remainder of the syllable. As Norris & Wise (2000) have pointed out, tasks that require phonemic segmentation cannot be performed by illiterate individuals, yet these individuals have no difficulty perceiving speech. The process of learning letter-sound correspondences during reading acquisition may foster an

awareness of sublexical phonological representations typically involved in speech output (rather than speech perception). Using a task in a neuroimaging study that requires attention to sublexical units may tap these output representations that have a frontal localization.

Spoken Word Production

NEUROPSYCHOLOGICAL STUDIES As with word recognition, deficits in spoken word production could arise from several sources, depending on the affected stage of processing (see Figure 1). The types of errors patients produce in naming (e.g., picture naming or naming to definition) or other speech production tasks (oral reading, repetition) have been used as one source of data for identifying the locus of damage. In naming, patients may produce predominantly semantically related words (e.g., "parsley" for "carrot") (e.g., Hodges et al. 1992), predominantly phonologically related words (e.g., "golf" for "glove") (Blanken 1990), or predominantly phonologically related nonwords (e.g., "brind" for "bread") (e.g., Caplan et al. 1986), which might suggest disruptions at the semantic, lexical phonological, and sublexical phonological levels, respectively.

Even though a certain type of error may predominate, most patients produce a mixture of error types. Dell et al. (1997) attempted to account for the different relative proportions of correct responses and different error types for fluent aphasic patients in terms of disruptions to all levels of an interactive activation model of word production initially derived to account for normal production (Dell & O'Seaghdha 1992). As shown in Figure 2, the model includes semantic, lexical, and phonological levels and feedforward and feedback connections between levels.

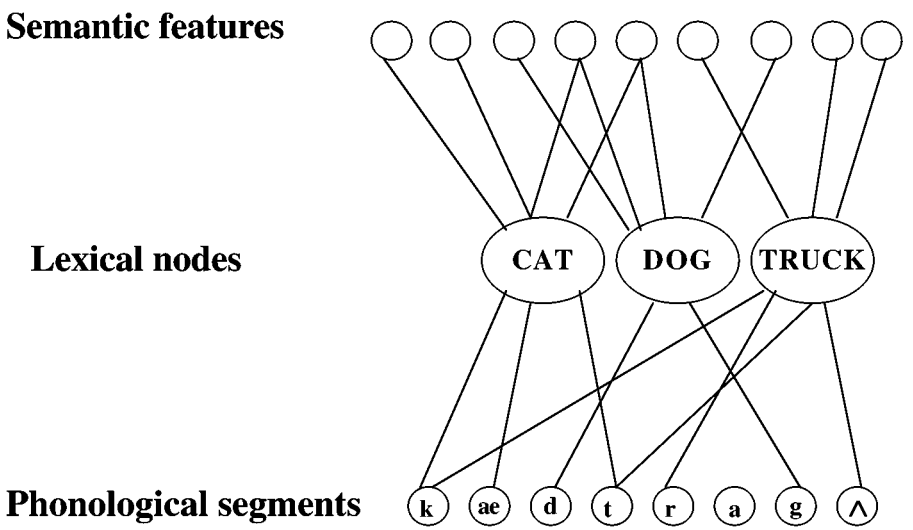

Figure 2 Dell & O'Seaghdha's (1992) model of word production.

In the first version of the model to be applied to patient data (Dell et al. 1997), global "lesions" were made that consisted of reducing connection strengths between all levels and increasing the decay rate at all levels (with changes in the two relevant parameters adjusted to fit individual patient data). Although this approach provided a relatively good fit to the error patterns of their patients, it did not account for patients who produce virtually all semantic errors (Caramazza & Hillis 1990, Cuetos et al. 2000) nor those who produce virtually all phonological errors (Caplan et al. 1986, Caramazza et al. 2000). Foygel & Dell (2000) proposed a revised model in which the globality assumption was abandoned and lesions were made separately to the connections between the semantic and lexical and between the lexical and phonemic levels. Although this approach provided a better fit to a response pattern of predominantly phonological errors and to word repetition data, the interactivity in the model still made it unable to fit a pattern of predominantly semantic errors (Rapp & Goldrick 2000) or one in which phonologically related nonword errors greatly outnumber phonologically related word errors (Caramazza et al. 2000). Thus, the data strongly suggest that selective deficits can occur to different levels in the production process, but the details of models to account for such errors are debated with regard to the degree of interactivity between levels (Ruml et al. 2000, Rapp & Goldrick 2000).

The approach of Dell and colleagues (Dell et al. 1997, Foygel & Dell 2000) used the proportion of different error types in naming to identify the functional locus of damage. Clearly, other data may be brought to bear. Among patients producing predominantly semantic errors, some show a disruption of semantic knowledge on comprehension tests (e.g., Howard & Orchard-Lisle 1984, Hillis et al. 1990, Hodges et al. 1992), whereas others do not (e.g., Caramazza & Hillis 1990, Cuetos et al. 2000). Thus, the former appear to have damage at the semantic level per se, whereas the latter have damage beyond the semantic level, that is, in accessing phonological representations from semantic representations. Another relevant factor in determining the locus of damage is the patients' ability to repeat words and nonwords. Some patients with preserved semantics but difficulty in accessing lexical phonology show preserved word and nonword repetition (e.g., Caramazza & Hillis 1990, Martin et al. 1999b), indicating that their deficit is not at the level of producing sublexical phonological segments. Patients who make phonologically related word or nonword errors in naming typically have difficulty with repetition as well (e.g., Caramazza et al. 1986, Caplan et al. 1986, Bub et al. 1987, Shallice et al. 2000). These cases have sometimes been claimed to have a deficit in a phonological output buffer, that is, a short-term storage system for maintaining phonological information while articulatory programs are derived.

Lesion localization for word production deficits has been addressed most often with respect to the clinical category of "anomic aphasia," which is characterized by pervasive word-finding impairments in spontaneous speech and naming in conjunction with intact repetition and fluent, grammatically correct speech (see Table 1, example 3). Damasio & Damasio (1989, Damasio et al. 1996) have reported that the lesions in anomic aphasia are outside the peri-sylvian region in anterior or

inferior temporal regions. Semantic abilities in comprehension have not typically been extensively assessed for these patients, however, and thus it is difficult to know whether the word-finding difficulties derive from a semantic disruption or from a disruption of lexical phonological retrieval. Some evidence suggests that a left posterior temporal region (BA 37) is crucial for the retrieval of phonological word forms in speech production, as patients with damage to this region have naming deficits even though they show preserved semantic knowledge (Raymer et al. 1997, Foundas et al. 1998, Hillis et al. 2001).

Less attention has been devoted to the issue of lesion localization with regard to sublexical phonological deficits. All but one of the cases described above that were claimed to have deficits to the output phonological buffer had left hemisphere lesions affecting a number of temporal-parietal regions, including cortical and underlying white matter; the remaining case had a similar lesion on the right. Thus, the evidence suggests posterior rather than anterior regions are involved in maintaining sublexical phonological representations.

NEUROIMAGING STUDIES Indefrey and Levelt (2000) reviewed 58 neuroimaging studies that used a variety of tasks involving speech production (e.g., picture naming, word reading, nonword repetition). Based on these data, they argued that the left posterior middle and superior temporal gyri are involved in phonological word-form retrieval, as this area was more activated in tasks involving word than nonword production. For sublexical phonological coding, no region strictly met their criterion for proportion of studies showing significant activation, but the regions coming closest were the left posterior inferior frontal gyrus and the left mid-superior temporal gyrus.

Indefrey & Levelt (2000) identified regions that were active in overt speech tasks compared with silent controls and not active in covert speech tasks as areas involved in articulatory coding and execution. The areas satisfying this criterion were bilateral pre- and postcentral gyri. Also, typically more active in overt than covert tasks were the left anterior superior temporal gyrus, the right supplementary motor area, and the left and medial cerebellum. A recent study by Wise et al. (2001), however, identified an area of the temporo-parietal junction that showed greater activation with overt than covert articulation of a phrase (see also Buchsbaum et al. 2001).

Independent or Common Lexical Phonological Representations?

NEUROPSYCHOLOGICAL STUDIES The model shown in Figure 1 has separate input and output lexical phonological representations. The Wernicke-Lichtheim model assumed separate sensory and motor representations for words, though access to the sensory representation was needed to insure access to the correct motor representation. There is currently considerable debate regarding whether independent input and output lexical representations are needed (Nickels et al. 1997, Martin et al. 1999b) or whether a single lexical phonological representation will suffice

(Hillis et al. 1999, Martin & Saffran 2002). Shallice (1988) argued that the semantic errors in single-word repetition (e.g., repeating "woman" as "lady") that define the syndrome of "deep dysphasia" provide strong support for the independent-lexicons view. These semantic errors indicate that the word has been perceived, so their deficit cannot be in accessing a lexical phonological form on the input side. This phonological form should be the basis of repetition if the form is the same for perception and production. In terms of a two-lexicon model, this pattern can be accounted for by a disruption in direct connections between input and output phonological forms, with the result that translation between input and output involves access to a semantic representation. Martin & Saffran (1992) have argued, however, that this deep dysphasic pattern can be accounted for with a single lexicon model by assuming that, even though the correct phonological form is initially activated, the activation decays very rapidly and the patient has to use whatever semantic information has been activated to reconstruct the phonological form for output (see Howard & Franklin 1990 for evidence counter to the rapid decay account).

NEUROIMAGING STUDIES Although an early positron emission tomography study showed no activation of left posterior temporal areas in word production (Petersen et al. 1989), more recent studies have uncovered similar temporal regions involved in speech perception and production (Indefrey & Levelt 2000). One might question whether exactly the same areas are involved. Anderson et al. (1999) found that intra-cranial cortical stimulation of the left superior temporal lobe caused significant phonological errors in production but did not impair the patient's auditory word comprehension. However, Wise et al. (2001) found in a positron emission tomography imaging study that a region in the left posterior superior temporal sulcus that responded to words in perception was also activated during word production.

It is possible that, in line with Wernicke's original hypothesis, there is a (input) phonological representation involved in both perception and production and a separate output lexical representation involved only in production. According to such a view, it should be possible to find patients with normal word perception but impaired output (Romani 1992, Howard 1995, Martin et al. 1999b), but those showing impairments of lexical phonology on the input side should also show impaired output (Hillis et al. 1999).

Semantic Representations of Words

Recent overviews by Shelton & Caramazza (1999, 2001) have summarized neuropsychological findings on the organization of semantic representations. They have dealt extensively with the nature of category-specific deficits (e.g., semantic deficits for living but not nonliving things), concerning their underlying cause and possible neuroanatomical basis, but these are not dealt with here (see also Devlin et al. 2002). Considerable lesion data suggest that regions of the middle and inferior temporal lobe are involved in the representation of semantic knowledge (Dronkers

et al. 1995). Semantic dementia cases (i.e., patients whose primary symptom is a progressively severe disruption of semantic knowledge) have damage to the left middle and inferior temporal lobes that begins at the temporal pole and proceeds posteriorly as the condition progresses (Mummery et al. 2000). Mummery et al. also found that the degree of temporal damage predicted the degree of semantic disruption. Neuroimaging studies have also uncovered left middle and inferior temporal lobe activations during semantic processing (e.g., Vandenberghe et al. 1996, Binder et al. 1997). The extent of right hemisphere involvement is unclear. Mummery et al. (2000) noted atrophy in the right temporal pole in most of their semantic dementia cases and suggested that bilateral damage was necessary for the most severe semantic deficits.

A surprising outcome of neuroimaging studies has been that robust activations appear in left inferior frontal regions (BA 45 and 47) across numerous tasks such as categorization, semantic generation, concrete/abstract judgments, and judgments of semantic similarity (see Poldrack et al. 1999 for a review) that can be differentiated from the more posterior frontal activations observed during phonological processing tasks such as phoneme monitoring or rhyme judgments (Poldrack et al. 1999, Roskies et al. 2001, McDermott et al. 2002). These frontal activations are surprising in that patients with damage to these regions do not typically show severe semantic deficits, at least on single-word processing tasks. The explanation offered for these findings is that whereas semantic knowledge may be stored posteriorly, these frontal areas serve as a semantic executive system involved in retrieving, storing, and manipulating these semantic representations (Roskies et al. 2001). Consistent with this claim are findings from patients with inferior frontal damage who show good semantic processing of single words but a short-term memory deficit specific to the retention of semantic information (Romani & Martin 1999, Freedman & Martin 2001).

Grammatical Representations of Words

WORD-CLASS EFFECTS Words contain grammatical information as well as semantic and phonological information. For example, word-class information (noun, verb) is needed in sentence comprehension and production for syntactic information to be understood or produced correctly. Many studies have reported double dissociations in the production of different word classes [e.g., function words versus content words (Goodglass 1993), nouns versus verbs (e.g., Miceli et al. 1984; Zingeser & Berndt 1988, 1990)] (see Druks 2002 for a review). For some patients these apparent word-class effects may have a semantic basis. For example, better production of nouns than verbs and better production of verbs than function words may occur because the patient is better able to produce more concrete words (e.g., Bird et al. 2002). For some patients, however, it appears that word-class effects cannot be reduced to a semantic basis (Berndt et al. 2002, Rapp & Caramazza 2002). Consequently, these deficits suggest that at some level in the production system words are distinguished neurally with regard to the grammatical role they play in a sentence.

Some models of word production assume two levels of lexical representations: a semantic/syntactic representation (termed the lemma) and a phonological representation (termed the lexeme), with the lemma necessarily accessed prior to the lexeme (Garrett 1993, Levelt et al. 1999). Taken as support for these two levels are findings from patients who in picture naming could correctly judge the grammatical gender of a target word (Badecker et al. 1995) or whether the word fits into count or mass phrasal contexts (Vigliocco et al. 1999), even though they were unable to retrieve any of the phonemes in the word. Others researchers have argued that these findings can be accommodated in a model assuming a single lexical representation that is phonological and that connects to general syntactic properties for words (Caramazza & Miozzo 1997). The two positions appear difficult to distinguish on the basis of the available data (Nickels 2001).

MORPHOLOGICAL PROCESSING Researchers have debated whether morphologically complex words (e.g., "worked," "teacher," "undecided") are broken down into their separate morphemes in language production and comprehension (e.g., Allen & Badecker 1999) or whether they are treated as unitary words (e.g., Bybee 1988). Many aphasic patients produce inflectional errors in speech and oral reading (e.g., producing "welds" for "welding" or "teaching" for "teacher"), which would appear to support a decompositional view of morphological processing (Allen & Badecker 1999). Such apparently inflectional or derivational errors might be attributed, however, to whole-word confusions based on phonological and semantic similarity (see Funnell 1987). Some findings argue against such an interpretation (Badecker & Caramazza 1991, Shapiro et al. 2000, Tsapkini et al. 2002). For instance, some of these patients make morphological errors only on words of one word class, that is, either nouns (Shapiro et al. 2000) or verbs (Tsapkini et al. 2000), which would not be expected if the errors were due solely to semantic and phonological similarity of the error to the target.

An issue that has recently attracted a great deal of debate is whether one or two systems are involved in computing past tense forms for regular versus irregular verbs. According to standard linguistic formulations, regular past tense forms in English (e.g., talked, gazed, needed) are computed by a rule that adds a past tense marker /d/ to verbs, with the pronunciation depending on the final phoneme of the verb. Irregular past tense forms must be retrieved from memory, as the pronunciations are not predictable from the present tense forms (e.g., "is, was"; "run, ran"). Because novel words (i.e., pseudowords such as "plag" or "spuff") have no representation in memory, the regular rule should apply to these as well. In opposition to the dual systems view, some have argued that a single connectionist system supports computation of the past tense for regular and irregular forms and for novel forms (e.g., Rumelhart & McClelland 1986).

Ullman et al. (1997) presented evidence supporting the dual systems approach. Patient groups with posterior damage (posterior aphasia, Alzheimer's disease) had difficulty producing irregular but not regular past tense forms for real words and novel words, whereas patients with frontal or basal ganglia damage (i.e., anterior

aphasics or Parkinson's patients) showed the reverse pattern. Ullman et al. argued that these results were due to general properties of posterior versus frontal systems in which posterior regions support declarative memory-based representations, whereas frontal/basal ganglia regions support procedural or rule-based knowledge. On the other side of the issue, Joanisse & Seidenberg (1999) and Patterson et al. (2001) have provided computational and empirical evidence for a single system approach. They argue that the observed double dissociation in the patient data derives from other factors, specifically, semantic deficits in patients with posterior damage (Patterson et al. 2001) and phonological deficits in patients with frontal damage (Patterson 2002). Tyler et al. (2002) argued, however, that a phonological deficit cannot account for anterior aphasics' difficulty with regular forms, as the anterior aphasics they tested who showed worse performance on regular than irregular verb inflection performed quite well on various phoneme discrimination tasks.

It should be noted that the plausibility of the claim that frontal brain regions support rule-based grammatical processing hinges in part on claims that anterior aphasics have difficulty with such processes in comprehension. As discussed in the next section, it is far from clear that this is the case.

SENTENCE PROCESSING

Sentence Comprehension

DISSOCIATIONS AMONG SYNTACTIC ABILITIES As mentioned in the introduction, proposals from the 1970s and 1980s about a general syntactic deficit in Broca's aphasia were undermined by several lines of evidence: for one, the not infrequent co-occurrence of agrammatic speech and preserved syntactic comprehension (Berndt et al. 1996). In addition, several studies demonstrated that many patients who showed asyntactic comprehension on sentence-picture matching did well on judging the grammatical acceptability of sentences (Linebarger et al. 1983, Shankweiler et al. 1989, Wulfeck 1988). Included among the types of errors the patients could detect were the omission or substitution of function words and inflections—the elements most affected in their speech. Linebarger and colleagues (Linebarger et al. 1983, Linebarger 1990, Saffran & Schwartz 1988) argued that these findings demonstrated a dissociation between two aspects of sentence-level processing: determining the grammatical structure of the sentence and the mapping between grammatical and thematic roles, with only the latter being affected in these patients. That is, for example, these patients would be able to determine that "The truck that the car splashed was green" is a grammatical sentence and that "car" is the grammatical subject and "truck" the grammatical object of "splashed." They would be unable to determine, however, that "car" should be mapped as the agent of "splashed" and "truck" as the theme (i.e., the entity acted upon). Thus, they would fail on all tasks requiring the interpretation of sentence meaning, such as sentence-picture matching, enactment (acting out the action with toy objects),

or sentence anomaly judgments (e.g., determining that "The quarterback that the football threw was old" was a nonsensical sentence). They would succeed if the task tapped only the determination of acceptable structure, even for sentences with complex structures, such as judging as ungrammatical a sentence like "The cake that the boy ate the pie was delicious" (Linebarger 1990).

INDEPENDENCE OF SEMANTIC AND SYNTACTIC REPRESENTATIONS Whereas in general agrammatic speakers do not provide the clearest evidence of a dissociation between the processing of syntax and semantics in comprehension, some patients do provide such evidence. Ostrin & Tyler (1995) reported a patient (JG) who performed poorly on sentence-picture matching when the distractor picture depicted a reversal of role relations but performed well when the distractor included a lexical substitution. Unlike the patients reported by Linebarger et al. (1983), however, this patient performed poorly on grammaticality judgment tasks and showed no sensitivity to violations of grammatical structure in a word-detection task. JG performed well on comprehension tests for single words and showed normal semantic priming. Although classified behaviorally as a Broca's aphasic, JG's lesion was left temporo-parietal.

Some patients with Alzheimer's dementia demonstrate the opposite dissociation of very impaired knowledge of word meanings but preserved grammatical knowledge. For example, they might be unable to realize that a phrase such as "The jeeps walked" is nonsensical but yet be able to detect the grammatical error in a phrase like "The jeeps goes" (Hodges et al. 1994). Two semantic dementia patients have been reported who showed a remarkable ability to understand grammatical structure and appropriately assign thematic roles such as agent or theme to complex constructions like "It was the tiger that the lion bit" even though they could not distinguish the meaning of the nouns in the sentence (Schwartz & Chawluk 1990, Breedin & Saffran 1999). Semantic dementia cases such as these typically have diffuse frontal and temporal damage, and their temporal lobe damage presumably underlies their inability to comprehend the nouns in the sentences (Mummery et al. 2000). It is difficult, though, to speculate about which preserved brain areas subserve their spared grammatical processing.

Although the neuropsychological findings indicate that semantic and syntactic knowledge may be independently represented, findings from normal subjects indicate that during sentence processing the two sources of knowledge interact in determining sentence interpretation (Boland 1997, Trueswell et al. 1994). Some patient data are consistent with this conclusion. Saffran et al. (1998) showed that some patients may use the grammatical structure of sentences during comprehension when there are weak semantic constraints (e.g., understanding that "boy" is the agent of "pushed" in "The boy that the girl pushed . . ."), but fail to use the grammatical structure when there are strong semantic constraints (e.g., mistakenly interpreting the "woman" as the agent of "spanked" in "The woman that the child spanked . . .") (see also Tyler 1989).

WORKING MEMORY AND SENTENCE COMPREHENSION Theories of comprehension often assume a role for a short-term or working memory system that is used to hold partial results of comprehension processes while the rest of a sentence is processed and integrated with earlier parts (e.g., Just & Carpenter 1992). Aphasic patients often have very restricted short-term memory spans, typically being able to recall only two or three words from a list, compared with normal subjects' five or six words (de Renzi & Nichelli 1975). Studies with normal subjects indicate that comprehension becomes more difficult when the working memory demands of sentence processing increase (Just & Carpenter 1992, Gibson 1998). Thus, one might hypothesize that patients' restricted memory span is the source of observed sentence comprehension difficulties. However, a number of studies have shown that patients with very restricted memory spans may show excellent sentence comprehension even for sentences with complex syntactic structures (Butterworth et al. 1986, Caplan & Waters 1999, Hanten & Martin 2001, Waters et al. 1991).

Caplan & Waters (1999; see also Caplan & Hildebrandt 1988) have interpreted such findings to indicate that there is a working memory capacity specific to sentence processing that is different from that tapped by span tasks. They divide the procedures involved in sentence processing into interpretive and post-interpretive processes. Interpretive processes include all on-line syntactic and semantic processes, including those involved in semantic interpretation based on the ongoing discourse. Post-interpretive processes involve using the products of interpretive processing to carry out some task, such as sentence-picture matching or enactment of the action in the sentence. Based on a large number of findings with normal and brain-damaged subjects, Caplan & Waters argue that interpretive processing draws on the capacity specific to sentence processing, whereas post-interpretive processing draws on the capacity tapped by span tasks.

Martin and colleagues (Hanten & Martin 2000; Martin et al. 1994, 1999b; Martin & Romani 1994) have provided a different view on the relation between the capacities involved in span tasks and sentence processing. They argue that span tasks tap both phonological and semantic retention (see also Martin & Saffran 1997). The phonological component of span tasks is independent of the capacity involved in sentence processing, as patients with difficulty retaining phonological information may show preserved sentence comprehension. However, the semantic component does play a role in sentence comprehension in the maintenance of word meanings prior to their integration with other word meanings. Specifically, patients with a semantic retention deficit had difficulty detecting the semantic anomaly in sentences with several adjectives preceding a noun (e.g., "The rusty old red swimsuit") or with several nouns preceding a verb (e.g., "Rocks, trees, and shrubs grew in the back yard") but did better when the adjectives followed the noun or the nouns followed the verb. Martin and colleagues agree with Caplan & Waters (1999) to some extent, as they argue that the retention of specifically syntactic structural information is independent of both phonological and semantic capacities (Martin & Romani 1994).

NEUROANATOMICAL BASIS OF SENTENCE COMPREHENSION

Lesion studies Although many studies investigating syntactic comprehension deficits have focused on Broca's aphasics, several studies have demonstrated a similar pattern of increasing comprehension difficulty with increasing syntactic complexity for patients falling into other syndrome categories or having lesions restricted to posterior regions (Naeser et al. 1987, Caplan & Hildebrandt 1988, Caplan et al. 1996). Dronkers et al. (1994) found that among Broca's aphasics, those who had lesions affecting a portion of the left anterior temporal lobe had difficulty computing sentence meaning based on syntactic information, whereas those with a lesion restricted to Broca's area did not. Dronkers & Larsen (2001) state, however, that lesions restricted to this temporal lobe region did not result in this comprehension deficit. Moreover, some of the patients in the Caplan et al. (1996) study who had syntactic comprehension deficits did not have lesions affecting this region. Several researchers have suggested that a complex system of brain regions underlies syntactic aspects of comprehension (Caplan et al. 1996, Dronkers & Larsen 2001, Dick et al. 2001) and that the degree of damage to the overall system predicts the degree of comprehension deficit. Although this may be the case, there are many aspects to syntactic processing (e.g., the assignment of hierarchical structure, the assignment of thematic roles, the processing of long-distance relations, the maintenance of working memory representations), and the failure of any of these could lead to difficulty with more complex constructions (Martin 1995). Individual cases need to be studied in enough detail to specify the nature of the deficit in order to determine if subcomponents of syntactic processing map onto more precise brain regions.

Neuroimaging studies As with the lesion data, there is no clear-cut conclusion emerging from neuroimaging studies regarding the brain regions underlying syntactic processing. The patient data imply that different brain regions are involved in determining syntactic structure and using that structure to assign thematic roles. Neuroimaging studies have typically employed tasks that emphasize one or the other, but without consideration of the implications of these methodological differences. Another methodological variation is the use of passive listening versus active tasks, with the attendant concerns, as in the word-comprehension literature, of whether the passive tasks do not sufficiently engage the subject to reveal activation of all critical brain regions and whether the active tasks introduce activation specific to task demands.

One approach to isolating syntactic from semantic processing has been to use sentence materials in which content words have been replaced with pseudowords (e.g., "The blives semble on the plim") or, less often, with semantically anomalous words (e.g., "The kitchens march on the clouds"). In a passive listening study, Mazoyer et al. (1993) found that the temporal poles were activated bilaterally in their normal prose and syntactic conditions (which employed both of these manipulations) but not in their word-list condition. Two recent studies

involving active processing of sentences with pseudowords required judgments of whether the stimuli had syntactic structure (Friederici et al. 2000) or were grammatically acceptable (Moro et al. 2001). Both studies found deep inferior frontal activation bilaterally for the pseudoword relative to control conditions. Importantly, however, Friederici et al. did not find such frontal activation when subjects made the same judgment about normal sentences. It is possible that the frontal activations with pseudoword stimuli derived from the difficulty in maintaining these stimuli in short-term memory long enough to extract grammatical information.

Another approach to isolating syntactic processing has been to use sentences with semantically appropriate words and active tasks that stress either syntactic processing (e.g., grammaticality judgments) or semantic processing (e.g., semantic acceptability). Two studies taking this approach failed to find regions that were selectively activated by their syntactic conditions (Ni et al. 2000, Kuperberg et al. 2000), though both found regions selectively activated by their semantic conditions. Of course, one might argue that the semantic conditions necessarily engage syntactic processing, and thus subtracting the semantic from the syntactic conditions takes out activation owing to syntactic processing. Dapretto & Bookheimer (1999) took a somewhat similar approach and found greater activation in Broca's area (left BA 44) for their syntactic condition and greater activation in a more anterior frontal region (left BA 47) in their semantic condition. This study, unlike the other two, required the determination of sentence meaning in the syntactic condition (e.g., determining that "The teacher was outsmarted by the student" meant the same as "The student outsmarted the teacher").

A third approach has been to determine the brain regions that show greater activation with increasing syntactic complexity of the stimuli. In a passive listening study, Stowe et al. (1998) found that the left posterior and middle temporal gyrus showed increasing activation with more complex structures. Their study did not, however, control for the specific words in the sentences and sentence length in the different conditions. Other studies have used active tasks in which subjects judge semantic acceptability or answer comprehension questions for different sentence types that are closely matched in length and content words but vary in syntactic complexity. Structures such as the following have been compared:

1. Center-embedded subject relative: The child that spilled the juice stained the rug.
2. Center-embedded object relative: The juice that the child spilled stained the rug.

Numerous studies with normal subjects have demonstrated that the object-relative structure is more difficult than the subject-relative structure and that a contributing factor is the memory demand in the object-relative form involved in reactivating the head noun following the embedded clause verb (e.g., "juice" following "spilled") after having processed the embedded clause subject (e.g., "child") (see Gibson 1998 for discussion).

Taking this approach, Just et al. (1996) found that activation increased with increasing sentence difficulty in the left inferior frontal gyrus and the left middle- and superior temporal gyri. Homologous areas on the right also showed increasing activation, but the overall level of activation was smaller. In contrast, Stromswold et al. (1996) found that only a region within Broca's area was more active for their more complex structure. Caplan et al. (1998) replicated this finding of greater activation in (or near) Broca's area but also showed greater activation for the more complex structures in a left medial frontal area and in the cingulate. More recently, Caplan (2001) reported some additional replications but also some failures to replicate activation in Broca's area in studies using similar sentence contrasts. In one study using elderly subjects and another using young subjects matched in education to the elderly subjects, activation was obtained in parietal and superior frontal areas, though the exact locations were different for the two groups. In an event-related design using young subjects Caplan et al. (2001) uncovered activation in the angular gyrus and a superior temporal region. Caplan et al. have suggested that one possible source of this variation across studies is the varying syntactic proficiencies of their subjects and the varying difficulty of the tasks assigned the subjects (i.e., whole sentence reading versus word-by-word reading).

It is perhaps premature to draw general conclusions about the brain regions underlying syntactic processing until a better understanding has been obtained about the effects of different tasks, different types of materials, different presentation modes, and different subject groups. It is the case, however, that inferior frontal activation has been observed in Brodmann's areas (BAs) 44 and 45 across several studies (though not all) that have required subjects to compute a meaning representation for complex structures. To interpret this activation one has to keep in mind that patients with damage to this region do not typically have difficulty with grammaticality judgments. Thus, this region should not be interpreted as one involved in assigning syntactic structure to a sentence. Instead, this region may be involved in semantic working-memory functions such as those related to reactivating a semantic representation for an earlier occurring noun that has to be linked to a later occurring position in a sentence (as in the object-relative constructions) (see Caplan et al. 1998, Stowe et al. 1998 for related discussion). Such an effect related to semantic retrieval and manipulation would be consistent with findings showing activation in this region for various types of semantic processing tasks (Poldrack et al. 1999).

SENTENCE PRODUCTION

Patterns of Sentence Production Deficits in Aphasia: Challenges to Traditional Claims

Sentence production deficits have also been a focus of research in aphasia, although in this domain much of the work originated from a syndrome-based approach, concentrating on Broca's aphasics who produce agrammatic speech, that is, speech

marked by simplified grammatical form and the omission of function words and inflections. Fluent speakers are said to produce "paragrammatic speech," which is characterized by the substitution of inappropriate function words and inflectional morphemes rather than their omission. Some researchers attributed these differences to a syntactic deficit in Broca's aphasics and a word retrieval deficit in fluent speakers that affects both content and function words (see Berndt 2001 for discusssion). As in the study of comprehension, more recent findings have challenged these claims concerning the differences between nonfluent and fluent speakers. Cross-linguistic studies of aphasia suggest that Broca's aphasics tend to produce a default form of a verb, which in English tends to be an infinitive form (i.e., without inflection), whereas in other languages this will be an inflected form (Menn 2001). Wernicke's aphasics also tend to over-produce verb forms that occur frequently in their native language but produce a wider range of options. Both omissions and substitutions of function words have been documented in detailed analyses of agrammatic (Miceli et al. 1989) and paragrammatic (Butterworth & Howard 1987) speech (see also Haarmann & Kolk 1992). Both nonfluent and fluent speakers show reduced structural complexity, with the deficit taking similar forms across the two groups (Bird & Franklin 1996).

Moreover, sentence-structure and function-word difficulties have been found to dissociate, arguing against the claim that both derive from the same syntactic deficit. Some patients demonstrate reduced sentence complexity but accurate production of function words and inflections (Bird & Franklin 1995/1996, Saffran et al. 1989), whereas others show the reverse (Miceli et al. 1983, Kolk et al. 1985, Nespoulous et al. 1988, Nadeau & Rothi 1992). A patient reported by Thompson et al. (2002) showed a particularly striking dissociation in this regard, as she produced complex syntactic structures as often as normal subjects but made many errors in the production of inflectional morphemes, though she was able to produce function words appropriately. As this indicates, the relative proportion of function words versus inflectional errors can vary substantially across patients, as can other properties of these errors such as the proportion of omissions versus substitutions and the relative difficulty with different kinds of function words (e.g., determiners versus auxiliary verbs) (Miceli et al. 1989).

THEORETICAL ACCOUNTS OF SENTENCE-PRODUCTION DISORDERS These similarities in grammatical deficits across syndromes and variations within syndromes imply that, as in the study of comprehension, a more fruitful approach than group comparisons would be the study of individual cases with respect to their implications for theory. Figure 3 shows a standard model of language production. The model was presented by Bock & Levelt (1994) and represents a somewhat modified version of a theory initially proposed by Garrett (1980). At the top level is a nonlinguistic representation of the message to be expressed. At the next level lexical-semantic forms are accessed and the functional relations among them are spelled out (e.g., grammatical roles of nouns with respect to verb, modification relations between adjectives and nouns). At the next level syntactic

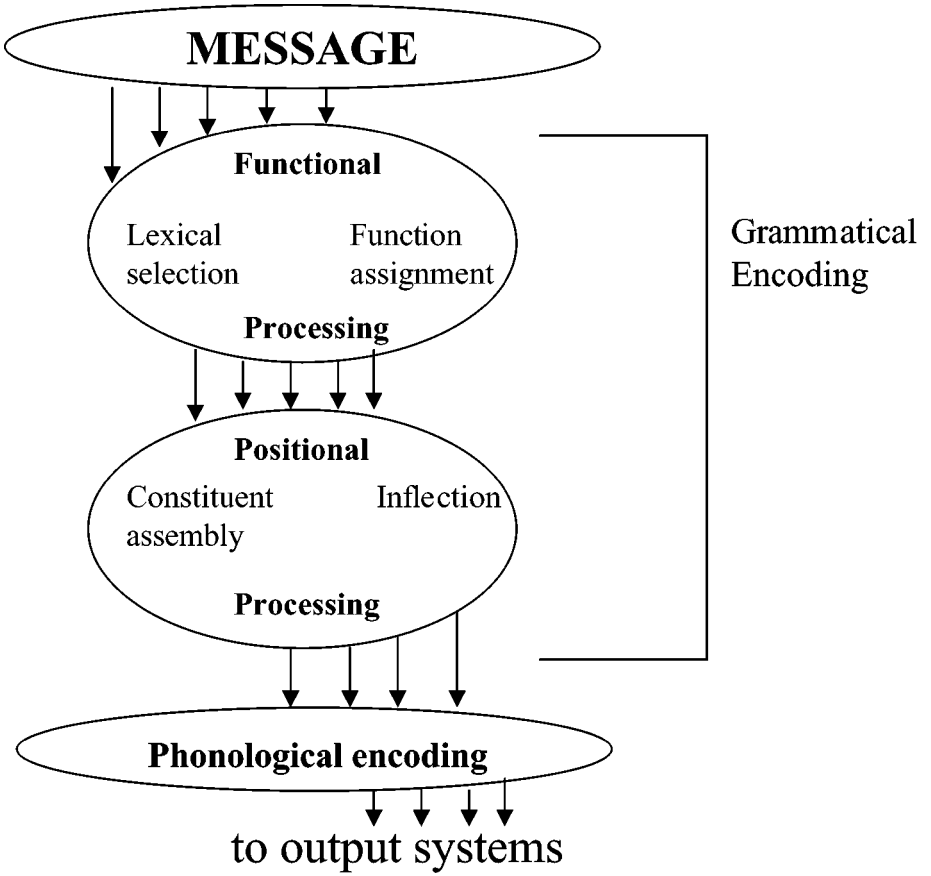

Figure 3 Bock & Levelt's (1994) model of sentence production.

frames are chosen to express the functional relations, and lexical phonological forms are inserted into the frame. At this point a linear ordering of the words is developed, and function words and grammatical markers are inserted at appropriate points. At the next stage the phonetic representation of the utterance is specified.

As the model postulates two levels of grammatical representation (the functional and positional levels), one might hypothesize that patients with grammatical deficits in production could have selective damage to one of these two components. At each level, however, there are various representations and processes involved, and thus, different types of deficits might appear from damage to the same level. At the functional level one major component involves mapping of the relations between the verbs and nouns that play thematic roles with respect to the verb (Bock & Levelt 1994). The specific verb to be used dictates what grammatical

role a noun with a specific thematic role will play (e.g., the recipient will be the subject of an active sentence using the verb "receive" but the indirect object of an active sentence using "give"). A deficit in knowledge of the relations of thematic and grammatical roles entailed by verbs could potentially lead to an incomplete specification at the functional level and a reduction in sentence structure, such as the failure to produce a required indirect object (Saffran et al. 1980). Berndt et al. (1997) found some support for this notion in a study of 10 patients who varied in their relative ability to produce nouns and verbs in single-word tasks. They found a strong relation between greater difficulty with verbs than nouns and impairments on structural measures of spontaneous speech such as mean sentence length, sentence elaboration with content words, and proportion of words in sentences. Two of the five verb-impaired patients showed a much greater proportion of grammatically acceptable sentences and greater production of the object noun when asked to generate a sentence using an experimenter-provided verb compared with a noun.

Another type of production deficit has also been attributed to a disruption at the functional level. Martin & Blossom-Stach (1986) and Caramazza & Miceli (1991) reported case studies of patients who produced appropriate nouns and verbs but often reversed the roles of the nouns with respect to the verb (e.g., saying "The boy was pushed by the girl" for a picture of a boy pushing a girl). They argued that these patients had difficulty with the mapping between thematic roles and grammatical roles at the functional level (i.e., they had a mapping deficit on the production side). Interestingly, these patients produced otherwise grammatically correct utterances, indicating that the other stages in the production process were executed appropriately. Also, both patients showed similar mapping deficits in comprehension, suggesting that a central process subserves the mapping between grammatical and thematic roles in both production and comprehension. Whereas these patients showed a general mapping deficit, other patients have demonstrated difficulties that appear limited to certain verbs, specifically those for which there exists a closely related verb with different mapping relations (e.g., give-take, buy-sell) (Breedin & Martin 1996, Byng 1988).

A disruption at the positional level could also take different forms. Difficulty with accessing syntactic frames could be another possible locus of reduced structural complexity (Goodglass et al. 1994), as patients might be able to access only the simplest, most frequent structural frames (Stemberger 1984). Also, Garrett (1980) claimed that function words and inflectional markers are part of the syntactic frame. If so, difficulty with accessing frames would lead to errors involving such elements. Caramazza & Hillis (1989) endorsed Garrett's approach in their interpretation of findings from a patient who omitted function words and inflections on sentence production tasks but who had no difficulty with functions words on single-word tasks. They pointed out, however, that Garrett's model is underspecified with regard to accounting for aspects of their patient's behavior (e.g., the greater proportion of omissions of function words than inflections) and the variation in substitutions versus omissions of function words across different patients.

Other theorists have argued that grammatical elements are retrieved in the same fashion as content words (Bock 1989). If so, some other means of explaining disorders of function words and inflectional markers would have to be proposed. Various hypotheses have been advocated: (*a*) a disruption of function words and grammatical markers per se, which constitute a specialized word class (Bradley et al. 1980); (*b*) a syntactic deficit that prevents determination of the correct function word or inflection to express grammatical relations (Thompson et al. 2002); (*c*) a deficit in accessing all low imageability words (Bird et al. 2002); (*d*) a phonological disturbance that affects late stages of the production process, during which the phonological forms of function words and inflections are determined (Saffran et al. 1980). Of course, these hypotheses would not have to be mutually exclusive; that is, the different patterns of function-word deficits for different patients may have different underlying causes.

WORKING MEMORY DEFICITS IN PRODUCTION Although the role of working memory in comprehension has been studied extensively, relatively little attention has been paid to its role in production. A model like that in Figure 3 would imply that working memory capacities of various types are involved, as the representations for several words would have to be activated and maintained simultaneously at different stages in the process. This maintenance would have to persist long enough for the processes at the next stage to be carried out.

Kolk (1995) has hypothesized that aphasic language production patterns derive from a disruption of temporal coordination during syntactic planning. He proposes that at the positional level both content words and function words are inserted into a syntactic frame. Difficulties arise owing to slow activation or overly rapid decay of some elements that should be expressed in the same syntactic unit. The more complex the syntactic structure, the more elements to be realized and the more likely some temporal mismatch will occur between elements. Patients can adapt to this deficit by either using simpler structures or sticking with more complex structures but omitting some elements. In support of this view, Kolk and his colleagues have shown that patients' production patterns can appear quite different under different task demands (Kolk & Heeschen 1992, Hofstede & Kolk 1994).

Martin & Freedman (2001) presented evidence that a semantic short-term memory limitation can affect patients' productions. Patients with a semantic retention deficit (but not those with a phonological retention deficit) had difficulty producing adjective-noun phrases (e.g., "small leaf," or "short dark hair"), although they were able to produce the individual adjectives and nouns in isolation. They argued that subjects plan speech in a phrase-by-phrase fashion, planning the head noun and all the preceding content words in the phrase at a lexical-semantic level prior to initiating phonological retrieval. These patients' lexical-semantic retention deficits prevented them from simultaneously maintaining such representations for several words. In support of this argument Martin & Freedman showed that these patients did better producing the same content in a sentence form (e.g., "The leaf is small" or "The hair is short and dark"), as these sentence forms allowed them to

produce fewer content words in an individual phrase (see also Linebarger et al. 2000 for findings supporting a capacity limitation as a factor in at least the structural limitations of agrammatic speech).

NEUROANATOMICAL BASIS OF SENTENCE PRODUCTION

Lesion studies As discussed earlier, Mohr et al. (1978) found that patients with a lesion restricted to Broca's area did not produce agrammatic speech. Larger lesions of the frontal and parietal opercula and the insula were required. The patient reported by Caramazza & Hillis (1989), who omitted a large proportion of function words, was left-handed and had a right frontal-parietal lesion. At least some types of grammatical deficits in production appear to derive from posterior lesions. The patients who had relatively preserved structural abilities in production but impaired production of function words (Miceli et al. 1983, Kolk et al. 1985, Nespoulous et al. 1988, Nadeau & Rothi 1992) had temporal or parietal lesions. The two patients reported above who showed a disruption in the assignment of thematic roles but preserved production and comprehension of grammatical morphemes had parietal (Caramazza & Miceli 1991) and temporo-parietal (Martin & Blossom-Stach 1986) lesions.

With regard to working memory deficits, the three cases reported by Martin & Freedman (2001) with lexical-semantic short-term memory deficits and disrupted production of adjective-noun phrases had lesions that overlapped in the left posterior inferior frontal gyrus and adjacent anterior parietal region, though one had an extensive lesion affecting temporal regions as well.

Neuroimaging studies So far, only two neuroimaging studies have assessed production beyond the single word. In one of these, subjects described Rorschach inkblots in a spontaneous fashion while being scanned (Kircher et al. 2000). The rate of word production per 20-second interval was measured. Speech rate was positively correlated with the degree of signal change in the left superior temporal lobe and supramarginal gyrus. The authors attributed this activation to lexical retrieval, though the degree of syntactic, prosodic, and phonological planning would also vary with speech rate.

Indefrey et al. (2001) used a more controlled production task to determine the regions involved in syntactic processing during production. Subjects saw animated scenes involving the movement of simple colored objects and described them in a prespecified fashion using either full sentences (e.g., "The red square launches the blue ellipse"), a sequence of phrases with local but not sentence-level syntactic structure (e.g., "red square" "blue ellipse" "launch"), or a sequence of words without syntactic structure (e.g., "square" "red" "ellipse" "blue" "launch"). A frontal region that overlapped Broca's area (BA 44) to some extent but was mainly posterior to Broca's area (most likely in BA 6) was found to be most activated in the sentence condition, next most active in the phrase condition and least in the word condition. Although the degree of prosodic planning and the amount of phonological material produced were not equated across conditions, the authors concluded

that this activation reflected syntactic planning, as a faster rate of stimulus presentation did not produce greater activation in this region. It should be noted, however, that according to the hypotheses of Martin & Freedman (2001), the demands on semantic short-term memory would also vary across conditions and could be the source of the frontal activation.

CONCLUSIONS

Since Wernicke's time, a great deal has been learned from neuropsychological studies about the functional organization of the language system. The study of word processing has moved from a consideration of only sensory and motor representations to the development of models containing levels of phonological, grammatical, and semantic representations. Studies of sentence comprehension have uncovered different components of grammatical processing and semantic integration. Progress in the study of sentence production has also been made, though here much more specific theoretical proposals are needed, as observed deficits can often be attributed to several different potential sources, and many details remain without explanation.

With regard to the neural systems underlying these complex processes, some progress has been made, though clearly much remains to be done. Some claims are well supported by both neuroimaging and lesion data: (*a*) Bilateral superior temporal lobes are involved in extracting the phonetic features of speech; (*b*) lexical phonological representations are represented in left posterior regions, most likely in the superior temporal lobe or sulcus; (*c*) semantic representations are localized in the left middle and inferior temporal gyri, with evidence that similar structures on the right may also be involved. Other claims have some support but need further verification, such as: (*a*) A left posterior temporal region (BA 37) is involved in linking semantic and phonological representations in word production; (*b*) left inferior frontal areas are involved in phoneme segmentation; (*c*) more anterior left inferior frontal regions are involved in semantic manipulation and retention. The localization of sentence-level processes seems much less clear based on both lesion and imaging data. Although some researchers seem committed to the notion that Broca's area is involved in syntactic processes in production and comprehension, there is much evidence indicating that syntactic deficits may derive from posterior lesions, and a number of neuroimaging studies show posterior activation related to syntactic processes.

One commonality across the several domains of language processing covered in this review is that frontal activation in neuroimaging studies is more likely to occur for phonological, semantic, and syntactic tasks that require active processing on the part of the subject. It is possible that, as has been hypothesized for semantic processing (Roskies et al. 2001), these frontal activations result from executive processes involved in manipulating or maintaining different types of representations—with different frontal regions specialized for maintaining different types of information (phonological, semantic, syntactic) or for carrying out

different types of functions (retention, selection). More routinized functions may be carried out in temporal/parietal regions. Such claims are clearly speculative at this point but may provide a basis for further investigation.

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LITERATURE CITED

Albert ML, Bear D. 1957. Time to understand: a case study of word deafness with reference to the role of time in auditory comprehension. *Brain* 97:373–84

Allen M, Badecker W. 1999. Stem homograph and stem allomorphy: representing and processing inflected forms in a multilevel lexical system. *J. Mem. Lang.* 41:105–23

Anderson JM, Gilmore R, Roper S, Crosson B, Bauer RM, et al. 1999. Conduction aphasia and the arcuate fasciculus: a reexamination of the Wernicke-Geschwind model. *Brain Lang.* 70:1–12

Auerbach SH, Allard T, Naeser M, Alexander MP, Albert ML. 1982. Pure word deafness: analysis of a case with bilateral lesions and a defect at the prephonemic level. *Brain* 105:271–300

Badecker W, Caramazza A. 1991. Morphological composition in the lexical output system. *Cogn. Neuropsychol.* 8:335–67

Badecker W, Miozzo M, Zanuttini R. 1995. The two-stage model of lexical retrieval: evidence from a case of anomia with selective preservation of grammatical gender. *Cognition* 57:193–216

Berndt RS, ed. 2001. *Handbook of Neuropsychology: Language and Aphasia*. Amsterdam: Elsevier. 2nd ed.

Berndt RS. 2001. Sentence production. See Rapp 2001, pp. 375–96

Berndt RS, Caramazza A. 1980. A redefinition of the syndrome of Broca's aphasia: implications for a neuropsychological model of language. *Appl. Psycholinguist.* 1:225–78

Berndt RS, Haendiges AN, Burton MW, Mitchum CC. 2002. Grammatical class and imageability in aphasic word production: their effects are independent. *J. Neurolinguist.* 15:353–71

Berndt RS, Haendiges AN, Mitchum CC, Sandson J. 1997. Verb retrieval in aphasia. 2. Relationship to sentence processing. *Brain Lang.* 56:107–37

Berndt RS, Mitchum C, Haendiges A. 1996. Comprehension of reversible sentences in "agrammatism": a meta-analysis. *Cognition* 58:289–308

Binder JR, Frost JA, Hammeke TA, Bellgowan PSF, Springer JA, et al. 2000. Human temporal lobe activation by speech and nonspeech sounds. *Cerebr. Cortex* 10:512–28

Binder JR, Frost JA, Hammeke TA, Cox RW, Rao SM, Prieto T. 1997. Human brain language areas identified by functional magnetic resonance imaging. *J. Neurosci.* 17:353–62

Binder JR, Frost JA, Hammeke TA, Rao SM, Cox RW. 1996. Function of the left planum temporale in auditory and linguistic processing. *Brain* 119:1239–47

Binder JR, Rao SM, Hammeke TA, Yetkin YZ, Jesmanowicz A, et al. 1994. Functional magnetic resonance imaging of human auditory cortex. *Ann. Neurol.* 35: 662–72

Bird H, Franklin S. 1995/1996. Cinderella revisited: a comparison of fluent and non-fluent

aphasic speech. *J. Neurolinguist.* 9:187–206

Bird H, Franklin S, Howard D. 2002. 'Little words'—not really: function and content words in normal and aphasic speech. *J. Neurolinguist.* 15:209–37

Blanken G. 1990. Formal paraphasias: a single case study. *Brain Lang.* 38:534–54

Blumstein SE. 1998. Phonological aspects of aphasia. See Sarno 1998, pp. 157–85

Blumstein SE. 2001. Deficits of speech production and speech perception in aphasia. See Boller et al. 2001, pp. 95–113

Blumstein SE, Baker E, Goodglass H. 1977. Phonological factors in auditory comprehension in aphasia. *Neuropsychologia* 15:19–30

Bock K. 1989. Closed-class immanence in sentence production. *Cognition* 31:163–86

Bock K, Levelt W. 1994. Language production: grammatical encoding. In *Handbook of Psycholinguistics*, ed. MA Gernsbacher, pp. 945–84. San Diego, CA: Academic

Boland J. 1997. The relationship between syntactic and semantic processes in sentence comprehension. *Lang. Cogn. Process.* 12: 423–84

Bradley DC, Garrett MF, Zurif EB. 1980. Syntactic deficits in Broca's aphasia. In *Biological Studies of Mental Proceses*, ed. D Caplan, pp. 269–86. Cambridge, MA: MIT Press

Breedin S, Martin RC. 1996. Patterns of verb deficits in aphasia. An analysis of four cases. *Cogn. Neuropsychol.* 13:51–91

Breedin S, Saffran E. 1999. Sentence processing in the face of semantic loss: a case study. *J. Exp. Psychol.: Gen.* 128:547–62

Bub D, Black S, Howell J, Kertesz A. 1987. Speech output processes and reading. In *The Cognitive Neuropsychology of Language*, ed. M Coltheart, G. Sartori, R Job, pp. 79–110. Hillsdale, NJ: Erlbaum

Buchsbaum BR, Hickok G, Humphries C. 2001. Role of left posterior superior temporal gyrus in phonological processing for speech perception and production. *Cogn. Sci.* 25:663–78

Burton M, Small S, Blumstein S. 2000. The role of segmentation in phonological processing: an fMRI investigation. *J. Cogn. Neurosci.* 12:679–90

Butterworth B, ed. 1980. *Language Production*, Vol. 1. London: Academic

Butterworth B, Campbell R, Howard D. 1986. The uses of short-term memory: a case study. *Q. J. Exp. Psychol.* 38A:705–37

Butterworth B, Howard D. 1987. Paragrammatisms. *Cognition* 26:1–37

Bybee JL. 1988. Morphology as lexical organization. In *Theoretical Morphology: Approaches in Modern Linguistics*, ed. M Hammond, M Noonan, pp. 119–41. San Diego, CA: Academic

Byng S. 1988. Sentence processing deficits: theory and therapy. *Cogn. Neuropsychol.* 5:629–76

Cabeza R, Nyberg L. 2000. Imaging cognition. II. An empirical review of 275 PET and fMRI studies. *J. Cogn. Neurosci.* 12:1–47

Caplan D. 1987. *Neurolinguistics and Linguistic Aphasiology: An Introduction.* New York: Cambridge Univ. Press

Caplan D. 2001. Functional neuroimaging studies of syntactic processing. *J. Psycholinguist. Res.* 30:297–320

Caplan D, Alpert N, Waters G. 1998. Effects of syntactic structure and propositional number on patterns of regional cerebral blood flow. *J. Cogn. Neurosci.* 10:541–52

Caplan D, Hildebrandt N. 1988. *Disorders of Syntactic Comprehension.* Cambridge, MA: MIT Press

Caplan D, Hildebrandt N, Makris N. 1996. Location of lesions in stroke patients with deficits in syntactic processing in sentence comprehension. *Brain* 119:933–49

Caplan D, Vanier M, Baker C. 1986. A case study of reproduction conduction aphasia. 1. Word production. *Cogn. Neuropsychol.* 3:99–128

Caplan D, Vijayan S, Kuperberg G, West C, Waters G, et al. 2001. Vascular responses to syntactic processing: event-related fMRI study of relative clauses. *Hum. Brain Mapp.* 15:26–38

Caplan D, Waters G. 1999. Verbal working

memory and sentence comprehension. *Behav. Brain Sci.* 22:77–126
Caramazza A, Berndt RS. 1978. Semantic and syntactic processes in aphasia: a review of the literature. *Psychol. Bull.* 85:898–918
Caramazza A, Hillis AE. 1989. The disruption of sentence production: some dissociations. *Brain Lang.* 36:625–50
Caramazza A, Hillis AE. 1990. Where do semantic errors come from? *Cortex* 26:95–122
Caramazza A, McCloskey M. 1988. The case for single patient studies. *Cogn. Neuropsychol.* 5:517–28
Caramazza A, Miceli G. 1991. Selective impairment of thematic role assignment in sentence processing. *Brain Lang.* 41:402–36
Caramazza A, Miceli G, Villa G. 1986. The role of the (output) phonological buffer in reading, writing, and repetition. *Cogn. Neuropsychol.* 3:37–76
Caramazza A, Miozzo M. 1997. The relation between syntactic and phonological knowledge in lexical access: evidence from the "tip-of-the-tongue" phenomenon. *Cognition* 64:309–43
Caramazza A, Papagno C, Ruml W. 2000. The selective impairment of phonological processing in speech production. *Brain Lang.* 75:428–50
Caramazza A, Zurif EB. 1976. Dissociation of algorithmic and heuristic processes in language comprehension: evidence from aphasia. *Brain Lang.* 3:572–82
Cuetos F, Aguado G, Caramazza A. 2000. Dissociation of semantic and phonological errors in naming. *Brain Lang.* 75:451–60
Damasio H. 1998. Neuroanatomical correlates of the aphasias. See Sarno 1998, pp. 43–70
Damasio H, Damasio AR. 1989. *Lesion Analysis in Neuropsychology.* New York: Oxford Univ. Press
Damasio H, Grabowski T, Tranel D, Hichwa R, Damasio A. 1996. A neural basis for lexical retrieval. *Nature* 380:499–505
Dapretto M, Bookheimer SY. 1999. Form and content: dissociating syntax and semantics in sentence comprehension. *Neuron* 24:427–32
Dell G, O'Seaghdha P. 1992. Stages of lexical access in language production. *Cognition* 42:287–314
Dell G, Schwartz M, Martin N, Saffran E, Gagnon D. 1997. Lexical access in aphasic and nonaphasic speakers. *Psychol. Rev.* 104:801-38
Démonet J-F, Chollet F, Ramsay S, Cardebat D, Nespoulous J-L, et al. 1992. The anatomy of phonological and semantic processing in normal subjects. *Brain* 115:1753–68
Démonet J-F, Price C, Wise R, Frackowiak RSJ. 1994. Differential activation of right and left posterior sylvian regions by semantic and phonological tasks: a positron emission tomography study in normal human subjects. *Neurosci. Lett.* 182:25–28
de Renzi E, Nichelli P. 1975. Verbal and nonverbal short-term memory impairment following hemispheric damage. *Cortex* 11:341–54
Devlin J, Russell R, Davis M, Price C, Moss H, et al. 2002. Is there an anatomical basis for category-specificity? Semantic memory studies in PET and fMRI. *Neuropsychologia* 40:54–75
Dick F, Bates E, Wulfeck B, Utman JA, Dronkers N, Gernsbacher MA. 2001. Language deficits, localization, and grammar: evidence for a distributive model of language breakdown in aphasic patients and neurologically intact individuals. *Psychol. Rev.* 108:759–88
Dronkers NF. 1996. A new brain region for coordinating speech articulation. *Nature* 384: 159–61
Dronkers NF, Larsen J. 2001. Neuroanatomy of the classical syndromes of aphasia. See Boller et al. 2001, pp. 19–30
Dronkers NF, Redfern BB, Knight RT. 2000. The neural architecture of language disorders. See Gazzaniga 2000, pp. 949–61
Dronkers NF, Redfern BB, Ludy CA. 1995. Lesion localization in chronic Wernicke's aphasia. *Brain Lang.* 51:62–65
Dronkers NF, Wilkins DP, Van Valin RD, Redfern BB, Jaeger JJ. 1994. A reconsideration of the brain areas involved in the disruption of morphosyntactic comprehension. *Brain Lang.* 47:461–63

Druks J. 2002. Verbs and nouns: a review of the literature. *J. Neurolinguist.* 15:289–315

Fiez JA, Raichle ME, Miezin FM, Petersen SE, Tallal P, Katz WF. 1995. PET studies of auditory and phonological processing: effects of stimulus characteristics and task demands. *J. Cogn. Neurosci.* 7:357–75

Foundas AL, Daniels SK, Vasterling JJ. 1998. Anomia: case studies with lesion localization. *Neurocase* 4:35–43

Foygel D, Dell GS. 2000. Models of impaired lexical access in speech production. *J. Mem. Lang.* 43:182–216

Franklin S, Howard D, Patterson K. 1994. Abstract word meaning deafness. *Cogn. Neuropsychol.* 11:1–34

Franklin S, Turner J, Ralph MAL, Morris J, Bailey PJ. 1996. A distinctive case of word meaning deafness? *Neuropsychology* 13:1139–62

Freedman ML, Martin RC. 2001. Dissociable components of short-term memory and their relation to long-term learning. *Cogn. Neuropsychol.* 18:193–226

Friederici AD, Meyer M, von Cramon DY. 2000. Auditory language comprehension: an event-related fMRI study on the processing of syntactic and lexical information. *Brain Lang.* 74:289–300

Funnell E. 1987. Morphological errors in acquired dyslexia: a case of mistaken identity. *Q. J. Exp. Psychol.* 39:497–539

Garrett MF. 1980. Levels of processing in sentence production. See Butterworth 1980, pp. 177–220

Garrett MF. 1993. Errors and their relevance for models of language production. In *Linguistic Disorders and Pathologies*, ed. G Blanken, J Dittman, H Grim, J Marshall, C Wallesch, pp. 69–96. Berlin: de Gruyter

Gaskell MG, Marslen-Wilson WD. 1997. Integrating form and meaning: a distributed model of speech perception. *Lang. Cogn. Process.* 12:613–56

Gazzaniga MS, ed. 2000. *The New Cognitive Neurosciences*. Cambridge, MA: MIT Press. 2nd ed.

Geschwind N. 1965. Disconnexion syndromes in animals and man. *Brain* 88:237–94, 585–644

Geschwind N. 1970. The organization of brain and language. *Science* 170:940–44

Gibson E. 1998. Linguistic complexity: locality of syntactic dependencies. *Cognition* 68:1–76

Goodglass H. 1993. *Understanding Aphasia.* San Diego, CA: Academic

Goodglass H, Christiansen JA, Gallagher RE. 1994. Syntactic constructions used by agrammatic speakers: comparison with conduction aphasics and normals. *Neuropsychology* 8:598–613

Griffiths TD, Rees A, Green GGR. 1999. Disorders of human complex sound processing. *Neurocase* 5:365–78

Haarmann HJ, Kolk HH. 1992. The production of grammatical morphology in Broca's and Wernicke's aphasics: speed and accuracy factors. *Cortex* 28:97–112

Hall DA, Riddoch MJ. 1997. Word meaning deafness: spelling words that are not understood. *Cogn. Neuropsychol.* 14:1131–64

Hanten G, Martin RC. 2000. Contributions of phonological and semantic short-term memory to sentence processing: Evidence from two cases of closed head injury in children. *J. Mem. Lang.* 43:335–61

Hanten G, Martin RC. 2001. A developmental phonological short-term memory deficit: a case study. *Brain Cogn.* 45:164–88

Hillis AE, Boatman D, Hart J, Gordon B. 1999. Making sense out of jargon: a neurolinguistic and computational account of jargon aphasia. *Neurology* 53:1813–24

Hillis AE, Rapp B, Romani C, Caramazza A. 1990. Selective impairment in semantics in lexical processing. *Cogn. Neuropsychol.* 7:191–243

Hillis AE, Kane A, Tuffiash E, Ulatowski JA, Barker PB, et al. 2001. Reperfusion of specific brain regions by raising blood pressure restores selective language functions in subacute stroke. *Brain Lang.* 79:495–510

Hodges JR, Patterson K, Tyler L. 1994. Loss of semantic memory: implications for the

modularity of mind. *Cogn. Neuropsychol.* 11:505–42

Hodges JR, Patterson K, Oxbury S, Funnell E. 1992. Semantic dementia: progressive fluent aphasia with temporal lobe atrophy. *Brain* 115:1783–86

Hofstede B, Kolk H. 1994. The effects of task variation on the production of grammatical morphology in Broca's aphasia: a multiple case study. *Brain Lang.* 46:278–328

Howard D. 1995. Lexical anomia: or the case of the missing lexical entries. *Q. J. Exp. Psychol.* 48A:999–1023

Howard D, Franklin S. 1988. *Missing the Meaning: A Cognitive Neuropsychological Study of Processing of Words by an Aphasic Patient*. Cambridge, MA: MIT Press

Howard D, Franklin S. 1990. Memory without rehearsal. In *Neuropsychological Impairments of Short-Term Memory*, ed. G Vallar, T Shallice, pp. 287–318. Cambridge, UK: Cambridge Univ. Press

Howard D, Orchard-Lisle V. 1984. On the origin of semantic errors in naming: evidence from the case of a global aphasic. *Cogn. Neuropsychol.* 1:163–90

Howard D, Patterson K, Wise R, Brown WD, Friston K, et al. 1992. The cortical localization of the lexicons. *Brain* 115:1769–82

Indefrey P, Brown CM, Hellwig F, Amunts K, Herzog H, et al. 2001. A neural correlate of syntactic encoding during speech production. *Proc. Natl. Acad. Sci. USA* 98:5933–36

Indefrey P, Levelt W. 2000. The neural correlates of language production. See Gazzaniga 2000, pp. 845–65

Joanisse MF, Seidenberg MS. 1999. Impairments in verb morphology after brain injury: a connectionist model. *Proc. Natl. Acad. Sci. USA* 96:7592–97

Just M, Carpenter P. 1992. A capacity theory of comprehension: individual differences in working memory. *Psychol. Rev.* 99:122–49

Just MA, Carpenter PA, Keller TA, Eddy WF, Thulborn KR. 1996. Brain activation modulated by sentence comprehension. *Science* 274:114–16

Kircher TTJ, Brammer MJ, Williams SCR, McGuire PK. 2000. Lexical retrieval during fluent speech production: an fMRI study. *NeuroReport* 11:4093–96

Kohn SE, Friedman RB. 1986. Word-meaning deafness: a phonological-semantic dissociation. *Cogn. Neuropsychol.* 3:291–308

Kolk H. 1995. A time-based approach to agrammatic production. *Brain Lang.* 50:282–303

Kolk H, Heeschen C. 1992. Agrammatism, paragrammatism, and the management of language. *Lang. Cogn. Process.* 7:89–129

Kolk HHJ, Van Grunsven MJF, Keyser A. 1985. On parallelism between production and comprehension in agrammatism. In *Agrammatism*, ed. ML Kean, pp. 165–206. Orlando, FL: Academic

Kuperberg GR, McGuire PK, Bullmore ET, Brammer MJ, Rabe-Hesketh S, et al. 2000. Common and distinct neural substrates for pragmatic, semantic, and syntactic processing of spoken sentences: an fMRI study. *J. Cogn. Neurosci.* 12:321–41

Lenneberg EH. 1973. The neurology of language. *Daedalus* 102:115–33

Levelt WJM, Roelofs A, Meyer AS. 1999. A theory of lexical access in speech production. *Behav. Brain Sci.* 22:1–75

Lichtheim L. 1885. On aphasia. *Brain* 7:433–84

Linebarger MC. 1990. Neuropsychology of sentence parsing. In *Cognitive Neuropsychology and Neurolinguistics: Advances in Models of Cognitive Function and Impairment*, ed. A Caramazza, pp. 55–122. Hillsdale, NJ: Erlbaum

Linebarger MC, Schwartz M, Saffran E. 1983. Sensitivity to grammatical structure in so-called agrammatic aphasics. *Cognition* 13:361–92

Linebarger MC, Schwartz MF, Romania JR, Kohn SE, Stephens DL. 2000. Grammatical encoding in aphasia: evidence from a "processing prosthesis." *Brain Lang.* 75:416–27

Marslen-Wilson W, Warren P. 1994. Levels of perceptual representation and process in lexical access: words, phonemes, and features. *Psychol. Rev.* 4:653–75

Martin N, Saffran EM. 1992. A computational

account of deep dysphasia: evidence from a single case study. *Brain Lang.* 43:240–74

Martin N, Saffran EM. 1997. Language and auditory-verbal short-term memory impairments: evidence for common underlying processes. *Cogn. Neuropsychol.* 14:641–82

Martin N, Saffran EM. 2002. The relationship of input and output phonological processing: an evaluation of models and evidence to support them. *Aphasiology* 16:107–50

Martin RC. 1995. Working memory doesn't work: a critique of Miyake et al.'s capacity theory of aphasic comprehension deficits. *Cogn. Neuropsychol.* 12:623–36

Martin RC, Blossom-Stach C. 1986. Evidence of syntactic deficits in a fluent aphasic. *Brain Lang.* 28:196–234

Martin RC, Breedin SD, Damian MF. 1999a. The relation of phoneme discrimination, lexical access, and short-term memory: a case study and interactive activation account. *Brain Lang.* 70:437–82

Martin RC, Freedman ML. 2001. Short-term retention of lexical-semantic representations: implications for speech production. *Memory* 9:261–80

Martin RC, Lesch MF, Bartha MC. 1999b. Independence of input and output phonology in word processing and short-term memory. *J. Mem. Lang.* 41:3–29

Martin RC, Romani C. 1994. Verbal working memory and sentence comprehension: a multiple-components view. *Neuropsychology* 8:506–23

Martin RC, Shelton JR, Yaffee LS. 1994. Language processing and working memory: neuropsychological evidence for separate phonological and semantic capacities. *J. Mem. Lang.* 33:83–111

Mazoyer BM, Tzourio N, Frak V, Syrota A, Murayama N, et al. 1993. The cortical representation of speech. *J. Cogn. Neurosci.* 5:467–79

McClelland JL, Elman JL. 1986. The TRACE model of speech perception. *Cogn. Psychol.* 18:1–86

McCloskey M. 1993. Theory and evidence in cognitive neuropsychology: a "radical" response to Robertson, Knight, Rafal, and Shimamura. *J. Exp. Psychol.: Learn. Mem. Cogn.* 19:718–34

McDermott K, Petersen S, Watson J, Ojemann J. 2002. A method for identifying regions selectively activated by semantic and phonological processing using functional magnetic resonance imaging. *Neuropsychologia.* In press

Menn L. 2001. Comparative aphasiology: cross-language studies of aphasia. See Boller et al. 2001, pp. 51–68

Miceli G, Mazzucchi A, Menn L, Goodglass H. 1983. Contrasting cases of Italian agrammatic aphasia without comprehension disorder. *Brain Lang.* 19:65–97

Miceli G, Silveri MC, Romani C, Caramazza A. 1989. Variation in the pattern of omissions and substitutions of grammatical morphemes in the spontaneous speech of so-called agrammatic patients. *Brain Lang.* 36: 447–92

Miceli G, Silveri MC, Villa G, Caramazza A. 1984. On the basis for the agrammatics' difficulty in producing main verbs. *Cortex* 20:207–20

Mohr JP, Pessin MS, Finkelstein S, Funkenstein HH, Duncan GW, Davis KR. 1978. Broca aphasia: pathologic and clinical. *Neurology* 28:311–24

Moro A, Tettamanti M, Perani D, Donati C, Cappa SF, Fazio F. 2001. Syntax and the brain: disentangling grammar by selective anomalies. *NeuroImage* 13:110–18

Mummery CJ, Patterson K, Price CJ, Ashburner J, Frackowiak RSJ, Hodges JR. 2000. A voxel-based morphometry study of semantic dementia: relationship between temporal lobe atrophy and semantic memory. *Ann. Neurol.* 47:36–45

Murdoch BE, Afford RJ, Ling AR, Ganguley, B. 1986. Acute computerized tomographic scans: their value in the localization of lesions and as prognostic indicators in aphasia. *J. Commun. Disord.* 19:311–45

Nadeau SE, Rothi LJG. 1992. Morphologic agrammatism following a right hemisphere stroke in a dextral patient. *Brain Lang.* 43: 642–67

Naeser MA, Mazurski P, Goodglass H, Peraino M, Laughlin S, Leaper WC. 1987. Auditory syntactic comprehension in nine aphasia groups (with CT scans) and children: differences in degree but not order of difficulty observed. *Cortex* 23:359–80

Nespoulous JL, Dordain M, Perron C, Ska B, Bub D, et al. 1988. Agrammatism in sentence production without comprehension deficits: reduced availability of syntactic structures and/or of grammatical morphemes? A case study. *Brain Lang.* 33:273–95

Ni W, Constable RT, Mencl WE, Pugh KR, Fulbright RK, et al. 2000. An event-related neuroimaging study distinguishing form and content in sentence processing. *J. Cogn. Neurosci.* 12:120–33

Nickels L. 2001. Spoken word production. See Rapp 2001, pp. 291–320

Nickels L, Howard D, Best W. 1997. Fractionating the articulatory loop: dissociations and associations in phonological recoding in aphasia. *Brain Lang.* 56:161–82

Norris D, Wise R. 2000. The study of prelexical and lexical processes in comprehension: psycholinguistics and functional neuroimaging. See Gazzaniga 2000, pp. 867–80

Ostrin R, Tyler L. 1995. Dissociations of lexical function: semantics, syntax, and morphology. *Cogn. Neuropsychol.* 12:345–89

Patterson K. 2002. *A double dissociation but a single mechanism? Further neuropsychological evidence on the past-tense debate*. Presented at Cogn. Neurosci. Soc. Meet., 9th, San Francisco

Patterson K, Lambon Ralph MA, Hodges JR, McClelland JL. 2001. Deficits in irregular past-tense verb morphology associated with degraded semantic knowledge. *Neuropsychologia* 39:709–24

Petersen SE, Fox PT, Posner MI, Mintun M, Raichle ME. 1988. Positron emission tomographic studies of the cortical anatomy of single-word processing. *Nature* 331:585–89

Petersen SE, Fox PT, Posner MI, Mintun M, Raichle ME. 1989. Positron emission tomographic studies of the processing of single-words. *J. Cogn. Neurosci.* 1:153–70

Poeppel D. 2001. Pure word deafness and the bilateral processing of the speech code. *Cogn. Sci.* 25:679–93

Poeppel D, Yellin E, Phillips C, Roberts TPL, Rowley HA, et al. 1996. Task-induced asymmetry of the auditory evoked M100 neuromagnetic field elicited by speech sounds. *Cogn. Brain Res.* 4:231–42

Poldrack RA, Wagner AD, Prull MW, Desmond JE, Glover GH, Gabrieli JD. 1999. Functional specialization for semantic and phonological processing in the left inferior prefrontal cortex. *NeuroImage* 10:15–35

Praamstra P, Hagoort P, Maassen B, Crul T. 1991. Word deafness and auditory cortical function: a case history and hypothesis. *Brain* 114:1197–225

Price CJ, Wise RJS, Warburton EA, Moore CJ, Howard D, et al. 1996. Hearing and saying: the functional neuro-anatomy of auditory word processing. *Brain* 119:919–31

Rapp B, ed. 2001. *The Handbook of Cognitive Neuropsychology: What Deficits Reveal About the Human Mind*. Philadelphia: Psychology Press/Taylor & Francis

Rapp B, Caramazza A. 2002. Selective difficulties with spoken nouns and written verbs: a single case study. *J. Neurolinguist.* 15:373–402

Rapp B, Goldrick M. 2000. Discreteness and interactivity in spoken word production. *Psychol. Rev.* 107:460–99

Raymer AM, Foundas A, Maher LM, Greenwald ML, Morris M, et al. 1997. Cognitive neuropsychological analysis and neuroanatomic correlates in a case of acute anomia. *Brain Lang.* 34:137–56

Romani C. 1992. Are there distinct input and output buffers? *Lang. Cogn. Process.* 7:131–62

Romani C, Martin RC. 1999. A deficit in the short-term retention of lexical-semantic information: forgetting words but remembering a story. *J. Exp. Psychol.: Gen.* 128:56–77

Roskies AL, Fiez JA, Balota DA, Raichle ME, Petersen SE. 2001. Task-dependent modulation of regions in the left inferior frontal

cortex during semantic processing. *J. Cogn. Neurosci.* 13:829–43

Rumelhart DE, McClelland JL. 1986. On learning the past tenses of English verbs. In *Parallel Distributed Processing: Explorations in the Microstructure of Cognition*, ed. JL McClelland, DE Rumelhart, 2:216–71. Cambridge, MA: MIT Press

Ruml W, Caramazza A, Shelton JR, Chialant D. 2000. Testing assumptions in computational theories of aphasia. *J. Mem. Lang.* 43:217–48

Saffran EM, Schwartz MF. 1988. "Agrammatic" comprehension it's not: alternatives and implications. *Aphasiology* 2:389–94

Saffran EM, Schwartz MF, Linebarger MC. 1998. Semantic influences on thematic role assignments: evidence from normals and aphasics. *Brain Lang.* 62:255–97

Saffran EM, Berndt RS, Schwartz MF. 1989. The quantitative analysis of agrammatic production: procedure and data. *Brain Lang.* 37:440–79

Saffran EM, Schwartz MF, Marin OS. 1980. Evidence from aphasia: isolating the components of a production model. See Butterworth 1980, pp. 221–41

Sarno MT, ed. 1998. *Acquired Aphasia*. San Diego, CA: Academic. 3rd ed.

Schwartz MF, Chawluk JB. 1990. Deterioration of language in progressive aphasia: a case study. In *Modular Deficits in Alzheimer-Type Dementia*, ed. M Schwartz, pp. 245–96. Cambridge, MA: MIT Press

Schwartz MF, Saffran EM, Marin OS. 1980. The word order problem in agrammatism. I. Comprehension. *Brain Lang.* 10:249–62

Scott SK, Blank CC, Rosen S, Wise RJS. 2000. Identification of a pathway for intelligible speech in the left temporal lobe. *Brain* 123:2400–6

Selnes OA, Knopman DS, Niccum N, Rubens AB, Larson D. 1983. Computed tomographic scan correlates of auditory comprehension deficits in aphasia: a prospective recovery study. *Ann. Neurol.* 13:558–66

Shallice T. 1988. *From Neuropsychology to Mental Structure.* New York: Cambridge Univ. Press

Shallice T, Rumiati RI, Zadini A. 2000. The selective impairment of the phonological output buffer. *Cogn. Neuropsychol.* 17:517–46

Shankweiler D, Crain S, Gorrell P, Tuller B. 1989. Reception of language in Broca's aphasia. *Lang. Cogn. Process.* 4:1–33

Shapiro K, Shelton J, Caramazza A. 2000. Grammatical class in lexical production and morphological processing: evidence from a case of fluent aphasia. *Cogn. Neuropsychol.* 17:665–82

Shelton JR, Caramazza A. 1999. Deficits in lexical and semantic processing: implications for models of normal language. *Psychonomic Bull. Rev.* 6:5–27

Shelton JR, Caramazza A. 2001. The organization of semantic memory. See Rapp 2001, pp. 423–43

Stemberger JP. 1984. Structural errors in normal and agrammatic speech. *Cogn. Neuropsychol.* 1:281–313

Stowe LA, Broere CAJ, Paans AM, Wijers AA, Mulder G, et al. 1998. Localizing components of a complex task: sentence processing and working memory. *NeuroReport* 9:2995–99

Stromswold K, Caplan D, Alpert N, Rauch S. 1996. Localization of syntactic comprehension by positron emission tomography. *Brain Lang.* 52:452–73

Takahashi N, Kawamura M, Shinotou H, Hirayama K, Kaga K, et al. 1992. Pure word deafness due to left hemisphere damage. *Cortex* 28:295–303

Tanaka Y, Yamadori A, Mori E. 1987. Pure word deafness following bilateral lesions: a psychophysical analysis. *Brain* 110:381–403

Thompson CK, Fix S, Gitelman D. 2002. Selective impairment of morphosyntactic production in a neurological patient. *J. Neurolinguist.* 15:189–207

Thompson-Schill SL, D'Esposito M, Aguierre GK, Farah MJ. 1997. Role of left inferior prefrontal cortex in retrieval of semantic knowledge: a reevaluation. *Proc. Natl. Acad. Sci. USA* 34:14792–97

Thompson-Schill SL, Swick D, Farah MJ, D'Esposito M, Kan IP, Knight RT. 1998.

Verb generation in patients with focal frontal lesions: a neuropsychological test of neuroimaging findings. *Proc. Natl. Acad. Sci. USA* 34:15855–60

Trueswell J, Tanenhaus M, Garnsey S. 1994. Semantic influences on parsing: use of thematic role information in syntactic ambiguity resolution. *J. Mem. Lang.* 33:285–318

Tsapkini K, Jarema G, Kehayia E. 2002. A morphological processing deficit in verbs but not in nouns: a case study in a highly inflected language. *J. Neurolinguist.* 15:265–88

Tyler LK. 1989. Syntactic deficits and the construction of local phrases in spoken language comprehension. *Cogn. Neuropsychol.* 6:333–55

Tyler LK, Randall B, Marslen-Wilson WD. 2002. Phonology and neuropsychology of the English past tense. *Neuropsychologia* 40: 1154–66

Ullman MT, Corkin S, Coppola M, Hickok G, Growdon JH, et al. 1997. A neural dissociation within language: evidence that the mental dictionary is part of declarative memory, and that grammatical rules are processed by the procedural system. *J. Cogn. Neurosci.* 9:266–76

Vandenberghe R, Price C, Wise R, Josephs O, Frackowiak RSJ. 1996. Functional anatomy of a common semantic system for words and pictures. *Nature* 383:254–56

Vigliocco G, Vinson DP, Martin RC, Garrett MF. 1999. Is "count" and "mass" information available when the noun is not? An investigation of tip of the tongue states and anomia. *J. Mem. Lang.* 40:534–58

Wang E, Peach RK, Xu Y, Schneck M, Manry C. 2000. Perception of dynamic acoustic patterns by an individual with unilateral verbal auditory agnosia. *Brain Lang.* 73:442–55

Waters G, Caplan D, Hildebrandt N. 1991. On the structure of verbal short-term memory and its functional role in sentence comprehension: evidence from neuropsychology. *Cogn. Neuropsychol.* 8:81–126

Wernicke C. 1874. *Der aphasische symptomenkomplex*. Breslau: Cohn Weigert. Transl. G Eggert. 1977. *Wernicke's Works on Aphasia*. The Hague: Mouton

Wise RJS, Scott SK, Blank SC, Mummery CJ, Murphy K, Warburton EA. 2001. Separate neural subsystems within 'Wernicke's area'. *Brain* 124:83–95

Wulfeck B. 1988. Grammaticality judgments and sentence comprehension in agrammatic aphasia. *J. Speech Hear. Res.* 31:72–81

Zatorre RJ, Evans AC, Meyer E, Gjedde A. 1992. Lateralization of phonetic and pitch discrimination in speech processing. *Science* 256:846–49

Zingeser LB, Berndt RS. 1988. Grammatical class and context effects in a case of pure anomia: implications for models of language production. *Cogn. Neuropsychol.* 5:473–516

Zingeser LB, Berndt RS. 1990. Retrieval of nouns and verbs in agrammatism and anomia. *Brain Lang.* 39:14–32