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Language Deficits, Localization, and Grammar: Evidence for a Distributive Model of Language Breakdown in Aphasic Patients and Neurologically Intact Individuals

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Selective deficits in aphasic patients' grammatical production and comprehension are often cited as evidence that syntactic processing is modular and localizable in discrete areas of the brain (e.g., Y. Grodzinsky, 2000). The authors review a large body of experimental evidence suggesting that morpho-syntactic deficits can be observed in a number of aphasic and neurologically intact populations. They present new data showing that receptive agrammatism is found not only over a range of aphasic groups, but is also observed in neurologically intact individuals processing under stressful conditions. The authors suggest that these data are most compatible with a domain-general account of language, one that emphasizes the interaction of linguistic distributions with the properties of an associative processor working under normal or suboptimal conditions.

The primary purpose of this article is to provide empirical arguments in support of a new view of language deficits and their neural correlates, particularly in the realm of syntax. *Selective* syntactic deficits are often cited as evidence that the human brain contains a bounded and well-defined faculty or module dedicated exclusively to the representation and/or processing of syntax (Caplan & Waters, 1999; Grodzinsky, 1995a, 1995b, 2000; Pinker, 1994). Here, we present a wide range of experimental evidence (both new and old) showing that such deficits are more accurately, and parsimoniously, characterized as interactions between specific linguistic environments and a domain-general (as opposed to language-specific) processor.

The link between brain injury and aphasia has been known for at least 3,000 years (O'Neill, 1980). For more than 100 years, researchers have also known that aphasia is more likely following injuries to the left side of the brain, and that different kinds of aphasia can result depending on the nature of the injury and its locus within the left hemisphere (Goodglass, 1993). Paul Broca was the first to associate deficits in language production with damage to an anterior region of the left hemisphere, now known as "Broca's area." Carl Wernicke is credited with the subsequent discovery of a contrasting form of aphasia, characterized by a comprehension deficit in the presence of fluent speech, usually associated with damage to a posterior region of the left hemisphere that is now called "Wernicke's area." These discoveries launched a century of debate, which is still unresolved, revolving around the nature of these and other contrasting forms of aphasia, and their neural correlates. Although any dichotomy is an oversimplification when it is applied to questions of this magnitude, the poles of this debate have been defined (and can still be defined) in terms of the theorist's stand on three related issues: localization, transparency of mapping and domain specificity.

Localizationists argue in favor of the idea that language (or specific subcomponents of language) is represented and processed in one or more bounded regions of the brain. This belief, which has continuous and explicit roots in Franz Gall's doctrine of phrenol-

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ogy (Fodor, 1983; Gall, 1810), is usually accompanied by two corollaries: (a) there is a transparent mapping between specific functions (i.e., specific behaviors, experiences, and domains of knowledge, or all of these) and the neural regions that mediate those functions, and (b) these neural regions are dedicated exclusively to the functional domains that they serve (e.g., an area that mediates grammar is not involved in other forms of sequenced perceptual or motor behavior). If one accepts these assumptions, then it is meaningful to describe a given stretch of tissue as a "language zone" or even a "grammar zone."

The alternative to localization is sometimes cast in negative terms (i.e., as nonlocalization). Early nonlocalizationists used terms such as *holism* (Goldstein, 1948), *equipotentiality,* and *mass action* (Lashley, 1950) to describe their alternative view, emphasizing a lack of specificity in cortical organization for language. These amorphous terms have not outlived their authors, and with good reason: It is now indisputable that the brain is a highly differentiated organ at birth, with substantial division of labor from one region to another (Clancy, Darlington, & Finlay, 2000; Elman et al., 1996).

The modern alternative to classic localization embraces this central tenet, but casts it in a different form: Complex functions like language emerge from the conjoint activity of many brain regions which may be spatially discontinuous and widely distributed. For present purposes, we use the term *distributivity* to describe this alternative to the localizationist view. Distributivity almost always coexists with the rejection of transparent mapping and domain specificity: A given region may be *relevant* for language, *participate* in language, and even be *essential* for language, but its relationship to language is not transparent, nor is it dedicated exclusively to the processing of language or any of its subcomponents. Instead, the regions involved in language processing are also involved in the mediation of processes that language shares with other domains, including specific forms of memory, attention, perception, and motor planning. From this point of view, it is no more appropriate to refer to a participating region as a language zone or a grammar zone than it would be to refer to the elbow as a "tennis organ."

Our goal in this article is to provide evidence in favor of a distributive approach to grammar and against the claim that deficits in the processing of grammar necessarily derive from damage to a localized, bounded, and self-contained module or organ dedicated exclusively to this aspect of language. We show that (a) deficits in grammatical processing (both receptive and expressive) are not restricted to any single type of aphasia, and hence are not associated with damage to specific regions, and that (b) the specific profile of deficits referred to as "receptive agrammatism" can be reproduced by testing college students under adverse processing conditions (e.g., temporal or spectral degradation of the acoustic signal, or both), suggesting that these grammatical deficits may have causes that lie outside of the linguistic domain. We review recent evidence on both these points, and also provide two new sets of data in their support. The first involves a large group of aphasic patients, who vary in their symptom profiles as well as the nature and location of their lesions. The second involves a large sample of college students who are tested on the same stimuli under one of nine different stress conditions. In both these data sets, we target what has been called the "core data" of agrammatism (Hickok & Avrutin, 1995). These are specific deficits in the processing of complex syntactic structures with noncanonical word order such as *passives* ("The boy was hit by the girl") and *object clefts* ("It was the boy who the girl hit"), relative to *actives* ("The girl is hitting the boy") and *subject clefts* ("It's the girl that is hitting the boy"). We show that results from both data sets are most parsimoniously interpreted in the framework of a distributive model—one that is nonlocalizationist in that it eschews the notions of transparent mapping and domain specificity. From this perspective, the linguistic system infiltrates and grows within a brain that is organized along sensorimotor coordinates that were already in place (phylogenetically and ontogenetically) when language first emerged.

What Dissociates in Aphasia: A Brief History

When the basic aphasic syndromes were first outlined by Broca, Wernicke, and their colleagues, differences among forms of linguistic breakdown were explained along sensorimotor lines, rooted in rudimentary principles of neuroanatomy. For example, the symptoms associated with damage to Broca's area were referred to collectively as *motor aphasia*: slow and effortful speech, with a reduction in grammatical complexity, despite the apparent preservation of speech comprehension at a clinical level. This definition made sense in view of the fact that Broca's area lies in frontal cortex, anterior to what we now refer to as the *motor strip.*

Conversely, the symptoms associated with damage to Wernicke's area were defined collectively as *sensory aphasia*: moderate-to-severe problems in speech comprehension in the presence of fluent but empty speech and moderate-to-severe word-finding problems. This characterization also made sense to early neurologists, because Wernicke's area lies in posterior cortex, at the interface between auditory cortex and the various sensory association areas that were presumed to mediate or contain word meaning. Isolated problems with repetition were further ascribed to fibers that link Broca's and Wernicke's area; other syndromes involving the selective sparing or impairment of reading or writing were proposed, with speculations about the fibers that connect visual cortex with the classical language areas (for an influential and highly critical historical review, see Head, 1926).

This classification of aphasia types in terms of sensory and motor "centers" still appears in many medical texts, perhaps because of its appealing neuroanatomical simplicity. However, as Sigmund Freud pointed out in his remarkably prescient book *On Aphasia* (Freud, 1891/1953), there were problems with this particular sensorimotor account of aphasic symptoms from the beginning. To illustrate Freud's concern, consider the following brief speech samples from two contrasting aphasic patients.[1] The first sample comes from a nonfluent Broca's aphasic, who attempts to describe the episode surrounding his stroke (which he suffered in a hot tub):

> Alright. . . . Uh . . . stroke and uh . . . I . . . huh tawanna guy . . . h . . . h . . . hot tub and. . . . And the . . . two days when uh . . . Hos . . . uh . . . huh hospital and uh . . . amet . . . am . . . ambulance.

This patient's struggle to produce meaningful speech is painful to watch and to hear, comprising multiple false starts and a predom-

[1] Many thanks to Dan Kempler for the patient videotapes from which these examples were drawn.

inance of content words with little or no connective tissue (but some function words are produced, something that is, in fact, typical for nonfluent patients who are able to speak at all).

The second sample comes from a fluent but severely impaired Wernicke's aphasic patient who is responding to the same question about the episode in which he suffered his stroke:

> It just suddenly had a feffort and all the feffort had gone with it. It even stepped my horn. They took them from earth you know. They make my favorite nine to severed and now I'm a been habed by the uh stam of fortment of my annulment which is now forever.

This patient produces fluent speech that appears to be largely intact at the levels of grammar and prosody. However, it is almost entirely devoid of content, a symptom known as *jargon aphasia* that includes semantic and phonological paraphasias (word substitutions and sound substitutions that make little sense in context) and neologisms (nonexistent words that probably represent blends of existing words).

Although Freud (1891/1953) was willing to accept that idea that Broca's aphasia has a motor basis, he pointed out that the bizarre speech of Wernicke's aphasic patients simply cannot be explained in terms of sensory loss (as an illustration, compare the output of the jargon-aphasia patient above with the relatively normal speech of an individual who became totally deaf late in life). In place of the static "centers and connections" view proposed by Wernicke and colleagues, Freud proposed a more dynamic and plastic model of brain organization in which concepts and their associated sounds are activated together in time in a bidirectional cascade of events that involve many different parts of the brain. Indeed, Freud's model is surprisingly similar to the distributed representations and activation dynamics proposed in modern neural network accounts. Within Freud's framework, the paraphasia and jargon produced by Wernicke's aphasic patients result not from damage to speech centers per se, but from catastrophic damage to broad temporal and parietal regions that contain the associations that comprise linguistic meaning, as well as the links from meaning to sound. Freud (1891/1953) concluded the following:

> It only remains for us to state the view that the speech area is a continuous cortical region within which the associations and transmissions underlying the speech functions are taking place; they are of a complexity beyond comprehension. (p. 62)

Despite Freud's admonitions (and related critiques by J. Hughlings Jackson, Kurt Goldstein, Henry Head, and many others), an account of aphasia subtypes in terms of discontinuous sensorimotor centers and their connections remained the dominant view until the 1970s, when a radical revision of the sensorimotor account was proposed (summarized in Kean, 1985). Psychologists and linguists were inspired by generative grammar, seeking an account of language breakdown in aphasia within the modular analysis of the human language faculty proposed by Noam Chomsky and his colleagues. By equating specific forms of brain injury with specific linguistic symptoms, these investigators hoped to provide evidence in favor of Chomsky's long-standing proposal that grammar constitutes an innate and autonomous "mental organ" (Chomsky, 1968, 1988).

This effort was fueled by the discovery that Broca's aphasic patients do indeed suffer from comprehension deficits (Caramazza & Zurif, 1976; Heilman & Scholes, 1976). Specifically, it was shown that Broca's aphasic patients display problems in the interpretation of sentences when they are forced to rely entirely on grammatical rather than semantic–pragmatic and grammatical cues. For example, Broca's aphasic patients may successfully interpret a semantically unambiguous sentence such as "The apple was eaten by the girl," but they often fail on semantically reversible sentences such as "The boy was pushed by the girl," where either noun can perform the action. The key point here is that Broca's aphasic patients fail on sentences that must be disambiguated through syntactic analysis—a receptive symptom that seems to involve the same aspects that are impaired in the patients' expressive speech. On the basis of this similarity, it was proposed that Broca's aphasia represents a central and selective impairment of grammar in patients who still have spared comprehension and production of lexical and propositional semantics. Caramazza, Berndt, Basili, and Koller (1981) stated this position succinctly, as follows:

> Although it is possible that Broca patients may suffer from deficits in addition to this syntactic processing deficit, it should be the case that all patients classified as Broca's aphasics will produce evidence of a syntactic impairment in all modalities. (p. 348)

From this point of view, it also seemed possible to reinterpret the problems associated with Wernicke's aphasia as a selective impairment of semantics (resulting in comprehension breakdown and in word-finding deficits in expressive speech), accompanied by a selective sparing of grammar (evidenced by the patients' fluent but empty speech). In some respects, the equation of Wernicke's aphasia with semantic deficits is closer to Freud's (1891/1953) proposal than it is to the sensorimotor account proposed by Wernicke. However, in contrast with Freud's dynamic and distributed approach to the relationship between sound and meaning, the linguistic parsing of the brain that emerged in the 1970s was strongly localizationist in flavor, involving clear claims about the dissociability of grammar and semantics and the transparent mapping of these two domains onto separate and domain-specific neural systems.[2]

It was never entirely obvious how or why the brain ought to be organized in just this way (e.g., why the supposed seat of grammar ought to be located near the motor strip), but for many investigators the absence of a neuroanatomical explanation was less compelling than the apparent isomorphism between aphasic syndromes and the components predicted by some linguistic theories. As noted by Mauner, Fromkin, and Cornell (1993),

> Theoretical interest in aphasia is due, in part, to the fact that focal brain injuries . . . may lead to specific impairments in either the construction of linguistic representations or specific language processing mechanisms. Aphasic deficits following brain damage may thus serve as a testing ground for theoretical models of the normal mental grammar. (p. 340)

[2] Use of double dissociations (in single case and group studies) for purposes of defining neurological functional mappings is increasingly under attack (Elman et al., 1996; Juola & Plunkett, 1998; Plaut, 1995; Van Orden, Pennington, & Stone, 2001). Furthermore, many linguistic claims are based on single dissociations, the validity of which has long been debated in neuropsychological circles (McCarthy & Warrington, 1990).

In the same vein, Hickok and Avrutin (1995) suggested that the selective deficits in sentence comprehension and production observed in Broca's aphasic patients may "be characterized in terms of a representational limitation in one or another module of the normal grammar" (p. 10).

The claim that selective grammatical deficits are correlated with lesions to Broca's area is now well known across the subfields of cognitive science that study language, and is frequently cited in the burgeoning literature on neural imaging of language processing in normal individuals, even by those with a decidedly nonphrenological bent (Kim, Relkin, Lee, & Hirsch, 1997; Rizzolatti & Arbib, 1998). Caplan, Alpert, Waters, and Olivieri (2000, p. 65) noted

> There is very strong evidence from deficit-lesion correlational analyses that the assignment of syntactic form is largely carried out in the dominant perisylvian association cortex [Caplan, et al., 1996]. Some researchers have argued that one aspect of syntactic processing—relating the head noun of a relative clause to its position in the relative clause—is affected only by lesions in Broca's area, and that lesions in this region affect only this syntactic process [Zurif, Swinney, Prather, Solomon, and Bushell, 1993; Swinney and Zurif, 1995; Grodzinsky, 2000], but others disagree that the data from aphasia can be interpreted this way [Caplan, 1995, 1999; Berndt and Caramazza, 1999].

As we show below, the existence of any correlation between discrete neural regions and syntactic processing is highly controversial. It is not at all clear that grammar can be dissociated from lexical semantics in any population (Bates & Goodman, 1997), nor is it at all clear that grammatical deficits are uniquely associated with any specific syndrome or lesion site (Caplan, Hildebrandt, & Makris, 1996). Four kinds of evidence have emerged that cast serious doubt on any first-order linguistic partitioning of the brain:

1. All aphasic patients have deficits in naming, and both agrammatic and paragrammatic aphasic patients show abnormal patterns of semantic priming; hence, there is no such thing as a full double dissociation between deficits in grammatical and lexical processing.
2. Agrammatic patients retain detailed knowledge of their native grammar despite marked deficits in the access and use of that knowledge in real time.
3. Grammatical deficits (both expressive and receptive, morphological and syntactic) have been demonstrated in many different clinical populations, with and without damage to Broca's area.
4. The symptoms of receptive agrammatism can be qualitatively and quantitatively reproduced in normal individuals who are forced to process language under stress.

After a brief review of prior evidence for all four points, we devote our remaining text to new evidence supporting Points 3 and 4.

All Aphasic Patients Have Lexical Impairments

The term *anomia* refers to deficits in the ability to retrieve and produce words. Anomic symptoms occur in many varieties, ranging from the temporary word-finding problems that are sometimes observed in young normal individuals (i.e., the tip-of-the-tongue state; Brown & McNeill, 1966; Levelt, 1989), the mild word-finding problems that accompany normal aging, the chronic naming deficits that are among the first signs of dementia (often accompanied by circumlocutions and "empty speech"), the moderate-to-severe word retrieval problems typically observed in fluent and nonfluent aphasics, to jargon aphasia—the debilitating lexical deficit. In addition to these variations in etiology and severity, variations in content are sometimes observed among anomic patients, including dissociations between proper and common nouns, action versus object names, animate versus inanimate objects, and frequent versus infrequent nouns (for reviews and commentary, see Goodglass, 1993; McRae & Cree, 2000; Shallice, 1988). Interestingly, in a series of simulations of individual aphasic patients' naming errors, Dell, Schwartz, Martin, Saffran, and Gagnon (1997) showed that dramatically different patterns of lexical processing breakdown in a single connectionist network can occur, depending on the severity and kind of degradation imposed. Despite these variations and regardless of their cause, one conclusion is clear: All aphasic patients, including agrammatic Broca's aphasics, display word-finding deficits. This does not imply that aphasic patients have lost their lexical "representations"—indeed, they often have detailed semantic knowledge of lexical items (Damasio, Grabowski, Tranel, Hichwa, & Damasio, 1996). As we show later, this preservation of knowledge despite deficits in processing is also seen in the grammatical realm.[3]

It should also be noted that lexical-processing deficits in aphasia are not limited to language production. Recent evidence from semantic priming studies suggests that a number of aphasic groups show significant impairments in the comprehension of lexical items. In particular, Broca's aphasic patients tend to show reduced or delayed semantic priming relative to normal participants, a pattern which has led a number of researchers to propose that the activation of lexical-semantic representations is disrupted in these patients (Milberg, Blumstein, & Dworetzky, 1987, 1988; Prather, Shapiro, Zurif, & Swinney, 1991; Utman, Blumstein, & Sullivan, in press; cf. Utman & Bates, 1998). Furthermore, electrophysiological studies of semantic priming in Broca's aphasia have suggested that these patients are impaired in the contextual selection and integration of lexical information (Swaab, Brown, & Hagoort, 1997, 1998). In contrast, Wernicke's patients demonstrate abnormally large semantic priming effect relative to normal participants, and can show robust semantic facilitation under conditions that produce only weak priming in normal individuals (Milberg et al., 1987, 1988). The pervasiveness of anomic symptoms in all forms of aphasia, coupled with the lexical-semantic activation disturbances apparent in both Broca's and Wernicke's aphasia, means that there can be no evidence for a full double dissociation between grammatical and lexical deficits (for further details, see Bates & Goodman, 1997).

Of course this does not mean that grammatical and lexical deficits are fully co-extensive. There are some patients whose grammatical deficits appear to be more severe than their lexical

[3] It could be argued that Broca's aphasic patients have a primary grammatical deficit, and that any word-finding problems they display are secondary to this grammar deficit, resulting from, for example, absence or reduction in the grammatical cues that help normal speakers and listeners to retrieve the appropriate content word. Although this possibility cannot be ruled out altogether, we note that naming problems are reliably observed in Broca's aphasic patients in confrontation naming tasks, in which lexical items must be retrieved in isolation, outside of a sentence context. These lexical problems tend to be greater for verbs than for nouns, but they are observed reliably for all word types.

problems, and vice versa. Nevertheless, as Bates and Goodman (1997) have pointed out, even in these cases there are often striking similarities in the kinds of deficits that occur in each domain. For example, jargon aphasic patients who make frequent lexical substitutions are also prone to substitutions in the grammatical domain (e.g., substituting one inflection or function word for another). Nonfluent patients who tend to err by omission in the grammatical domain show a parallel pattern of word finding (i.e., lexical omissions are far more common than lexical substitutions). Mild anomic patients who tend to overuse pronouns and light forms in the lexical domain (e.g., "That thing, that guy who does that" rather than "That boy who is kicking the dog") show a parallel tendency to avoid complex syntactic forms (e.g., passives) in favor of simpler and more frequent syntactic constructions. Hence there are qualitative as well as quantitative links between lexical and grammatical deficits in aphasic patients, even though the correlation in severity across domains is imperfect.

Broca's Aphasic Patients Still "Know" Their Grammar

Evidence for the preservation of grammatical knowledge in agrammatic Broca's aphasic patients has been reviewed in considerable detail elsewhere (Bates & Wulfeck, 1989; Bates, Wulfeck, & MacWhinney, 1991; Menn, Obler, & Miceli, 1990). For present purposes, the main findings can be summarized as follows.

First, a large number of studies have shown that Broca's aphasic patients who meet the usual criteria for both receptive and expressive agrammatism are still able to recognize grammatical errors in someone else's speech at above-chance levels (Devescovi et al., 1997; Linebarger, Schwartz, & Saffran, 1983; Shankweiler, Crain, Gorrell, & Tuller, 1989; Wulfeck, 1988). Although their performance is certainly well below that of normal control participants, the fact that agrammatic Broca's aphasic patients retain above-chance sensitivity to grammatical well-formedness is difficult to reconcile with the idea that grammatical knowledge is stored in or around Broca's area.

Second, cross-linguistic studies have shown that the expressive and receptive symptoms displayed by nonfluent Broca's aphasic patients differ markedly from one language to another, in ways that can only be explained if it is assumed that language-specific knowledge is preserved. For example, cross-linguistic differences have been observed in the order in which words and morphemes are produced; for example, subject–verb–object (SVO) orders predominate in aphasic speakers of SVO languages such as English, German, and Italian (Bates, Friederici, Wulfeck, & Juarez, 1988), whereas subject–object–verb (SOV) orders predominate in aphasic speakers of languages such as Turkish in which the most common word order is SOV (Slobin, 1991).

Cross-language differences are also observed in the retention or omission of grammatical inflections and function words in contexts where these inflections are required. For example, German Broca's aphasic patients produce a higher proportion of articles in obligatory contexts than English Broca's aphasic patients, reflecting the crucial role of the article as a carrier of case information in German (Bates, Friederici, & Wulfeck, 1987b). This trend toward language-specific patterns of retention and omission is also seen in the production of features such as tone in Chinese (Tzeng, Hung, & Bates, 1996), or pragmatic word order variations in Hungarian (MacWhinney & Osmán-Sági, 1991). Parallel differences between languages have been observed in cross-linguistic studies of receptive language processing (Bates, Wulfeck, et al., 1991); for example, differences in the degree to which patients based their sentence interpretations on word order (high in English, low in languages such as Italian with extensive word order variation) versus grammatical morphology (low in English, higher in languages such as German, Italian, Turkish, and Hungarian). In short, it turns out that it is surprisingly difficult to "take the Turkish out of the Turk, and the English out of the Englishman."

Such results are compatible with predictions of the competition model (MacWhinney & Bates, 1989), an interactive-activation model of language processing that emphasizes the role of *cue validity* and *cue cost* in predicting the strength or vulnerability of linguistic structures in brain-injured patients. Cue validity refers to the information value of a particular source of information in a given language. Cue cost refers to the costs involved in processing that piece of information, including variations in perceptual salience and imageability (factors that raise the costs of processing inflections and function words), as well as variations in informational and integrational load (e.g., the number of actors in a sentence or the special demands associated with the processing of subject–verb agreement, where two agreeing elements may be separated by more than one intervening phrase or clause, as in "The *boy* who we told you about last night *is coming*"). Cue cost can also be determined by the *frequency* of a particular informational structure in the language environment—a point we return to below. The competition model predicts that linguistic cues high in validity and low in cost (such as SVO word order in English) will be relatively spared in aphasia, whereas low-validity, high-cost cues (like English subject–verb [SV] agreement) will be vulnerable to brain injury. These predictions have been largely borne out in tests of aphasic patients in more than a dozen languages (Bates & Wulfeck, 1989).

Returning to the point at hand, we have established (a) that grammatical deficits always co-occur with lexical deficits in aphasia and (b) that patients with expressive and receptive agrammatism still retain sensitivity to the details of their native grammar, in ways that are predicted by the cue validity or information value of grammatical forms in that language. Hence the grammatical deficits observed in Broca's aphasia are a matter of degree, and are not dissociable from lexical processing. Despite these limitations, one could salvage the argument that grammatical knowledge or some specific component of grammatical processing is localized in and around Broca's area by showing that damage to this area produces a particularly severe disruption of grammar, or perhaps a unique form of grammatical impairment that is not observed in other clinical populations. We now turn to the case for a "special" grammatical deficit in Broca's aphasia.

Grammatical Deficits in Patients Without Broca's Aphasia

In modern studies of grammatical processing in aphasia, the term *agrammatic aphasia* is often used interchangeably with *Broca's aphasia,* implying that the most severe or the most selective and specific form of agrammatism is the one found in nonfluent patients with left frontal pathology. Yet a careful look at the literature across languages and populations reveals that this assumption is false: Both expressive and receptive forms of agram-

matism are found in Wernicke's aphasia, and in many other language disorders. Evidence relevant to the pervasiveness of grammatical deficits in aphasia can be garnered from three research areas: expressive agrammatism, deficits in the perception and comprehension of grammatical morphology, and deficits in the receptive processing of complex syntax.

Expressive Agrammatism

From the two examples of speech by English-speaking aphasic patients that we just provided, it seems fair to conclude that the nonfluent Broca's aphasic patient suffers from a marked deficit in the ability to produce grammatical forms. By contrast, the grammatical abilities of the Wernicke's patient appear to be relatively intact despite his or her severe word-finding problems (but note that subcategorization violations and morphological errors are present in this example). Although this seems to provide compelling evidence for the dissociability of grammatical and lexical production, cross-linguistic evidence suggests that this apparent dissociation is an artifact of English. Studies of speech production in richly inflected languages show that Wernicke's aphasic patients make grammatical errors similar in quantity and severity to the errors produced by Broca's aphasic patients, although there are interesting differences between the two syndromes in terms of the form these errors take.

This point was first made by Arnold Pick (1913/1973), who originated the term *agrammatism.* On the basis of his observations with German- and Czech-speaking patients, Pick noted that there are two forms of agrammatism: (a) a nonfluent form—characterized by omission and reductions in complexity—that is usually associated with frontal lesions and (b) a fluent form—involving substitutions of one grammatical form for another (paragrammatism)—that is typically associated with posterior (temporal lobe) lesions. In Pick's view (1913/1973), the fluent (temporal) form of agrammatism is actually the more interesting of the two:

> Temporally determined expressive agrammatism is characterized by erroneous grammatical constructions (paragrammatisms), in contrast to the frontal type with its telegraphic style. . . . This temporally determined form is characterized, in pure cases, by disturbances in the use of auxiliary words, incorrect word inflections, and erroneous prefixes and suffixes. . . . In contrast to motor agrammatism, the tempo of speech is not retarded, tending rather to logorrhea with intact sentence pattern and intonation. Occasionally some motor (i.e., telegrammatic) phenomena are found, such as the dropping of inflections, with juxtaposition of the words which comprise the skeleton of the sentence. (pp. 76–77, also cited in Bates, Friederici, & Wulfeck, 1987a)

As we have noted in other reviews (Bates & Goodman, 1997; Bates & Wulfeck, 1989; Bates, Wulfeck, et al., 1991), the English system of grammatical morphology is so impoverished that it offers few opportunities for grammatical substitution errors. This is not the case in languages such as Italian, German, Turkish, Hungarian, or Serbo-Croatian, where the substitution errors observed in Wernicke's aphasia are as obvious today as they were to Pick. To illustrate this point, consider the following passages from three Wernicke's aphasic patients (one English, one Italian, and one German) who are trying to describe the same set of three-picture cartoons in which a cat is giving a flower to a rabbit, dog, or boy. Morphological and lexical substitutions are indicated by asterisks. (Examples are adapted from Bates et al., 1987b.)

> *English:* "And the dog* is doing* the flower to the bagetle* . . . rabbit."
> *Italian:* "Allora, questo è il coso* che, come si chiama, il gattino che porta la*, il coso* al coniglio." (Translation: "Well, this is the thing* that, how is called, the kitty that brings the* thing* to the rabbit.")
> *German:* "Der*, der*, die, die Katze beschenkt ein* Mann oder den Jungen ein* Bibel (Bibel, ja ja). (Translation: "The*, the*, the, the cat gives a* man or a boy a* bible*.")

As should be clear from these examples, the Italian and German Wernicke's aphasic patients produce a kind of substitution error (paragrammatism) that is observed in English patients only when the patient is struggling to retrieve a content word (paraphasia). When a language has structures that permit morpheme substitutions to emerge, it becomes apparent that there are striking parallels in the lexical and grammatical errors produced by Wernicke's aphasics—in short, a tendency to err by substitution, replacing the intended item with one that is a close semantic and/or phonological neighbor.

On the basis of such results, and on related results from fluent and nonfluent forms of language impairment in children, Bates and Goodman (1997) have proposed that omission and substitution errors lie along a continuum, and that individual patients at a particular point on this continuum tend to produce the same kinds of errors at both the lexical and the grammatical levels (see also Kolk & Heeschen, 1992). When speech production is exceptionally slow, both lexical and grammatical items may fail to reach or maintain the levels of activation required for normal production, resulting in a profile characterized by omission of weak elements. Conversely, when a speaker produces language at a rate that exceeds his or her central processing capacity, errors of commission (e.g., substitution) are more likely to occur than errors of omission. For example, if a Wernicke's aphasic patient produces speech at a rate comparable to a neurologically intact speaker, he or she may exceed the capabilities of the processing system. In support of this view, Bates, Appelbaum, and Allard (1991) have shown that speech rate is correlated significantly with the occurrence of substitution errors in agrammatic speakers of Italian. In the same vein, Kolk and Heeschen have shown that substitution errors increase when individual patients are forced to produce speech at a more rapid rate. Findings like these lead to the suggestion that the contrast between omission and substitution errors may reflect a speed–accuracy trade-off, representing a language-specific manifestation of a general phenomenon that is well characterized in the literature on attention and performance.

If this argument is correct, then it offers a new explanation for the contrasting forms of expressive agrammatism observed in Broca's and Wernicke's aphasia patients. According to this view, the contrast between the omission errors of Broca's aphasia patients and the substitution errors of Wernicke's aphasia patients lies not in grammar per se, but in the striking differences in rate of speech that are used to define these syndromes. As Bates and Goodman (1997) noted, this argument leads to the prediction that a similar contrast in error types should be observed in other syndromes that differ in relative fluency. This does, in fact, seem to be the case: Omission errors predominate in several different low-fluency syndromes, including Broca's aphasia, Down's syn-

drome and specific language impairment, whereas commission errors are more often observed in high-fluency syndromes, including Wernicke's aphasia and Williams syndrome.

Most important for our purposes here, there are simply no grounds for the argument that expressive agrammatism is uniquely associated with Broca's aphasia, or that the expressive language deficits in Broca's are restricted to grammar. Rather, grammar and lexical semantics are impaired in both fluent and nonfluent language disorders, with the disruption taking the form of omission errors in nonfluent syndromes, and commission–substitution errors in fluent syndromes.

Receptive Agrammatism: Grammatical Morphology

The argument that Broca's aphasia represents a kind of central agrammatism was based in large measure on the parallels observed in the expressive and receptive language abilities of these patients. The very elements that are missing or impaired in the telegraphic speech of Broca's aphasic patients are the ones that these patients find most difficult to process in receptive language tasks. For example, Heilman and Scholes (1976) used a picture-pointing task to show that Broca's aphasic patients have difficulty distinguishing between sentences that turn on a single function word (e.g., "He showed her the baby pictures" vs. "He showed her baby the pictures"). In a study that falls somewhere between expressive and receptive processing, von Stockert and Bader (1976) asked Broca's aphasic patients to construct sentences out of cards representing individual content words and function words. Their patients were able to produce strings of content words in an appropriate order (mirroring results cited previously regarding the preservation of canonical word order). However, they were markedly impaired in the use and placement of grammatical function words, or *closed-class morphemes,* often leaving the cards carrying words such as *the* and *of* off to the side in an unorganized pile.

These deficits in the processing of freestanding function words co-occur with deficits in the receptive processing of bound inflections, especially the inflections required to compute agreement. Selective deficits in the processing of agreement morphology have been reported across receptive tasks, including grammaticality judgment (Devescovi et al., 1997; Mauner et al., 1993, in a reanalysis of Linebarger et al., 1983) and sentence comprehension (Bates et al., 1987a; MacWhinney, Osmán-Sági, & Slobin, 1991).

This point is illustrated in Figures 1A and 1B (redrawn from Bates et al., 1987a), which show that English, Italian, and German Broca's aphasic patients are all markedly impaired in their ability to use agreement morphology in interpreting simple sentences, although the magnitude of the deficit is proportional to the strength of grammatical morphology in the patient's native language. Aphasic and control participants were presented with grammatical sentences or sentence fragments in which transitive action verbs agreed with the first noun (e.g., "The horse is pushing the cows"), the second noun (e.g., "The horse are pushing the cows"), or both nouns (e.g., "The horse is pushing the cow"). If participants rely heavily on subject–verb agreement to make their interpretations, then they should choose the first noun close to 100% of the time when the first noun agrees, and close to 0% of the time when the second noun agrees (collapsing across word order conditions, which included noun–verb–noun, noun–noun–verb, and verb–noun–noun items). Figure 1A shows that Italian-speaking normal participants relied mostly on subject–verb agreement, whereas their English-speaking counterparts relied very little on this cue; German speakers fell somewhere between these two poles. Figure 1B shows that this cross-language difference is still operating in Broca's aphasic patients (reflected in a significant interaction between language and agreement conditions), although patients in all three language groups relied less on agreement than normal control participants.

The relative vulnerability of morphology observed in sentence comprehension and judgment tasks mirrors the pattern of spared word order and impaired morphology that is typically observed in language production by the same patients. To account for this selective profile, a number of investigators in the early 1980s proposed the *closed-class theory of agrammatism* (Bradley, Garrett, & Zurif, 1980; Kean, 1985), restricting the central deficit of Broca's aphasia to the representation and processing of bound morphemes and freestanding function words. However, as Goodglass (1993) pointed out in his brief history of this period, most of the studies cited in support of the closed-class theory of agrammatism restricted their comparisons only to Broca's aphasic patients and age-matched normal control participants. In fact, by the end of the 1980s it became increasingly apparent that selective deficits in grammatical morphology occur in other aphasic populations as well.

The existence of such deficits across aphasic groups was illustrated by the use of agreement cues of Italian-speaking college students, Broca's aphasic patients, neurological control participants (e.g., patients with polio or myaesthenia gravis), and non-neurological control participants (i.e., patients from the orthopedic ward) in Bates et al. (1987a). Although there were variations in severity over groups, all patient groups—not just brain-damaged aphasic patients—display a significant decrease in their ability to use subject–verb agreement when they are compared with healthy young control participants. In other words, receptive agrammatism can even occur as a consequence of hip fracture. Results such as this (for results in Hungarian and Turkish, see MacWhinney et al., 1991) strongly suggest that impairments in the comprehension of SV agreement occur because these elements constitute a weak link in the chain of language processing, one that can be disrupted for many reasons. This particular form of receptive agrammatism is not unique to Broca's aphasia, or for that matter, to any kind of aphasia.

Receptive Agrammatism: Complex Syntax

As noted earlier, beginning in the 1980s Broca's aphasia began to be regarded as a "testing ground for theoretical models of the normal mental grammar" (Mauner et al., 1993, p. 340). Case studies of Broca's aphasic patients' comprehension of diverse syntactic structures have been used to vet increasingly specific claims concerning the role of Broca's area in syntactic processing. One area of particular interest has been Broca's aphasic patients' comprehension of syntax involving "transformational movement" (Beretta, Piñango, Patterson, & Harford, 1999; Berndt & Caramazza, 1999; Berndt, Mitchum, & Haendiges, 1996; Caplan & Futter, 1986; Caramazza & Zurif, 1976; Druks & Marshall, 1995, 2000; Grodzinsky, 1995a, b, 2000; Grodzinsky, Piñango, Zurif, & Drai, 1999; Hickok & Avrutin, 1995; Kean, 1995; Mauner et al., 1993; Schwartz, Saffran, & Marin, 1980; Thompson, Tait, Ballard,

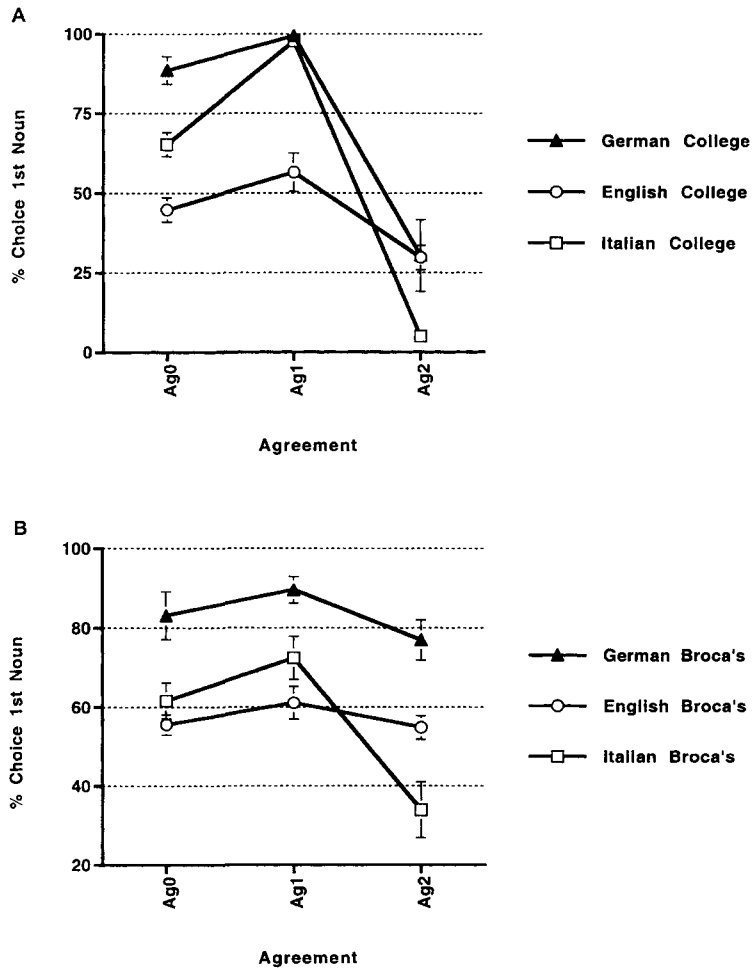

Figure 1. A: Word Order × Agreement Cue interaction for German-, Italian-, and English-speaking college students. B: Word Order × Agreement Cue interaction for German-, Italian-, and English-speaking aphasic patients. Error bars represent ±1 *SEM*. Ag = agreement. From "Comprehension in Aphasia: A Cross-Linguistic Study," by E. Bates, A. Friederici, and B. Wulfeck, 1987, *Brain and Language, 32*, p. 39. Copyright 1987 by Harcourt, Inc. Adapted with permission.

& Fix, 1999). According to this view, noncanonical orders, such as passives and object relatives, are derived by moving elements from their canonical place in deep structure (*or D structure*) to a more marked position in surface structure (*or S structure*). Moved elements leave behind a kind of footprint or *trace* that is co-indexed to mark its original position; these traces are used by normal listeners to reconstruct the underlying logical structure of the sentence. In agrammatic (Broca's) patients, some researchers have suggested that this ability to perceive traces and perform the necessary operation of co-indexation is lost because of injury to a particular module of the grammar (Grodzinsky, 1995a, b, 2000).

Strong claims have been advanced regarding the locus and specificity of Broca's aphasic patients' deficits in this regard. In a recent article, Grodzinsky (2000) made the following claim:

> Mental modularity, moreover, is also a property of syntax itself: the neurology indicates that syntax is not supported by one piece of neural tissue. Within this picture, syntax is entirely represented in the left cerebral hemisphere, but for the most part, it is not located in Broca's area. This cerebral region, so the evidence suggests, has a crucial, highly specific role: it is neural home to mechanisms involved in the computation of transformational relations between moved phrasal constituents and their extraction sites. (p. 2)

Grodzinsky (2000) further suggested that Broca's-related deficits will be evidenced by chance performance on transformed structures, with above-chance comprehension of nontransformational syntax (for discussion of these claims, see Berndt & Caramazza, 1999; Drai & Grodzinsky, 1999; Grodzinsky et al., 1999). If these hypotheses are true, one should expect that damage to the right hemisphere should cause no deficits in syntactic comprehension. Furthermore, syntactic deficits (particularly those involving transformation) should map relatively transparently to lesion site.

Caplan and his colleagues have conducted a series of large-scale studies on syntactic comprehension directly relevant to these claims. Caplan, Baker, and Dehaut (1985) administered a sentence comprehension task to 150 French- or English-speaking patients, testing 12 different syntactic types of differing complexity. They

initially predicted that syntactic deficits would be more prominent in nonfluent patients with frontal lesions. Instead, they found that patients varied along a single dimension of comprehension severity, with no unique associations between severity and lesion location. A series of post hoc cluster analyses suggested that the data might reflect syntactic comprehension subtypes, but these types also failed to correlate with any particular lesion site. Such results are consonant with earlier reports of syntactic comprehension deficits in patients other than Broca's aphasics (Caplan & Hildebrandt, 1988; Tramo, Baynes, & Volpe, 1988; see Dronkers, Redfern, & Knight, 2000, for an overview of lesion-language relationships). Caplan et al. (1996) expanded on these earlier findings, testing the syntactic comprehension of 60 English- or French-speaking aphasic patients (as well as 21 normal control participants) on an exhaustive battery of 24 sentence types, each represented by 20 tokens. Caplan et al. found that both right- and left-hemisphere damage caused deficits in syntactic processing, with comprehension of more complex syntactic structures significantly more affected than simpler ones. Consistent with many previous studies (for a review, see Goodglass, 1993), left-hemisphere damage had a greater impact on syntactic comprehension than did right-hemisphere damage. However, in a comprehensive analysis of syntactic complexity, lesion extent, volume, and location did not predict degree of syntactic deficit. In stark contrast to Grodzinsky and associates, Caplan et al. (1996) concluded that, within the bounds of the lesion–correlation methodology, there appears to be no special relationship between damage to Broca's area and syntactic processing—or any other area, for that matter. They suggest that, on the basis of functional imaging studies, syntactic processing preferentially (but not always) resides in the perisylvian region of the left hemisphere, with a lesser, but significant, contribution from the right hemisphere. They further suggested that the location of processing resources for language may be subject to wide individual variation because of age, education, gender, and other variables (cf. Caplan, 1987).

Additional evidence regarding the opacity of lesion-to-behavior mapping comes from the landmark studies of Metter, Kempler, and colleagues (Metter, Hanson, Jackson, & Kempler, 1990; Metter, Jackson, Kempler, & Hanson, 1992; Metter, Kempler, Jackson, & Hanson, 1987; Metter, Kempler, Jackson, & Hanson, 1989; Metter, Riege, Hanson, & Jackson, 1988). Using a combination of structural (computerized tomography [CT]) and resting perfusion (positron emission tomography [PET]) neural imaging data, Metter et al. elucidated the metabolic consequences of focal lesions in large groups of aphasic patients, showing that the locus and extent of structural lesions (as detected by CT) often vastly underrepresented true functional anomalies (as measured by hypoperfusion in the affected hemisphere). Of particular interest is the relationship between structural lesions and hypoperfusion on the one hand, and language deficits on the other. In this regard, Kempler, Metter, Curtiss, Jackson, and Hanson (1991) examined CT and PET data from 43 aphasic patients, correlating the neural findings to overall language deficit (using the Western Aphasia Battery [WAB], Kertesz, 1982) as well as to deficits in morphosyntactic comprehension (Curtiss–Yamada Comprehensive Language Evaluation—Receptive Measures [CYCLE–R]; Curtiss & Yamada, 1987). As in Caplan's studies (Caplan et al., 1985, 1996; Caplan & Hildebrandt, 1988), deficits in complex syntax comprehension were present in all aphasic groups, with Broca's and Wernicke's aphasic patients even more affected than anomic and conduction aphasic patients. Interestingly, *structural* damage in the two "classical" language areas—the left middle/superior temporal lobe (Wernicke's) and the left inferior frontal lobe (Broca's area)—did appear to weakly correlate with *degree* (not type) of syntactic (but not morphological) deficit. However, a much different picture emerged from the PET data: Deficits in both morphology and syntax were highly correlated with hypometabolism in the left occipital and temporal regions; syntactic deficits were further highly correlated with parietal hypometabolism. These results are in keeping with this group's previous findings of left temporal hypometabolism in all aphasic groups (Metter et al., 1989), as well as with current data from functional neuroimaging studies in normal participants (Bates et al., 2000). The resting metabolism findings suggest that aphasic patients' morphosyntactic deficits may be the product of functional abnormalities in large interconnected regions of cortical and subcortical areas (see Lieberman, 2000, for an overview of subcortical contributions to syntactic processing).

Given the results cited earlier, it is clear that deficits in the ability to process closed-class words and bound grammatical morphemes occur in both fluent and nonfluent aphasics, and in some nonaphasic patients as well. It is equally as clear that deficits in syntactic comprehension can occur with damage to either hemisphere; moreover, the locus of such damage within the left hemisphere appears to have little or no predictive value vis-à-vis syntactic deficits. Finally, both the lack of syntactic localizability and the extent of hypofunctional cortical and subcortical regions in aphasic patients suggest that syntactic deficits may arise from a generalized "stress" on processing resources.

In the following section, we first review *processing-based* accounts of aphasia, and then survey the handful of studies aimed at simulating such morphological and syntactic deficits in healthy control participants through the imposition of adverse processing conditions.

Simulations of Receptive Agrammatism in Normal Participants Under Stress

In view of the evidence pointing toward the nonlocalizability of grammatical deficits as well as their graded and varied character, several groups have proposed alternatives to the loss-of-representation account of syntactic comprehension deficits. Caplan and Waters (1999) theorized that such deficits come about not through loss of linguistic knowledge (e.g., loss of traces), but through loss of a separate language interpretation resource (SLIR). The SLIR is computational space dedicated exclusively to grammar in which language rules are processed separately from other information. Noncanonical structures such as the passive or object cleft require more of this syntax-specific resource, perhaps because of the number and kind of movement operations involved. According to this account, syntactic deficits reflect the degree to which the language-specific working memory module is damaged. Caplan and Waters further proposed that additional *sentence*-processing deficits may be due to loss of a more general verbal working memory responsible for computations involving propositional knowledge; we discuss this distinction in the following paragraph.

Just and Carpenter (1992; Just, Carpenter, & Keller, 1996) proposed a production system model that resembles the Caplan

and Waters (1999) account in attributing deficits in syntactic processing to a reduction in processing resources. However, Just and Carpenter argued that language processing is carried out by a *general verbal* (language-, but not syntax-specific) working memory, where "procedures," contextual information, and other cues can be integrated in parallel. Although Caplan and Waters predicted that an individual's syntactic working memory capacity is independent from working memory resources subserving other language skills, Just and Carpenter proposed that syntactic ability should positively covary with general verbal working memory capacity (as measured by memory span tasks; Daneman, Carpenter, & Just, 1982). Hence, the extent to which selective syntactic deficits are observed should be predicted by such memory span measures—a matter of current debate between these two schools of thought (Caplan & Waters, 1999; Just & Carpenter, 1992; Just et al., 1996; Miyake, Emerson, & Friedman, 1999; Waters & Caplan, 1996).

Like both of the production system models, connectionist accounts of language processing and breakdown (such as the competition model described previously) emphasize processing decrements—rather than knowledge loss—as the main cause of syntactic deficits in aphasia. In contrast to both production system accounts, the performance of connectionist models of syntactic processing is highly contingent upon the distributional information present in the linguistic environment. Particularly important are the *frequency* and *regularity* of a given syntactic structure. Frequency refers to the rate a single structure occurs, whereas regularity refers to the extent the structure "patterns" with other similar structures (see MacDonald, Pearlmutter, & Seidenberg, 1994, for an extended discussion). For instance, the subject cleft structure ("It's the actor that is hitting the director") is rare in informal discourse; however, the similar subject relatives ("The actor that is hitting the director") are relatively frequent, and the underlying word order (subject [actor]–verb [hit]–object [director]) is by far the dominant one in English (Dick & Elman, 2001). Thus, a connectionist account would predict that structures with an underlying SVO word order would remain relatively impervious to brain damage, whereas structures such as center-embedded object relatives ("The actor that the producer kicked liked the comedian"), which contain a low-frequency word order (OSV), should be difficult for aphasics to comprehend. This prediction follows from previous work on inflectional morphology (Marchman, 1993) and syntactic comprehension (St. John & Gernsbacher, 1998), showing that diffuse damage to a distributed neural network (in the form of altered weights or injected "noise" [McClelland, 1993]) can cause seemingly discrete deficits in processing. Importantly, the character of these deficits is directly related to the frequency and regularity of the structures in question.

As we noted in the introduction, connectionist and distributive approaches emphasize that language processing emerges from, and shares neural resources with, more evolutionarily entrenched sensorimotor substrates. Therefore, one would expect that damage to these sensorimotor substrates might have deleterious effects on higher order language processing. Indeed, studies of children with both congenital motor impairments (Alcock, Passingham, Watkins, & Vargha-Khadem, 2000a, b; Vargha-Khadem, Watkins, Alcock, Fletcher, & Passingham, 1995) and auditory processing deficits (Merzenich et al., 1996; Tallal et al., 1996) show that such nonlinguistic syndromes may incur profound, high-level linguistic impairments in language development. In adults, aphasia (particularly of the expressive or agrammatic type) co-occurs with motor apraxia at extremely high rates (Kertesz, 1979; Kertesz & Hooper, 1982); links between auditory processing and receptive aphasia are suggestive, but less clearly drawn (Alcock, Wade, Anslow, & Passingham, 2000; Clarke, Bellmann, Meuli, Assal, & Steck, 2000; Varney, 1984a, b; Varney & Damasio, 1986). (We hasten to note that aphasia does not entail a global profile of deficits over modalities; for instance, color priming—a low-level visual task—is preserved in both fluent and nonfluent Italian-speaking aphasic patients who are entirely impaired in gender priming; see Bates, Marangolo, Pizzamiglio, & Dick, 2001). If it is the case that selective deficits in grammar reflect demands on factors that lie outside of "language proper," then it should be possible to reproduce these patterns in normal adults by subjecting them to exogenous processing constraints that mimic the endogenous processing conditions that may be a cause of receptive agrammatism.

To the best of our knowledge, the first test of this hypothesis was performed by Kilborn (1991). Following up on the findings for English, Italian, and German patients reported by Bates et al. (1987a) discussed earlier, Kilborn suggested that English- and German-speaking college students might present with a similar pattern if they had to comprehend auditorily presented sentences under a partial noise mask. Specifically, normal listeners under noise should decrease their use of subject–verb agreement, as do aphasic listeners; use of word order information should remain at normal levels and may actually increase to compensate for the loss of morphology, again as is observed in aphasia.

Kilborn's (1991) design was similar to that of Bates et al. (1987a; Figures 1A, 1B) with the following additions: (a) sentences were presented on-line in a reaction time paradigm and (b) two different morphologically ambiguous conditions were used—one in which both nouns agreed with the verb, and another in which neither noun agreed with the verb. Within each language, college students were randomly assigned to a normal or a noise condition. Figures 2A and 2B (redrawn from Kilborn, 1991) demonstrate the effects of the noise mask on use of word order and agreement cues, respectively, effects reflecting significant Language × Noise interactions that roundly confirm Kilborn's predictions. In line with cue validity predictions of the competition model, the impact of agreement cues is much larger in German than it is in English under either normal or noisy conditions, whereas use of word order is larger in English. However, the noise mask had a massive effect on use of agreement in both languages, wiping it out entirely in English (where it was weak to begin with), and reducing it to levels similar to those observed with aphasic patients in German. By contrast, noise had little or no effect on the use of word order. If anything, the use of word order was slightly enhanced (especially in German) under the noise condition. We should note that these effects may be due, at least in part, to the relatively low acoustical and phonological salience of agreement cues compared with word order cues; therefore, the observed deficits might be the results of a procedural artifact, rather than a true "simulation" of aphasic deficits. However, the following study suggests that the vulnerability of agreement morphology in such simulations is not due solely to perceptual factors.

In a series of experiments from our laboratory published only in abstract form (Bates et al. 1994), we have taken Kilborn's (1991)

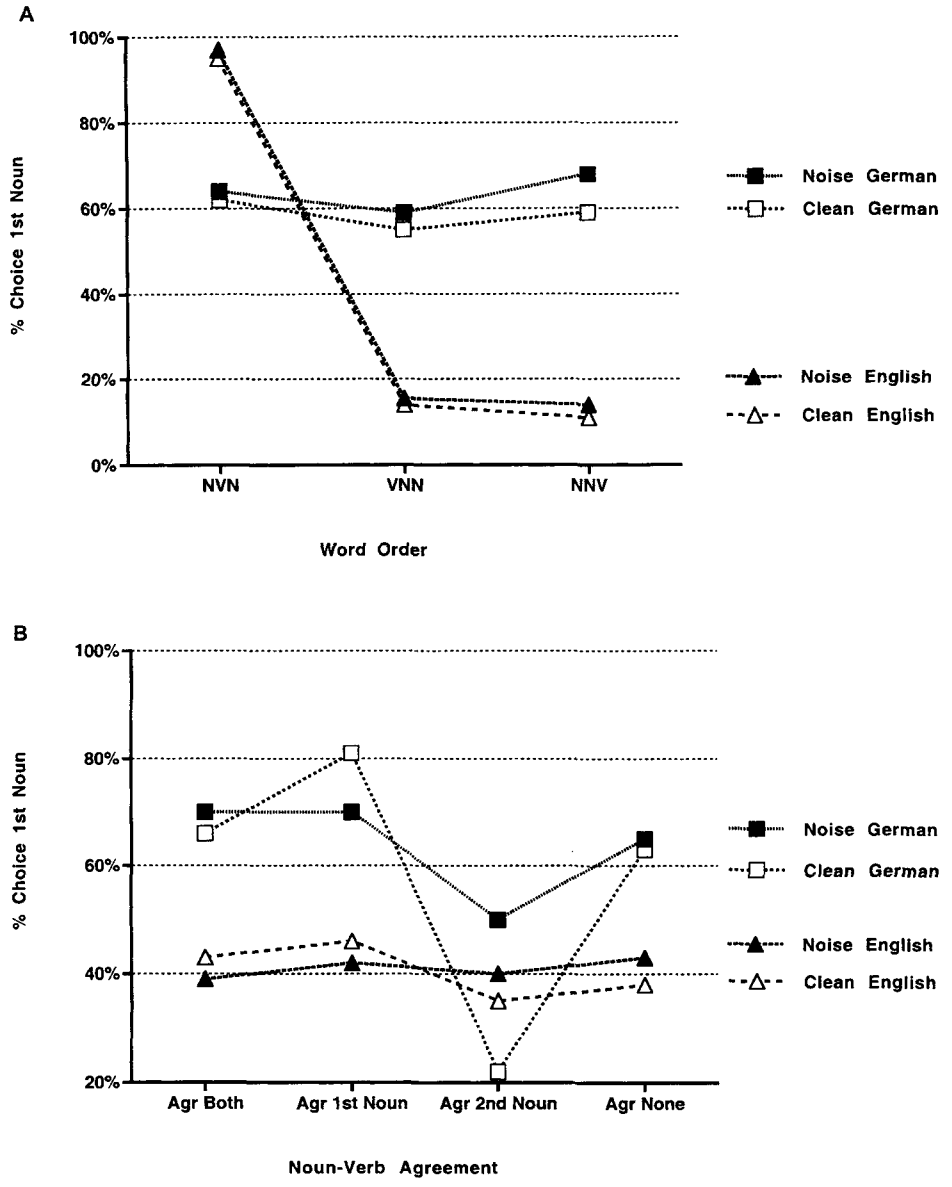

Figure 2. A: First-noun choice as a function of word order in English and German under clean and noise conditions. B: First-noun choice as a function of noun–verb agreement in English and German under clean and noise conditions. NVN = noun–verb–noun; VNN = verb–noun–noun; NNV = noun–noun–verb: Agr = agreement. From "Selective Impairment of Grammatical Morphology Due to Induced Stress in Normal Listeners: Implications for Aphasia," by K. Kilborn, 1991, *Brain and Language, 41*, pp. 282–283. Copyright 1991 by Harcourt, Inc. Adapted with permission.

approach with a different pair of languages and a different form of stress. On-line auditory versions of the English and Italian stimuli used by Bates et al. (1987a) were administered to college students, older control participants, and aphasic patients. All participants were asked to decide "who did the action" in the sentence, pressing a button under one of the two pictures ("mugshots") representing the nouns in the sentence. College students were tested under normal listening conditions, or with a digit load. In the digit load version, participants viewed a random sequence of two to six digits presented in rapid serial visual presentation (RSVP) prior to each sentence stimulus. After they made their sentence decision, they were given the same digit sequence or a sequence that differed by only one digit and were asked to press a button indicating whether the digit sequence was the same or different.

Cross-linguistic results paralleled those obtained in other studies: Italians in all groups relied much more on subject–verb agreement to make their decisions than English listeners, whereas English participants relied far more on word order. Again, however, the aphasic patients in each language (and, to a lesser extent, the older control participants) showed significantly less use of subject–verb agreement than normal participants. Most interesting for our purposes here, college students in the digit condition also

showed a significant decrease in the use of agreement information. In English, this decrement in agreement was compensated for by an even greater use of word order; in Italian, the decrement in agreement was accompanied by an increase in the use of animacy information (a third variable in the study).[4] These results replicate the cross-linguistic findings for aphasic patients reported in earlier studies. In addition, they show that the selective vulnerability of morphology can be created through cognitive overload in this dual-task situation, paralleling the results by Kilborn (1991) in which a noise mask was used. These results also demonstrate that the vulnerability of agreement morphology in such simulations is not a purely sensory phenomenon. The mechanisms involved in these dual-task effects are currently unclear; we speculate that the digit load may tie up the phonological loop (see Baddeley, Gathercole, & Papagno, 1998, and Caplan & Waters, 1999, for a discussion of the importance of the phonological loop in language learning and comprehension).

Blackwell and Bates (1995) used the digit load manipulation described previously in a grammaticality judgment task, similar to that of the Wulfeck and Bates (1991) study of aphasic patients. Here, in addition to the agreement and word order violations used in Wulfeck and Bates, there were also violations involving auxiliary or determiner omission (e.g., "She is selling several rare paintings" → "She selling several rare paintings"—see Figure 3). The digit load manipulation caused participants to make more errors in detecting all types of violations, with word order, omission, and agreement errors all detected significantly less accurately under a six-digit load than under no digit load. The agreement violations appeared to be particularly vulnerable, as post hoc tests showed participants to be less accurate even in the two-digit condition.

The results cited previously attest to the generality of the findings obtained in sentence comprehension tasks: Aphasic-like selective deficits in the use of morphology (especially agreement morphology) can reliably be reproduced in normal control participants under various forms of stress, including perceptual degradation and cognitive overload. The results of Blackwell and Bates (1995) suggest that detection of a subclass of syntactic violations (word order substitutions and omissions) can also be hindered under stress. However, as we note previously, aphasic patients have problems not only in detecting syntactic violations, but also in simply comprehending some grammatical syntactic structures (such as passives and object relatives). The possibility of simulating such syntactic comprehension deficits was first explored by Miyake, Carpenter, and Just (1994), who administered complex sentence stimuli (derived from those of Caplan & Hildebrandt, 1988) to college students in a serial visual presentation format, in which each word was briefly shown on a video screen in sequential order. Half of their participants received the stimuli at a comfortable presentation rate, and the other half read the sentences under speeded visual presentation (the RSVP task). Students in the RSVP condition produced significantly more errors and displayed a hierarchy of difficulty that was strikingly similar to that of Caplan and Hildebrandt's aphasic patients. Post hoc cluster analyses of the RSVP participants also revealed performance profiles congruent with those demonstrated by the aphasic patients; moreover, college students with small working memory spans appeared to be less accurate in comprehending more complex syntactic constructions, particularly in the RSVP condition. (This last point is heatedly debated, see Caplan & Waters, 1999; Just & Carpenter, 1992; Just et al., 1996; Miyake et al., 1999; Waters & Caplan, 1996.)

Despite the similarities between Miyake et al.'s (1994) stressed normal participants and Caplan and Hildebrandt's (1988) aphasic patients in terms of comprehension deficits, it is not clear how clean a parallel can be drawn between performance on a RSVP task (in which individual words are flashed quickly upon a screen) and the more "naturalistic" task of listening to spoken sentences (the usual task presented to aphasic patients). Many investigators report qualitative modality- and task-dependent differences in language processing (reviewed in Federmeier & Kutas, 2001). To determine whether such syntactic deficits could be induced with a more ecologically valid paradigm, Dick, Gernsbacher, and Robertson (2000) compared normal participants' auditory comprehension of similar sentence materials under one of two stress conditions. The first stressor was an auditory version of the RSVP technique, in which sentences were reduced to 66% of their original length; the second "dual-stress" condition was simply a superposition of a low-pass filter over the speeded sentences. Results indicated that presentation modality and task do make a difference: When the speeded condition was compared with normal listening, there was only a small decrease in overall accuracy, and no interaction with sentence complexity—a result in stark contrast to those reported by Miyake et al. However, the results of the dual-stress condition were much more in keeping with those of Miyake et al. Here, sentence difficulty interacted strongly with stress condition, in which comprehension of simple sentences was relatively preserved under stress, whereas performance on more complex sentence types fell close to or at floor levels.

While the results of Miyake et al. (1994) and Dick et al. (2000) generally support the notion that syntactic deficits can arise in normal participants under certain conditions, there are several facets of both studies that make more definitive conclusions difficult to draw. First, as both Caplan and Waters (1999) and Miyake et al. pointed out, these sentence materials (their own stimuli) have serious limitations, in that the sentence types are not balanced for propositional load or sentence length. Because of these confounds, the general sentence processing difficulty metric observed here could be a product of syntactic complexity, logical complexity, length, or a combination thereof. Results of several studies by Caplan, Waters, and their colleagues (Caplan & Waters, 1996; Waters & Caplan, 1997; Waters, Rochon, & Caplan, 1998) suggest that some patient populations (Alzheimer's and temporal lobectomy) are selectively impaired in comprehending sentences with high propositional load, but are not impaired relative to age-matched control participants in comprehending complex syntactic structures (cf. Almor, Kempler, MacDonald, Andersen, & Tyler, 1999; Bates, Harris, Marchman, & Wulfeck, 1995; Kempler, Almor, Tyler, Andersen, & MacDonald, 1998). In their view, these results are not compatible with a model in which syntactic deficits result from a loss in general working memory resources. Moreover, Caplan and Waters have also tested the syntactic compre-

[4] Interestingly, there was no clear effect on comprehension of the number of digits that participants had to recognize; in other words, maintenance of six digits did not appear to have any more deleterious effects on the use of agreement cues than did maintenance of three digits, in contrast with grammaticality judgments studied by Blackwell and Bates (1995).

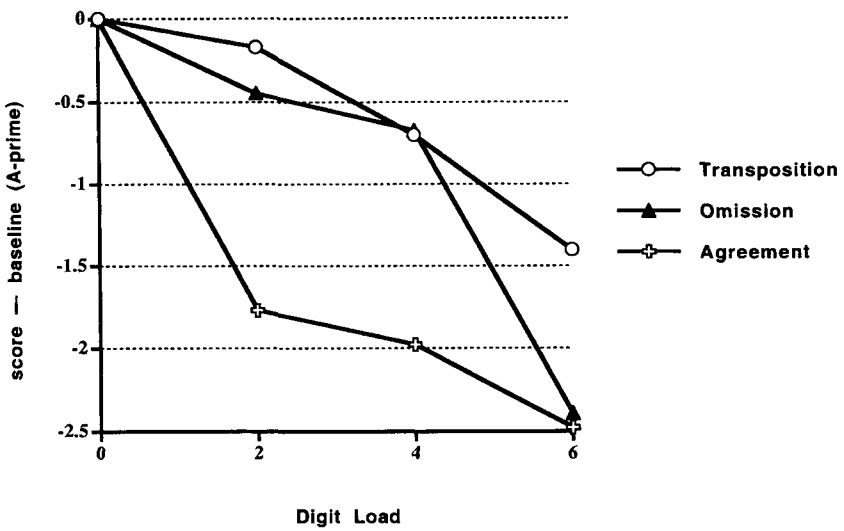

Figure 3. Effect of digit load on detection of transposition, omission, and agreement errors, as expressed in A-prime scores. From "Inducing Agrammatic Profiles in Normals: Evidence for the Selective Vulnerability of Morphology Under Cognitive Resource Limitation," by A. Blackwell and E. Bates, 1995, *Journal of Cognitive Neuroscience, 7*, p. 237. Copyright 1995 by The MIT Press. Adapted with permission.

hension of both Alzheimer's patients and college students under a concurrent digit load task, a task thought to reduce the amount of working memory available for processing. Here again, they did not find the significant interaction of digit load, working memory span, and syntactic complexity that a general working memory account of syntactic deficits might have predicted (but see response in Miyake et al., 1999). However, these results are not entirely consonant with those of other investigators (Blackwell & Bates, 1995; Strube, 1996; Vos, Gunter, Schriefers, & Friederici, 2001) who have found a digit load to impair morphosyntactic processing in both English and German.

Second, the number of exemplars per sentence type in both studies is small (eight), possibly leading to lack of reliability and stability in the assessments of individuals' syntactic comprehension (see comments by Caplan 1995, 2000, in this regard). Indeed, with this number of trials, the 95% confidence boundaries for chance performance by a single individual span almost the entire response range (~12–88%).

Third, the response task used by both groups may itself confound results, in that accuracy is assessed through a yes/no comprehension question, always in the active voice. Not only might the active sentence interfere with comprehension through a type of syntactic priming (Bock, 1986), but, in the view of some investigators, the retrospective nature of comprehension questions may tap into different processes than those used for on-line or immediate syntactic processing (Marslen-Wilson & Tyler, 1998).

Finally, any inferences about the relationship of normal participants' performance and that of aphasic patients are complicated by the fact that no aphasic patients have been tested on exactly such tasks. Although a comparison of results across different tasks is not uncommon and is often extremely useful, any claims about the similarities of stressed normal participants and aphasic patients are made less secure by this fact, in that seemingly minute task-related demands could have significantly differential effects on the two groups. In addition, the limited range of stress conditions makes it difficult to ascertain whether the effects in normal participants were specific only to a particular stressor type.

This last point brings up a more fundamental question; namely, the reason that such stressors should have an effect on language in general and that they should have an effect on morphosyntactic processing in particular. The first part of this question has been addressed in some detail in speech and hearing research, in which there has been continuing interest in simulating or evaluating the effects of age-related hearing loss, low-level perceptual deficits, and cognitive slowdown. Several investigators in this field have suggested that such syndromes, and simulations thereof, can usefully be classed as *peripheral* and *central* perturbations of the processing system.

A peripheral (sometimes referred to as *exogenous*) perturbation is one acting at the level of sensory encoding, such as a noise mask or a low-pass filter. These are believed to simulate the effects of aging- or brain-damage-related hearing loss and/or deficits in complex spectral resolution (Gordon-Salant & Fitzgibbons, 1999; Phillips, Gordon-Salant, Fitzgibbons, & Yeni-Komshian, 2000). Such peripheral effects reduce the amount of spectral information that is perceived, and thereby interfere with the bottom-up encoding of speech contrasts. The effects of the information loss in the speech signal may percolate through the processing system, because less incoming information may result in reduced activation of the lexical items and discourse schemas that map on to these acoustic cues.

By contrast, central or endogenous perturbations include compressed speech, digit load, or general cognitive slowdown during aging or after brain damage (Gordon-Salant & Fitzgibbons, 1993, 1995a, b, 1997; Salthouse, 1996). These stressors do not have as great an effect on the intelligibility of the acoustic input. Rather, they impose a limit on the amount of processing that can be carried out on a particular chunk of information; when listening to sen-

tences spoken at twice their normal speed, the amount of time each phoneme/word/sentence can be processed on-line is halved. Likewise, a slower processing system cannot do as much work on each phoneme/word/sentence as could a normal system in which both systems receive a speech signal at the same rate. Hence, language comprehension under a central perturbation may suffer from a superficiality of processing.

In previous studies, peripheral and central perturbations have also been shown to affect different aspects of lexical processing. For example, Utman and Bates (1998) have shown that reducing the amount of information provided by spectral cues in a sentence will also reduce *facilitation* of a congruent target word; in contrast, reducing the amount of processing time available to the listener to encode the linguistic message reduces or eliminates the *inhibitory* effects of context on incongruent targets.[5] Thus, it appears that peripheral degradation may interfere with the activation–enhancement of congruent targets, whereas central degradation may interfere with the inhibition–suppression of incongruent targets. If both types of distortion are combined, the language-processing system is no longer able to make use of semantic information from the sentence context to enhance compatible entries or to suppress incompatible entries, resulting in a superadditive reduction in contextual priming. A similar effect may emerge in grammatical processing: If we reduce effectiveness in one aspect of language processing (e.g., enhancement), we might see fairly limited, but reliable, effects on sentence processing. However, if we reduce effectiveness in two or more aspects, we may see dramatic effects on overall processing efficacy (see also Gordon-Salant & Fitzgibbons, 1995b).

As noted previously, connectionist or distributive accounts predict that low-level deficits (induced naturally or through simulation) may have consequences for high-level language processing, such as syntax, and that the character of the resulting syntactic deficits should be discernible in part from the *frequency* and *regularity* of syntactic structures in the linguistic environment. Data from a new study by Dick and Elman (2001) of one oral and two written grammatically parsed electronic corpora (*Switchboard, Brown,* and *Wall Street Journal;* Godfrey, Holliman, & McDaniel, 1992; Marcus, Santorini, & Marcinkiewiz, 1993) allow us to make concrete predictions about processing of the sentences comprising the "core data" of agrammatism (actives, subject clefts, object clefts, and passives). These predictions differ in one important aspect from those of the trace-deletion hypothesis of Grodzinsky (2000). As pointed out by Grodzinsky and others (Ferreira & Stacey, 2000; Grodzinsky, 2000), relative frequency alone does not account for the kinds of deficits seen in aphasic patients' sentence processing. For instance, when comparing the frequency of actives (SVO order) and passives (OVS order), Dick and Elman found that the difference in active–passive frequency is less than one would have expected given previous estimates (summarized in St. John & Gernsbacher, 1998; see also Roland & Jurafsky, 1998), with ratios ranging from 1:2 to 1:9 across different corpora (with passives being less frequent in spoken than written corpora; see Dick & Elman, 2001, for further comments on this issue). Moreover, the frequency of both subject and object cleft sentences is vanishingly small, with both types represented in less than 0.05% of sentences; recall that agrammatic performance on subject clefts is high, whereas comprehension of object clefts is poor.

However, as we noted in the introduction, subject clefts share a word order (SV for intransitives; SVO for transitives) that is much more frequent than the OSV order of object clefts; the ratios here range from 1:55 to 1:63. The ratio of the word order used in passives (OVS) to SV falls in the middle and varies more across corpora, in which the ratio in the written corpus is ~ 1:5, and in the spoken corpus ~ 1:29. So what might be the impact of these relative frequencies in processing terms? MacDonald and Christiansen (in press) have shown in behavioral and computational language acquisition paradigms that structures that are themselves infrequent (subject clefts) can "piggyback" on high-frequency structures (actives) sharing the same word order.[6] St. John and Gernsbacher (1998) found that processing of frequently encountered sentences remained relatively impervious to noise injected into a neural network, whereas infrequently encountered sentences became more difficult to process. These results are compatible with a connectionist framework for sentence processing, but it appears that the driving force is not simply frequency, but regularity (i.e., frequency of one pattern vs. another).

The Present Study

To address all the preceding methodological and theoretical questions, we designed a single complex study (Experiments 1A and 1B) that directly compares aphasic patients' syntactic comprehension to that of normal patients working under a range of stress conditions. We focus on exactly those syntactic structures in English that permit assessment of syntactic complexity without confounds of propositional complexity or length, namely, the "core data of agrammatism" referred to previously. All four sentence types contain the same number of nouns (two), verbs (one), and propositions (one) and are controlled for number of words (from six to eight, with subject and object clefts matching exactly). (These represent a balanced subset of the sentence types Grodzinsky, 2000, listed as evidence for the trace deletion hypothesis, pp. 5–7.) The smaller number of sentence types allowed us to increase the number of exemplars per type to 24 without overly taxing the normal, older, or aphasic participants that we compare directly using the same on-line task of sentence comprehension. Our repertoire of stress conditions includes several central (temporal compression and digit load) and peripheral (low pass filter and noise mask) degradations, as well as combinations thereof.

Given the results of previous aphasia simulation experiments and the distributional analyses of Dick and Elman (2001) and others, one should predict the following outcomes of Experiments 1A and 1B. Experiment 1A is conducted with aphasic patients and control participants under normal processing conditions. Two predictions are offered. First, all aphasic patients, regardless of lesion

[5] Here, peripheral perturbations were accomplished through low-pass filter, central through compressed speech. Targets were unaltered.

[6] It is worth pointing out that the subject cleft is only an active sentence with three words added in, with no changes in basic ordering: "*It's the* dog *that* is hitting the cat."

site or classification,[7] should exhibit a pattern of syntactic processing that follows frequency of overall word order, in which actives = subject clefts > passives ≥ object clefts. Second, the difference in the *selective deficit* in passive and especially object cleft comprehension should increase with overall severity of aphasic syndrome. Analogous predictions are offered for Experiment 1B, which compares performance by college students under different stress conditions. We predict that the same profile of performance observed in aphasic patients under normal processing conditions will be in normal populations under stress, with increasing stress levels leading to increasing selective grammatical deficits.

Method

Experiment 1A: Aphasic Patients and Control Participants

Participants. A total of 56 aphasic patients from Veterans Affairs Medical Centers, San Diego or Martinez, took part in the study, as did 15 older control participants from surrounding communities (see Table 1 for patient information). Both aphasic patients and older control participants were paid for taking part. All aphasic patients were classified by using the Western Aphasia Battery (WAB; Kertesz, 1982). A highly trained speech pathologist administered the WAB; clinical and research staff at both institutions carried out classification of patients by WAB score. We made one alteration in the WAB classification criteria because we became concerned that the classification of Broca's aphasia was not entirely in keeping with that of other neurological batteries. Specifically, it appeared to us that the cutoff for Broca's aphasia on the fluency subscale was too low, judging from the description provided for each level of fluency. Therefore, we reclassified our aphasic patients so that the fluency score of Broca's aphasia could range from 0–6, rather than from 0–4. This reclassification resulted in a change of classification for only 2 patients, and did not appreciably affect trends in the data. Of this patient sample, 30 participants were classified as anomic, 12 as Broca's aphasics, 10 as conduction aphasics, 3 as Wernicke's aphasics, and 1 as a transcortical motor aphasic.

All aphasic patients were screened for etiology of the neurological insult: Those patients with aphasia induced by head trauma, multiple infarcts, or metastatic tumors were excluded from the study. Aphasic patients and control participants were screened for hearing impairment with a standard questionnaire, audiometer, or both. Older control participants were screened for cognitive deterioration and dementia using the Mini-Mental Status Exam (Folstein, Folstein, & McHugh, 1975). All participants were right-handed native English speakers, and all were treated in accordance with the *Ethical Principles of Psychologists and Code of Conduct* (American Psychological Association [APA], 1992).

Design and materials. In this experiment, we used a 1 (sentence type) × 1 (patient group) design. Sentence type, with four levels—active, subject cleft, object cleft, and passive—was the within-subject variable; patient group, with five levels—anomic patients, Broca's aphasics, conduction aphasics, Wernicke's aphasics, and elderly control participants—was the between-subjects variable. We collected both accuracy and reaction time data; because of the focus of the current paper, and in the interests of economy, we will report only accuracy data. However, the results of the reaction time data tend to parallel those of accuracy, in which lower accuracy scores co-occur with slower reaction times (e.g., we did not observe any speed–accuracy trade-offs). In addition, we collapsed over a second within-subject variable, presence of noun–verb agreement cue. As we expected, given results from earlier studies in English, this manipulation had little or no effect on comprehension accuracy or reaction time and did not significantly interact with sentence type.

Experimental materials consisted of both visual and auditory stimuli. Visual stimuli were 3 in. × 2 in. digitized black-and-white line drawings of familiar animals culled from several picture databases (Abbate & LaChapelle, 1984a, b; Snodgrass & Vanderwart, 1980). Displayed on a VGA color monitor, each drawing was embedded in a solid gray rectangle over a white background; drawings were presented in pairs determined by sentence content and projected to the left and right sides of the monitor (see Figure 4 for example drawings as presented on monitor).

Sentence stimuli consisted of 96 sentences that were generated by first randomly assigning 2 animate nouns (from a pool of 12) to 1 transitive verb (from a pool of 16). All 12 nouns referred to familiar animals, and all could be assigned to either agent or patient roles. All 16 verbs were semantically similar, in that they expressed a "bad action," such as killing or hurting. Twenty-four NV pairs were then randomly assigned to each of the four syntactic structures: active ("The dog is biting the cow"), subject cleft ("It's the dog that is biting the cow"), object cleft ("It's the cow that the dog is biting"), and passive ("The cow is bitten by the dog"). The present progressive form of the verb was used for all 96 sentences to retain continuity with related studies.

Sentence stimuli were digitally recorded in a sound-insulated chamber by an experienced female speaker and were normalized for speed, length (within sentence type), amplitude, and intelligibility. Recordings were then converted to SoundEdit 16 files, with a 22.255 kHz sampling rate and an eight-bit quantization.

Equipment. PsyScope software (Versions 1.0.1 and 1.0.2) was used to deliver stimuli and collect data (Cohen, MacWhinney, Flatt, & Provost, 1993). Software was run on Macintosh Performa 6214 computers, connected to a VGA color monitor and Apple external speakers (AppleDesign Powered, with tuned port bass reflex speakers). A PsyScope button box was used for response and experimental timing. (See Figure 4 for drawing of experimental setup.)

Procedure. Participants sat in a small room in front of a color monitor, speakers, and a PsyScope button box. Experimenters read instructions to the participants before baseline, practice, and experimental blocks. The baseline measure, which provided data on participants' response rate to simple visual stimuli, consisted of 30 presentations of a line-drawn face to either side of the monitor (following a warning beep). Participants indicated where the face appeared on the screen by using their right index finger to press the left or right button on the button box as quickly as possible.

After completing the baseline task, a practice block of 8 trials was run, followed by two experimental blocks of 48 trials each, with a rest period between the two latter blocks. A trial consisted of the following: After a warning beep, drawings of two animals were projected on the left and right sides of the monitor over a gray background. The nouns referring to the animals were heard in succession (to unambiguously identify the drawings), followed by presentation of a sentence involving both animals. Participants were instructed to use their right index finger to press the button corresponding to the picture of the animal doing the bad action; the picture chosen by the participant was briefly highlighted before the screen was reset for the next trial. Each trial was cued up by the experimenter, who observed the participants' performance and demeanor to assure that they were remaining attentive and alert.

Order of visual and auditory stimuli presentation was fully randomized for each participant, as was presentation of trials. Accuracy feedback was not provided. It was emphasized that participants should attempt to respond

[7] We do not suggest that lesions to any region of the brain will have an equal impact on language processing per se; rather, we suggest that the many areas involved in language do not conveniently map on to linguistic divisions. See the Conclusion section for further comments regarding this issue.

Table 1
Biographical, Testing Site, Aphasia Classification, and Neurological Information for Aphasic Patients

Patient	Age at onset	Age at WAB testing	Age at syntax testing	Years of school	Site	Aphasic category by WAB	Neurologic description	Reduced neurologic profile
C.A.	75	75	75	18	M	Anomic	Subcortical	SC
J.H.	53	62	64	17	M	Anomic	Left frontal	F
J.C.	61	68	70	16	M	Anomic	Left frontal	F
C.I.	60	67	71	18	M	Conduction	Left frontal	F
R.S.	53	66	67	10	M	Broca	Left frontal, temporal, parietal	FP
J.D.	58	65	67	20	M	Anomic	Left frontal, subcortical	SC
H.F.	46	49	52	19	M	Anomic	Left subcortical	SC
C.G.	46	47	49	14	M	Conduction	Left temporal, parietal, some frontal	FP
C.W.	67	74	76	18	M	Anomic	Left temporal, parietal, some frontal	FP
W.R.	45	48	51	14	M	Broca	Left frontal, temporal	F
W.A.	64	71	73	15	M	Anomic	Left frontal, subcortical	SC
R.D.	51	58	60	5	M	Anomic	Left frontal	F
K.K.	27	32	34	14	M	Anomic	Left frontal	F
R.A.	57	62	62	10	M	Conduction	Left temporoparietal	P
M.B.	54	63	63	14	M	Broca		
N.J.	62	66	68	14	M	Anomic		
E.S.	71	72	72	14	M	Anomic	Left subcortical	SC
W.T.	48	61	62	14	M	Anomic	Left frontotemporal	FP
E.H.	67	72	74	7	M	Anomic	Left subcortical	SC
R.C.	NA	75	75	15	M	Anomic	Right subcortical	SC
L.L.	70	71	71	16	M	Broca		
L.C.	38	48	48	14+	M	Anomic		
O.A.	51	63	63	8	M	Anomic		
F.Y.	71	72	73	12	M	Conduction		
F.B.	53	55	56	12	M	Anomic	Brainstem	SC
G.F.	57	66	67	14	M	Broca	Left frontal, parietal	FP
B.K.	49	50	50	16	M	Anomic		
A.L.	58	64	64	14	M	Anomic	Left parietal, occipital	P
J.C.	68	68	70	12	M	Wernicke	Left temporal, parietal, possible right parietal	—
M.L.	55	57	57	17	M	Anomic	Cerebellar	SC
E.E.	66	66	67	15	M	Anomic		
G.M.	57	68	70	12	SD	Broca	Left frontal, parietal	FP
J.S.	62	68	69	16+	SD	Anomic	Left temporoparietal, some subcortical	SC
H.P.	67	73	75	9	SD	Anomic	Left frontoparietal	FP
D.R.	74	81	81	12	SD	Broca	Left frontal, temporal, parietal	FP
C.F.	52	54	56	16	SD	Anomic	Left parietal, occipital	P
D.C.	56	58	58	18	SD	Broca	Left cortical, subcortical	SC
T.C.	50	50	51	18	SD	Wernicke	Left frontal, temporal, parietal, possible right frontal	—
T.B.	63	64	65	16	SD	Conduction		
D.B.	64	64	66	11	SD	Broca	Left frontoparietal	FP
B.U.	69	78	80	18	SD	Conduction	Left frontal	F
C.G.	55	60	61	12	SD	Broca	Left subcortical	SC
S.C.	53	68	69	16	SD	Conduction	Left frontal and parietal	FP
L.B.	67	70	70	12	SD	Anomic	Left thalamic, subcortical	SC
M.C.	NA	66	67	15	SD	Anomic	Left frontal, temporal, parietal	FP
M.W.	35	37	38	18	SD	Anomic	Left frontal	F
J.T.	53	55	55	12	SD	Conduction	Left	—
J.L.	73	73	73	16	SD	Broca	Left temporal, occipital, parietal, subcortical	SC
F.T.	55	55	55	14	SD	Wernicke	Left frontoposterior	FP
K.O.	62	63	63	12	SD	Anomic		
A.W.	64	64	64	8	SD	Broca	Left frontal, temporal, parietal, some subcortical	SC
W.B.	35	66	66	12	SD	Anomic		
P.A.	56	57	57	12	SD	Anomic	Left posterior	P
J.H.	51	52	52	12	SD	Conduction	Basal ganglia	SC
L.M.	47	51	51	14	SD	Transmotor		
I.T.	79	79	79	18	SD	Conduction	Left posterior parietal	P

Note. Dashes indicate patient not included in the neurologic profile. WAB = Western Aphasia Battery (Kertesz, 1982); M = Veterans Affairs Northern California Health Care System, Martinez; SC = subcortical; F = frontal; FP = frontoposterior; P = posterior; NA = information not available; SD = Veterans Affairs Medical Center, San Diego, California.

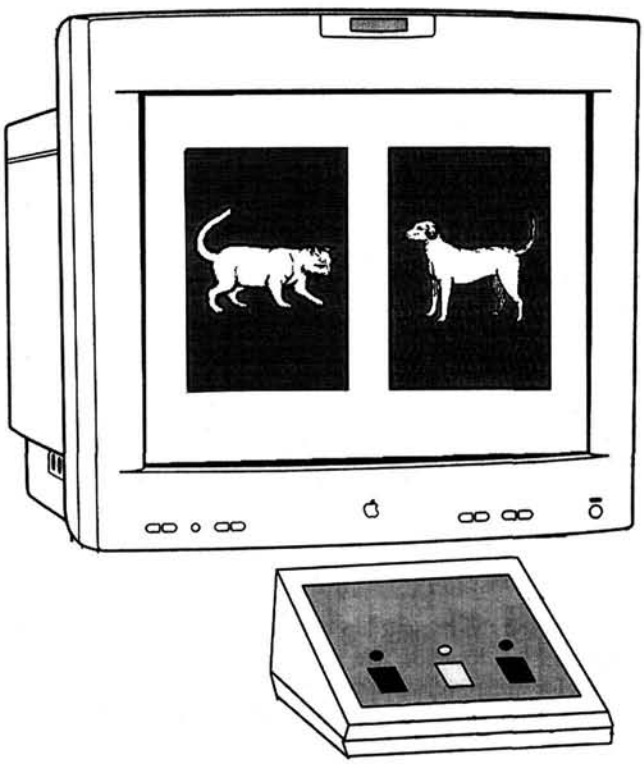

Figure 4. Experimental setup and sample visual stimuli (Experiments 1A and 1B).

as accurately and quickly as possible to the stimuli. At the end of the experimental session, the experimenter thanked and debriefed the participant.

Experiment 1B: College Students Under Normal and Stressful Conditions

Participants. A total of 216 students from the University of California, San Diego, took part in the study. All students received either 1 hr. of course credit or $5 for their participation. Participants were distributed among experimental conditions as follows: 25 students in the normal listening condition, 24 students in the 50% speech rate compression condition, 21 students in the visual six-digit load condition, 22 in the auditory six-digit load condition, 28 students in the 50% noise mask condition, 23 students in the 600 Hz low-pass filter condition, 24 students in the 50% speech rate compression plus visual six-digit load condition, 23 students in the 50% noise mask plus visual six-digit load condition, and 26 students in the 600 Hz low-pass filter plus 50% speech rate compression condition. All participants were right-handed native English speakers with no significant exposure to a language other than English before age 12; all participants had normal hearing and normal or adjusted-to-normal vision as assessed by a standard intake questionnaire. All were treated in accordance with the *Ethical Principles of Psychologists and Code of Conduct* (APA, 1992).

Design and materials. Design and materials differ from the aphasic experiment only in that college students listened to materials under one of the nine different between-subjects conditions. A 1 (sentence type) × 1 (stress condition) design was used, with sentence type as the within-subject variable and stress condition as the between-subjects variable. The latter is composed of nine levels: normal listening, visual six-digit load, auditory six-digit load, 50% speech rate compression, 50% noise mask, 600 Hz low-pass filter, 50% speech rate compression plus visual six-digit load, 50% noise mask plus visual six-digit load, and 600 Hz low-pass filter plus 50% speech rate compression.

Preprogrammed proprietary algorithms on SoundEdit16 were used to impose all temporal and spectral changes on duplicates of the sentence stimuli. The speech rate manipulation, similar to that used by Utman and Bates (1998) and Gordon-Salant and Fitzgibbons (1993, 1995a, b), excised redundant waveforms in the 5–20-ms range; this procedure primarily affected temporal information, thereby leaving spectral properties of the speech signal relatively intact. The low-pass filter manipulation reduced all information above 600 Hz by less than 20 decibels with overall amplitude normalized to prefilter levels; the noise mask was imposed over the sentence stimuli at 50% of maximum amplitude (average signal:noise ratio was −12.18, as measured by the Praat sound package) and was distributed equally throughout the frequency spectrum available in SoundEdit16.

In the visual digit condition, a series of six numbers was rapidly presented digit by digit, with each digit centrally presented on the monitor for 333 ms, followed by a 350-ms interstimulus interval. The first digit sequence was followed by the presentation of the animal pictures and corresponding nouns, sentence, and agent identification as described earlier. After the participant identified the agent, another series of six digits was displayed. Participants were asked to signal whether the latter sequence of digits was the same one as displayed at the beginning of the trial by pushing the appropriate yes/no button (the left and right buttons on the button box, respectively). The auditory digit condition differed from the visual one only in that each number was spoken, rather than presented on the monitor. Timing parameters were the same, in that digits were presented every 683 ms. Experimental procedure for college students was identical to that for aphasic patients, except that trials advanced automatically rather than being experimenter cued.

Results

We report results as follows: for Experiment 1A, results for (a) aphasic patients only and (b) aphasic patients compared with older controls; for Experiment 1B, results for (a) college students under normal and stress conditions and (b) comparison of aphasic patients and college students. Included in our results are standard analyses of variance (ANOVAs), cluster analyses, and nonparametric categorizations by the Grodzinsky (2000) definition of agrammatism. We carried out all ANOVAs with SuperAnova and Statview 5.0 for Macintosh; cluster analyses were performed using SPSS 9.0 for Windows. The p values reported for all within-subject variables are Geisser–Greenhouse corrected (Geisser & Greenhouse, 1958), and all pairwise comparison values are Bonferroni adjusted. In all analyses we used participants as the random variable because items were homogeneous (Clark, 1973).

Experiment 1A: Aphasic Patients Only

Aphasic patients grouped by the Western Aphasia Battery (WAB). Our initial set of analyses investigated possible differences in comprehension profile over aphasic group (as defined by the WAB). The transcortical motor aphasic patient was not included in any group analyses, being a single-case study. This patient performed at high levels on active and subject cleft sentences (96% and 100% accuracy, respectively), somewhat less accurately on passives (90%), and considerably lower on object clefts (70%).

We first compared anomic, Broca's, Wernicke's, and conduction aphasic patients' accuracy over sentence type (see Figure 5). The 1 (patient group) × 1 (sentence type) ANOVA revealed a significant main effect of patient group, $F(3, 51) = 7.915$, $p <$

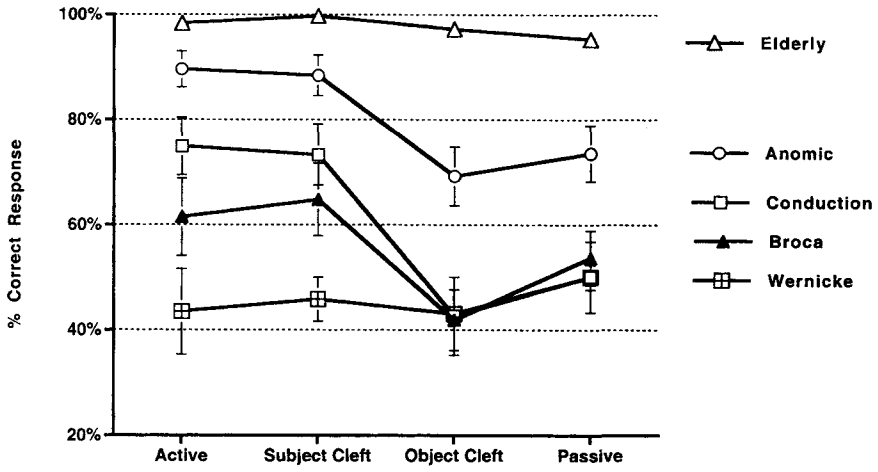

Figure 5. Percentage of correct responses by sentence type for aphasic patients (grouped by the Western Aphasia Battery) and older control participants (Experiment 1A). Error bars represent ±1 *SEM*.

.0002, in which anomic patients were more accurate than the other three patient groups (anomics > Broca's, $p = .0018$, anomics > conduction, $p = .0318$, anomics > Wernicke's, $p = .0222$). There was also a main effect of sentence type, $F(3, 51) = 0.215$, $p < .0014$, with pairwise comparisons showing that both actives and subject clefts were comprehended more accurately than either passives and object clefts (actives > object clefts, $p = .0006$, subject clefts > object clefts, $p = .0006$, actives > passives, $p = .0036$, subject clefts > passives, $p = .0036$). Passives were also comprehended marginally more accurately than were object clefts ($p = .0571$). There was no significant interaction of sentence type with patient group, $F(9, 153) = 1.107$, $p = .3607$; however, the trend was for anomic, Broca's, and conduction aphasic patients to adhere to the agrammatic profile (in which active and subject cleft was greater than passive and object cleft), whereas Wernicke's patients performed almost at chance levels on all sentence types. The low number of Wernicke's aphasics in our sample ($n = 3$) precludes further speculation about this trend.

We then performed a series of pairwise comparisons between each sentence type for each aphasia subgroup. Anomic patients comprehended both actives and subject clefts better than either passives or object clefts (actives > object clefts, $p = .0072$; actives > passives, $p = .04$; subject clefts > object clefts, $p = .012$; subject clefts > passives, $p = .06$). Accuracy on actives versus subject clefts did not differ significantly, nor did comprehension accuracy of passives versus object clefts. Results for conduction aphasics were identical, in which actives = subject clefts > passives = object clefts (actives > object clefts, $p = .0108$; actives > passives, $p = .0468$; subject clefts > object clefts, $p = .012$, subject clefts > passives, $p = .06$). Our group of Broca's aphasic patients comprehended object clefts marginally less accurately than either actives ($p = .1134$) or subject clefts ($p = .06$); passives fell in between these two groupings and did not significantly differ from them. As was observed previously, the average comprehension accuracy of the 3 Wernicke's aphasic patients did not differ across any sentence type comparison.

Aphasic patients grouped by lesion site. Because aphasia categorization by the WAB is based purely on behavioral criteria, we also examined the relationship between lesion site and syntactic comprehension accuracy. We included in these analyses the 41 patients for whom we had detailed neurological data (either CT or MRI structural scans). All patients had lesions in the left hemisphere only, but for 1 patient who had damage to the right subcortical areas only (we excluded 2 participants with bilateral cortical lesions from these analyses). To maximize power and retain continuity with previous studies (Bates et al., 1987a), we grouped patients into one of four categories: (a) lesion confined to frontal regions ($n = 8$); (b) lesion extending frontoposteriorly ($n = 12$); (c) lesion confined to posterior regions ($n = 5$); and (d) subcortical involvement with either frontal or posterior lesions ($n = 16$).

A 1 (sentence type) × 1 (lesion site) ANOVA did not show a significant effect of lesion site on comprehension, $F(3, 37) = 2.203$, $p = .1262$, or a Lesion Site × Sentence Type interaction, $F(9, 111) = 0.499$, $p = .6750$, with sentence type the within-subject variable and lesion type the between-subjects variable (see Figure 6). There was, however, a main effect of sentence type, $F(3, 37) = 9.958$, $p = .005$, reflecting higher accuracy on actives and subject clefts than on passives and object clefts (actives > object clefts, $p = .0012$; actives > passives, $p = .0156$; subject clefts > object clefts, $p = .0024$; subject clefts > passives, $p = .0258$).

Because of the large amount of interindividual variance and relatively small group size, it would be unwise to draw strong conclusions from a failure to reject the null hypothesis. However, no clear group trends appear to emerge in the data, with the possible exception of a somewhat higher mean score for patients with strictly posterior lesions compared with other groups. Again, this trend is overshadowed by interindividual variance and should be regarded warily. The more important observation here is that sentence comprehension profiles did not appear to vary in any systematic way with lesion site.

Cluster analyses of aphasic patients. Although there was no Sentence Type × Patient Group interaction or a Sentence Type × Lesion Site interaction (suggesting that all aphasic groups tended to adhere to a common profile), we felt that "accepting the null

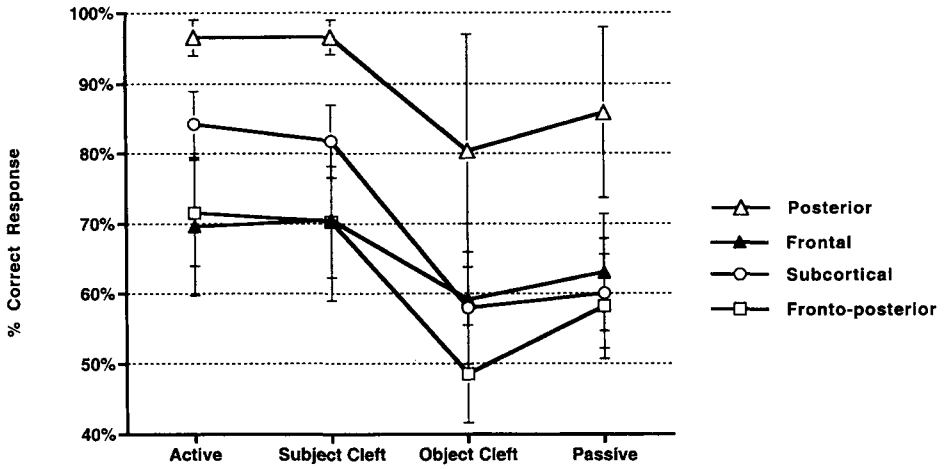

Figure 6. Percentage of correct responses by sentence type for aphasic patients (grouped by lesion site) (Experiment 1A). Error bars represent ±1 *SEM.*

hypothesis" was not a particularly meaningful metric for assessing similarities or differences over groups. To better characterize aphasic groups' profiles of comprehension, we looked at patterns of individual differences by way of *k*-means cluster analyses, a method similar to that used by Caplan and Hildebrandt (1988) and Miyake et al. (1994) to assess aphasics patients' performance. This descriptive statistical technique "looks for" groups of participants that display similar profiles of performance over the levels of the variable in question and clusters them together around a prototype, or centroid. We prespecified six cluster centers, as preliminary clustering indicated that inclusion of more centers was counterproductive, because some clusters would consist of only 1 or 2 participants. We have informally characterized these clusters—shown in Figure 7—as (a) *near-normal performance,* with accuracy in all sentence types above 90%; (b) *high-agrammatic performance,* with passives and object clefts comprehended much less accurately than actives and subject clefts, but still above chance levels; (c) *low-agrammatic performance,* with passives and object clefts comprehended around chance levels, and actives and subject clefts comprehended above chance, but not at normal levels; (d) *random performance,* with accuracy in all sentence types around 50%; (e) *first-noun strategy,* in which the first noun mentioned is overwhelmingly chosen as agent; and (f) *reverse,* in which actives and subject clefts are almost always interpreted incorrectly, and passives and object clefts are interpreted at chance. (As cluster center values were similar over all our cluster analyses, we collapsed clusters for the sake of economy. Hence, cluster values represented in Figure 7 are the averages for the cluster centers in the three cluster analyses reported here.) We tested the reliability of cluster membership by means of a split-half comparison; here, clustering was performed on sentence type means from only the first half of the trials in the experiment (in which sentences were randomly assigned by PsyScope to trial number). Using the original cluster centers as initial seeds, we found that 51 of 55 participants included in the group analyses (~93%) remained in their original cluster; 3 participants switched between the low-

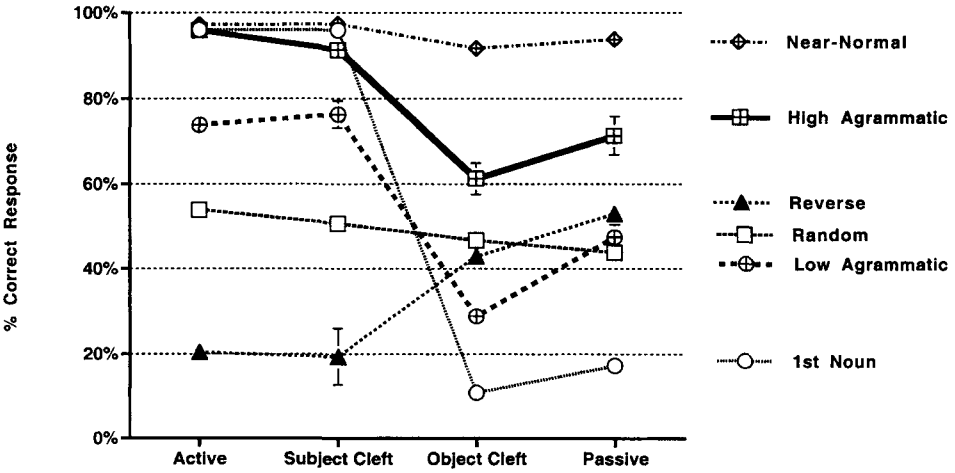

Figure 7. Percentage of correct responses by sentence type for cluster centroids, averaged over three cluster analyses (Experiment 1A). Error bars represent ±1 *SEM.*

A

Aphasic Group	1st Noun	Reverse	Random	Lo agrammatic	Hi Agrammatic	Near-Normal
Anomic	3	1	2	3	4	17
Broca	0	2	3	5	1	1
Conduction	1	0	2	5	1	1
Wernicke	0	1	2	0	0	0

B

Lesion Site	1st Noun	Reverse	Random	Lo agrammatic	Hi Agrammatic	Near-Normal
Frontal	0	1	2	2	0	3
Fronto-posterior	1	1	4	3	0	3
Posterior only	0	0	0	1	0	4
Subcortical	2	0	5	1	3	5

Figure 8. A: Number of Western Aphasia Battery–grouped aphasic patients per cluster (Experiment 1A). B: Number of lesion-site–grouped aphasic patients per cluster (Experiment 1A). Lo = low; Hi = high.

agrammatic and random clusters, and 1 participant switched from low-agrammatic to reverse.

Analyses revealed no clear differences in cluster membership over WAB-based aphasic groups with the exception of the 3 Wernicke's aphasic patients, all of whom performed at a low level of accuracy (see Figure 8A for cluster membership). Dissimilarities among anomic, Broca's, and conduction patients were essentially tied to severity, in which more anomics than Broca's patients were included in the cluster in which accuracy approached normal sentence comprehension, whereas more Broca's were included in the cluster in which comprehension was at chance levels for all four sentence types.

Cluster analyses also uncovered no reliable relationships between lesion site and syntactic comprehension. Not only was there little correlation between cluster and lesion site ($r^2 = .0465, p = .4391$) but all lesion groups generally contributed members to each cluster, with the most notable exception being the fairly large number of frontal and frontal-posterior patients in a random performance cluster, with no strictly posterior patients contributing members to this group (see Figure 8B). This small group with posterior-only damage ($n = 5$) also contributed only one member to the most agrammatic clusters, which contained members from all other lesion groups (serving as weak evidence in favor of a localization hypothesis). We should also note that there was again a trend for patients with exclusively posterior lesions to fall into the cluster of near-normal performance; however, patients with frontal, frontal-posterior, and mixed subcortical lesions contributed to the bulk of this cluster.

Aphasia classification, lesion site, and Grodzinsky's (2000) agrammatic profile. We also examined the relationship between *aphasia* classification, lesion site, and agrammatism as defined by Grodzinsky (2000), requiring comprehension of active and subject cleft sentences at above-chance levels, with passive and object cleft performance at, but not below, chance. We defined *chance performance* as a score falling within the 95% confidence interval predicted for a binomial distribution for 24 trials (as there were 24 exemplars per sentence type). Hence, a score falling between 29% and 71% correct would be classed as chance performance, with all scores above 71% correct classed as above chance, accordingly. (This binomial classification applies only to an individual subject's performance and cannot be used for averages over subjects.)

When we applied this classification scheme to all our aphasic patients, we found that 1 anomic, 3 Broca's, and 3 conduction aphasics fell into this strictly defined agrammatic category, with all others again falling somewhere on a continuum from random, through quasi-agrammatic to near-normal or normal performance. In other words, there was no evidence supporting the contention that Broca's aphasic patients alone exhibit a "true" agrammatic profile.

In addition, when we looked at the profiles of the 41 aphasic patients for whom we had detailed neurological reports, only 4 were classified as agrammatics by the Grodzinsky (2000) criterion (1 frontal, 2 frontoposterior, and 1 strictly subcortical), with all other patients falling somewhere on the continuum between random to quasi-normal performance (with an additional 4 patients consistently choosing the first noun as agent). Again, there was no correlation between lesion site and agrammatic performance. In short, we found no evidence that Grodzinsky's definition of agrammatism is associated with any particular lesion site or aphasic syndrome.

Experiment 1A: Aphasic Patients Compared With Older Controls

The concept of a selective deficit in grammatical comprehension is tightly yoked to the relative difference in comprehension profiles between normal control participants and agrammatic patients, in which both normal and (idealized) agrammatic participants perform well on canonical sentence types (actives and subject clefts), whereas only agrammatic participants falter on the noncanonical types (passives and object clefts). This difference should be reflected as a statistical interaction of sentence type and subject group. Hence, to ascertain whether any or all of our aphasic groups met this criterion for agrammatism, we compared all aphasic groups with older control participants in a 1 (sentence type) × 1 (patient group) omnibus ANOVA, again with patient group and sentence type as between- and within-subjects variables, respectively (see Figure 5).

There was a significant main effect of response accuracy, with older control participants more accurate overall than any aphasic group, $F(4, 65) = 16.064$, $p < .0001$. Pairwise comparisons showed that the older participants were overall more accurate than anomic patients ($p = .006$), Broca's ($p = .0004$), conduction ($p = .0004$), or Wernicke's patients ($p = .0004$). There was also a significant main effect of sentence type, $F(3, 65) = 11.042$, $p = .0004$, reflecting the expected difference between easy structures (actives and subject clefts) and hard structures (passives and object clefts): actives > object clefts, $p = .0006$; actives > passives, $p = .003$; subject clefts > object clefts, $p = .0006$; and subject clefts > passives, $p = .0024$). In contrast with our earlier analyses of aphasic patients only, here there was a significant interaction between sentence type and group, $F(12, 195) = 2.304$, $p = .0464$. As Figure 5 shows, the interaction is due to near-ceiling performance by older control participants, compared with the marked disparity between hard and easy structures for all the aphasic groups but Wernicke's aphasics (who were near chance on all structures).

Given this general interaction, we followed up with separate Group × Sentence Type ANOVAs comparing each aphasic subgroup with older control participants; each ANOVA confirmed that, compared with older participants, anomic, $F(3, 129) = 4.109$, $p < .0386$; Broca's, $F(3, 75) = 4.211$, $p < .0283$; and conduction aphasic, $F(3, 69) = 15.643$, $p < .0001$, patients were all differentially impaired across sentence types, with more errors on the difficult structures for aphasic patients compared with older control participants. (The 3 Wernicke's aphasic patients all performed at near-chance levels, thereby failing to interact with the older profile.) These positive results lend further weight to the assertion that the characteristic agrammatic profile occurs across a number of aphasia classifications.

Interestingly, when the older participants were analyzed alone, there was a marginally significant effect of sentence type, such that passives were comprehended less accurately than other sentence types, $F(3, 14) = 3.221$, $p = .061$. There was also a small but marginally significant interaction of subject group with sentence type when older participants were compared with normal college students, $F(3, 114) = 2.87$, $p = .0528$. This slight decrement in passive performance in older control participants is in keeping with previous findings of subtle changes in language comprehension over the lifespan (Devescovi et al., 1997); however, it is a small decrement indeed, and not too much theoretical weight should be attached to it.

Experiment 1B: College Students Under Normal and Stress Conditions

As noted earlier, a diagnosis of agrammatism relies in part on the demonstration of a statistical interaction between sentence type and patient group. As a first step toward establishing that agrammatism can occur in stressed normal participants, we performed the logical equivalent of the comparison between older control participants and aphasic groups by performing an ANOVA over students working under normal or stressor conditions.

The omnibus 1 (sentence type) × 1 (stress condition) ANOVA revealed a main effect of sentence type, $F(3, 206) = 51.487$, $p < .0001$; stress condition, $F(8, 206) = 29.362$, $p < .0001$; and Sentence Type × Stress Condition, $F(24, 618) = 8.882$, $p < .0001$, with sentence type and stress condition as the within-subject variable and the between-subjects variable, respectively. As was found for the aphasic patients, pairwise comparisons showed that passive and object cleft sentences were comprehended less accurately overall than active and subject cleft sentences (all comparisons at $p = .0006$); object clefts were also comprehended less accurately than passives ($p = .0018$). In addition, students under almost all stress conditions performed less accurately overall than did students under normal conditions.

To better understand the interaction of sentence type with stress conditions, we performed several separate ANOVAs, each comparing a single-stress condition to normal listening (Figure 9A). Students under the 50% speech compression condition were significantly less accurate overall than students under normal listening, $F(1, 47) = 6.284$, $p = .016$; a significant interaction between Sentence Type × Condition suggested that comprehension of passives and object clefts (relative to actives and object clefts) in the compressed speech was more impaired than in normal listening conditions, $F(3, 141) = 3.686$, $p = .0313$. Bonferroni- and Greenhouse–Geisser-corrected pairwise contrasts over sentence types on the compression condition alone showed that active and subject cleft sentences were comprehended equally well, with accuracy on object clefts significantly lower than either actives or subject clefts ($p = .0228$ and $p = .0192$, respectively). Although passives differed numerically from actives and subject clefts in the predicted direction, the contrasts did not reach significance; a trend for object clefts to be comprehended less accurately than passives was also marginally significant ($p = .0924$).

Similar results were obtained under the 50% noise mask condition, with reliable differences in overall accuracy, $F(1, 51) = 50.791$, $p < .0001$, and relative comprehension of sentence types, $F(3, 153) = 7.932$, $p = .0002$. Contrasts comparing active and subject clefts to object clefts were again significant ($p = .0006$ and $p = .0018$, respectively), as was the contrast between active and passive sentences ($p = .0396$) and between object clefts and passives ($p = .0039$). Likewise, in the low-pass filter condition, there was a reliable overall difference in accuracy, $F(1, 46) = 5.213$, $p = .0271$, and again an interaction of sentence type with listening condition, $F(3, 138) = 3.311$, $p = .0413$. Pairwise contrasts showed that actives and subject clefts were comprehended more accurately than both passives ($p = .0306$ and $p =$

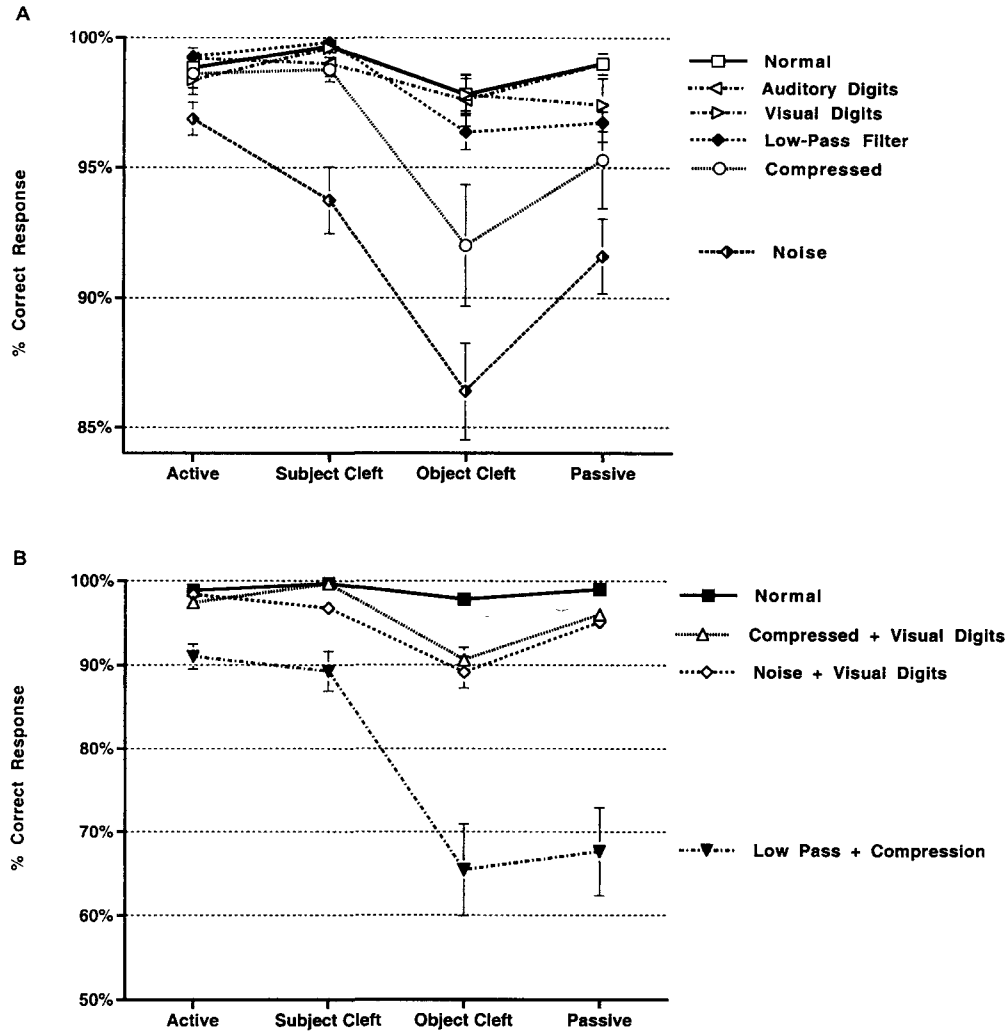

Figure 9. A: Percentage of correct responses by sentence type for students under single-stress conditions (Experiment 1B). B: Percentage of correct responses by sentence type for students under dual-stress conditions (Experiment 1B). Error bars represent ±1 *SEM*.

.006) and object clefts (p = .0102 and p = .0018), with no difference between the latter two types.

Interestingly, in direct contrast with the various perceptual stressors (noise, compression, and low-pass filtering), the comparison of visual digit load with normal listening showed neither a significant difference in overall accuracy, $F(1, 44) = 1.655$, p = .2037, nor an interaction of condition with sentence type, $F(3, 132) = 0.851$, p = .4385. The lack of a digit effect was not due to participants "ignoring" the digit task because when we removed participants who scored poorly on the digit task, results did not change. Results for the auditory digit load were similar, in that comparisons with normal participants showed no significant difference in overall accuracy, $F(1, 44) = 0.113$, p = .74, and no Sentence Type × Condition, $F(3, 132) = 0.315$, p = .73. A direct comparison of visual and auditory digit load conditions showed no differences between the two in overall accuracy, $F(1, 40) = 0.794$, p = .38, or a Digit Modality × Sentence Type interaction, $F(3, 120) = 1.182$, p = .316. Accuracy on the digit task itself also did not vary significantly with modality in overall accuracy, $F(1, 40) = 1.004$, p = .3225, and modality did not interact with sentence type, $F(3, 120) = 0.604$, p = .59.

Furthermore, the visual digit load did not impose any additional stress when it was combined with either compressed speech or noise (Figure 9B). An ANOVA comparing the compressed speech and compressed speech plus visual digit conditions showed no main effect, $F(1, 46) = 0.044$, p = .8345, nor a Stress Condition × Sentence Type interaction, $F(3, 138) = 0.614$, p = .5337. Indeed, in the complementary noise/noise plus visual digit comparison, analyses showed that overall accuracy in the noise mask plus visual digit load condition was actually slightly higher than with noise alone, $F(1, 49) = 5.889$, p = .019, although this difference did not interact with sentence type, $F(3, 147) = 0.245$, p = .8117. However, both the noise plus visual digit condition and the compression plus visual digit condition still induced a selective deficit in comprehension of passives and object clefts, as seen from the ANOVA comparison with normal listening: $F(3, 138) = 8.218$,

p = .0005, and $F(3, 141) = 10.284$, p = .0001, respectively. Pairwise contrasts for the compression plus visual digit conditions showed that active and subject cleft sentences were comprehended more accurately than object cleft sentences (p = .0006 and p = .0006), with subject clefts comprehended more accurately than passives (p = .05), and passives comprehended better than object clefts (p = .001). For the noise plus visual digit condition, active and subject cleft sentences were comprehended more accurately than object cleft sentences (p = .0006 and p = .003), as were passives (p = .0024). Mean accuracy for passive sentences was again numerically less than either active or subject cleft sentences in both conditions, but it did not reach our strict level of statistical significance. In other words, when a digit load is added to other stressors, it does not appear to increase processing costs.

There generally appeared to be little interaction between the digit task and the sentence interpretation task in this dual-task paradigm, with one exception: When we combined data from all visual digit conditions (e.g., digits alone, noise plus digits, and compression plus digits), we found that accuracy on object clefts significantly predicted accuracy on digits trials in analyses over all object cleft sentences (such that lower object cleft accuracy would predict lower accuracy for digits remembered over object clefts). However, we did not find this for any of the other sentence types (most notably passives), so the theoretical significance of this correlation is limited.

In short, these particular digit manipulations appear to have little or no effect on processing of these sentence types. The visual digit load also adds nothing to the effects of noise or speech compression, except for (paradoxically) a slight increase in overall accuracy when digits are added to noise. Because the digit task has played an important role in the debate between Caplan and Waters (1999) and Just and Carpenter (1992) and Miyake et al., (1999), we return to these somewhat counterintuitive results for digits in more detail in the final discussion.

In stark contrast to the lack of interaction between digit load and noise or speech compression, the addition of a low-pass filter to speech compression has dramatic and superadditive effects on comprehension, above and beyond those seen for the two stressors alone. Students in this dual-stress condition differ from those in normal listening in overall accuracy, $F(1, 49) = 45.663$, p = .0001, with presentation condition strongly interacting with sentence type, $F(3, 147) = 16.014$, p = .0001, again such that comprehension of passives and object clefts (relative to actives and subject clefts) was significantly more impaired than in normal listening. Pairwise comparisons on the stress condition alone corroborated this interaction, with all of these comparisons at p = .0006 (but note that there was no significant difference between object clefts and passives). Indeed, students in this dual-stress condition appeared to be both qualitatively and quantitatively similar to aphasic patients in their comprehension profile, a hypothesis we address in the following section.

Experiment 1B: Comparisons of Aphasic Patients and College Students Under Stress

ANOVAs comparing aphasic patients and students under low-pass filter plus speech compression. To directly compare students and aphasic patients, we first performed a series of ANOVAs comparing each aphasic group to the students under low-pass filter plus compression. When these students were compared with the 30 anomic patients, there was no main effect of group on overall comprehension accuracy, $F(1, 54) = 0.139$, p = .7110, nor was there a Patient Group × Sentence Type interaction, $F(3, 162) = 0.649$, p = .4887. In other words, the combination of compression and filtering used in this condition resulted in a performance profile that was statistically indistinguishable from anomic aphasic patients. Conduction aphasic patients were more impaired than these college students overall, $F(1, 34) = 9.956$, p = .0005, but the profile of sentence comprehension did not differ significantly, $F(3, 102) = 0.301$, p = .7232. Our sample of Broca's aphasic patients also performed less accurately overall, $F(1, 36) = 17.791$, p = .0002, than did students under low-pass filter plus compression, but again there was no significant Patient Group × Sentence Type interaction, $F(3, 108) = 1.279$, p = .2835. If anything, the Broca's aphasic patients looked less agrammatic than the students under dual-stress conditions, because the difference between sentence types appeared more pronounced in the students than in Broca's. Wernicke's aphasic patients were again generally more impaired across the board than these dual-stress students, $F(1, 27) = 13.541$, p = .001. There was also a marginally significant Sentence Type × Patient Group interaction, $F(3, 81) = 2.389$, p = .1015, such that the 3 Wernicke's aphasic patients were almost at chance for all sentence types, whereas the college students under low-pass filter and speech compression performed closer to the theoretical agrammatic profile.

Cluster analyses of aphasic patients and students under low-pass filter plus compression. As we noted previously, failure to reject the null hypothesis is perhaps not the best method of assessing similarities between groups. Therefore, we again performed cluster analyses on the aphasic groups and students under low-pass filter plus compression together (prespecifying six centers for the reasons previously mentioned). Reliability of cluster membership was again high (89%), as assessed by the split-half technique detailed earlier. Cluster centers were almost identical to those obtained for the aphasics-only clustering (see Figure 10). Almost half of the students under low-pass filter plus compression were assigned to the high- or low-agrammatism clusters, with the remainder in the almost-normal or first-noun clusters. The student cluster distribution was most like the anomic patients, with a slightly larger number of anomics in the chance-performance cluster, and a greater proportion of dual-stress students in the prototypically agrammatic clusters. Compared with the students, Broca's and conduction aphasic patients tended to be assigned more to the chance-performance-across-the-board cluster (this characterized almost half of the WAB-classified Broca's) but otherwise, these two patient groups shared cluster membership with anomic patients and students. As before, the 3 Wernicke's aphasic patients were all assigned to the chance-across-the-board performance cluster.

Students under low-pass filter plus compression, grouped by the Grodzinsky (2000) definition of agrammatism. Finally, we categorized these students using the Grodzinsky (2000) definition of agrammatism (actives/subject clefts above chance; passives/object clefts at chance). We found that 6 of the 26 students fit this definition, with the remaining 19 students distributed again over the random to quasi-normal range, just as we found with the aphasic patients.

Group	1st Noun	Reverse	Random	Lo Agrammatic	Hi Agrammatic	Near Normal
Anomic	3	1	2	3	4	17
Broca	0	1	5	4	1	1
Conduction	1	0	4	3	1	1
Wernicke	0	0	3	0	0	0
LPC	3	0	0	4	7	12

Figure 10. Number of Western Aphasia Battery–grouped aphasic patients and low-pass filter plus compression college students per cluster (Experiments 1A and 1B). Lo = low; Hi = high; LPC = low-pass filter condition.

Summary of results with students under stress. In summary, students processing under most single-stress conditions (compressed speech, noise mask, and low-pass filter) produced profiles that were qualitatively similar to the agrammatic profile we observed in our aphasic groups, in which stressed students tended to be selectively hindered in comprehension of object clefts and passives compared with students under normal listening; most groups were more impaired on object clefts than on passives. Both the digit load manipulations appeared to have no significant effect on performance, either as a single stressor or when combined with other stress conditions. In contrast, the group of students under the dual low-pass filter plus compressed speech condition experienced a superadditive effect of these stressors, such that their performance was indistinguishable both qualitatively and quantitatively from our largest aphasic group (anomics). Both anomic patients and students under low-pass filter plus compression bore the closest resemblance to the theoretical profile of agrammatism, as seen both in cluster analyses and in the Grodzinsky (2000) grouping scheme.

Discussion

The experiments reported here show quite unequivocally that selective deficits in syntactic comprehension can occur in all aphasic groups (classified both behaviorally and by lesion site) and can also come about in neurologically intact college students working under adverse conditions. Indeed, the size of the gap in comprehension between actives/subject clefts and passives/object clefts can be titrated by the amount of stress imposed on the processing system. Imposition of single perceptual stressors (noise mask, compressed speech, and low-pass filter) causes reliable but relatively mild decrements in performance on passives and object clefts, with relatively intact performance on actives and subject clefts. A digit load appears to have little overt effect on syntactic processing (either alone or in combination with other stressors), whereas imposition of dual perceptual stressors causes a superadditive effect on comprehension, rendering it indistinguishable from that of anomic aphasic patients. These results are largely compatible with a prior literature (reviewed in the introduction) showing that selective deficits in grammatical processing (particularly in agreement morphology) are also observed in a wide array of patient groups and can be simulated in young normal participants with a number of perceptual and attentional stressors. Putting these lines of evidence together, we suggest that the defining profile of receptive agrammatism has no localizing value, is not specific to Broca's aphasia, is not specific to damage at any particular lesion site, and can be reproduced in normal participants under a broad array of adverse processing conditions. Moreover, the emergence of this profile closely follows that suggested by the distribution of the grammatical structures in the linguistic environment.

A potential counterargument to this general position might be found in the surprising absence of any deleterious effect for the visual and auditory digit load manipulations, either alone or in tandem with peripheral stressors. This finding is particularly striking in view of the fact that this same manipulation does have a clear impact on morphological processing, reducing sensitivity to errors of agreement in a grammaticality judgment tasks (e.g., Blackwell & Bates, 1995) and markedly reducing (albeit in language-specific patterns) the use of SV agreement in a sentence comprehension task in English and Italian (Bates et al., 1994) as well as German (Strube, 1996). Several explanations for this puzzling disparity could be advanced.

Perhaps the most parsimonious is simply that the digit task used is less stressful than the perceptual manipulations and, therefore, should only have an effect on "weak links" in the processing chain, such as NV agreement. In support of this conjecture, pilot work in our laboratory has shown that the effect of a digit load on morphological processing is much smaller when compared with the effect of a noise mask on the same task (both in grammaticality judgment and sentence interpretation). Therefore, the digit load tasks we used may simply be much weaker than all of the other stress conditions and, therefore, effective on only the weakest language structures (such as NV agreement morphology in English). Hence, its effect on a strong source of information would be negligible. This hypothesis is supported by recent data in German (Dick, Bates, Ferstl, & Friederici, 1999), suggesting that NV agreement (a weak cue in German) is somewhat susceptible to digit load, whereas case information is impervious to digit load and

possibly somewhat more robust to perceptual distortions as well. Paired electrophysiological and behavioral studies of morphosyntactic comprehension in Dutch also support the notion that interactions of syntactic and digit load effect may be difficult to observe with some psychometric measures, but surface when a more sensitive technique (event-related potentials) is used (Vos, Gunter, Kolk, & Mulder, 2001). Indeed, the lack of a digits effect cited by Caplan and Waters (1999) as evidence for a domain-specific syntactic module may stem from the same weakness (although some of their own on-line results suggest there may be an interaction of digit load and syntactic complexity; see Caplan & Waters, 1999, p. 84). The lack of interaction with other perceptual stressors may also be the outcome of this weakness; in other words, the effect of a powerful stressor (such as a noise mask) may swamp any influence of a weak one (digits). Perceptual stressors may also be more effective in impairing morphological processing simply because of the acoustical vulnerability of closed-class items. We are currently conducting more experiments on the effect of various speech compression manipulations (a more "central" stressor) on inflectional morphology to tease these issues apart.[8]

It is important to note that the digit task used here involves recognition of a second string of numbers, rather than the recall of these numbers. Innumerable studies on memory have shown that recognition is less effortful than recall and may in fact involve different mechanisms and strategies (Shanks & St. John, 1994; Squire & Knowlton, 1995). Hence, imposition of a digit recall may have a more dramatic effect on syntactic comprehension. Importantly, we should compare effects of both these manipulations on performance of linguistic and nonlinguistic tasks (such as mathematical calculation or visuospatial processing). In addition, we should consider alternative explanations for the underlying processes involved in digit and other memory tasks; for example, whether these manipulations affect memory "stores" per se or are better characterized as a kind of learning or attentional effect (see MacDonald & Christiansen, in press, for an extended discussion of related points).

If the digit load is simply exerting a weak attentional or perceptual effect, one should expect to see some superadditive effects of digits and perceptual stress on the weakest language cues (e.g., ones that are affected by digits alone). We are currently testing this hypothesis in our laboratory. However, the fact that digit load effects have yet to show an interaction with such perceptual stressors suggests that there may be an underlying distinction between the mechanisms affected. One possibility may lie in the difference between the processes of *encoding* versus *maintenance*. By encoding we refer to the processes by which linguistic cues are identified by the perceptual system and transformed into a format that can be used for the purposes of interpretation and role assignment. By maintenance we refer to the processes by which these representations are held, or alternatively, "repercolated," until the interpretation is complete. For present purposes, we suggest that the perceptual stressors used in our experiments (i.e., noise, filtering, and compression) might have their primary effects on the peripheral and/or central processes involved in stimulus encoding. By contrast, the digit load manipulation might have its primary effect on the processes involved in maintenance.

If a digit load does primarily affect maintenance, then we should see (a) significant effects of a digit load on comprehending long-distance dependencies (such as noun–verb agreement in sentences with many intervening dependent clauses) and (b) significant effects of the number of digits in the digit load (in which increasing digit numbers should cause increasing difficulties in maintaining the aforementioned long-distance dependencies). Results in the current literature on digit load effects are equivocal with regards to this point; future studies should help to resolve the character of the mechanisms underlying the differential effects of this stressor.

Conclusion

Agrammatism has been defined in terms of a constellation of expressive and receptive deficits, including omissions and substitution of function words and inflections in language production, and receptive deficits in the processing of closed-class morphemes and complex syntactical structures involving noncanonical word order. Since the 1970s, research based primarily on research with English-speaking patients has shown a tendency to identify this complex of deficits with Broca's aphasia—a nonfluent syndrome that is correlated with damage to frontal areas of the left hemisphere (although the reliability and magnitude of this correlation are still controversial; see Dronkers et al., 2000; Willmes & Poeck, 1993). The putative correlation between grammatical symptoms and a specific lesion site has been cited in support of a localizationist view in which the mind/brain is organized into a set of dedicated modules (i.e., transparent mapping), each devoted to a particular cognitive domain (i.e., domain specificity). In other words, agrammatism has been used to argue for the existence of a mental organ for grammar.

In this article, we have shown that the landscape of grammatical deficits is in fact much more expansive than might previously have been believed. Expressive and receptive agrammatism are not unique to any single aphasic group, and hence, by extension, they are not uniquely identified with lesions to any specific region of the brain (see Dick, Wulfeck, et al., 1999, for developmental evidence speaking to this issue). If one looks outside the boundaries of English (with its impoverished system of grammatical morphology), then it becomes clear that expressive agrammatism occurs in both fluent and nonfluent aphasia. The nature of the symptoms may be better understood as a function of relative fluency, in which patterns of errors within groups converge over lexical and grammatical structures: Errors of lexical and grammat-

[8] D. Caplan (personal communication, October 1998) has suggested that these perceptual stressors do not affect syntax at all, but instead reduce or eliminate the listener's ability to perceive the function and content words that are critical inputs to the autonomous syntactic module. In other words, our participants would simply be "guessing," applying a high-probability first-noun strategy that earns them above-chance performance on actives and subject clefts but chance performance on the other types. However, if this were the case, we would see commensurate and equivalent decreases in comprehension accuracy for passives and object clefts (such as those in our first-noun strategy cluster). Furthermore, if lexical items were not perceived under stress, it would be impossible to apply a first-noun strategy in the randomized and counterbalanced design, in which the identity of the first noun does not correspond to any nonlinguistic information.

ical *omission* are observed in nonfluent patients, whereas errors of lexical and grammatical *substitution* and *commission* occur in fluent patients. These patterns of omission or commission are observed in a wide range of patient groups and may have more to do with a speed–accuracy trade-off than with lesions to any specific cortical region.

A similar story emerges for receptive agrammatism: Deficits in the receptive processing of function words, inflectional morphology, and complex syntactic structures are observed in a wide range of populations. Even more compelling, these receptive deficits can be reproduced in healthy young normal individuals by having them process sentence materials under adverse processing conditions—conditions designed to simulate various kinds of perceptual, attentional, and mnemonic deficits. Selective impairments in the use of grammatical morphology are observed under a wide range of conditions, including a partial noise mask, low-pass filtering, temporal compression, and cognitive overload from a secondary task. As shown in our experiments here, selective deficits in the interpretation of noncanonical syntax are also observed in normal participants under stress, although there are differences in the range of stressors that elicit morphological versus syntactic deficits. Specifically, grammatical morphology is affected by the digit task, but the same task often has little or no effect on the processing of complex syntax. By contrast, both aspects of grammar are affected by compression and perceptual degradation, and superadditive effects are observed when these two perceptual stressors are combined, reproducing aphasia profiles both quantitatively and qualitatively.

Interestingly, we have replicated both the findings of Miyake et al. (1994; who simulated receptive deficits in syntax in normal control participants through speeded presentation) and the apparently contradictory findings of Caplan and Waters (1999; who generally failed to find receptive deficits in syntax in normal control participants with a secondary digit load). With reference to the latter point, we have already discussed some possible reasons for the partial dissociation shown by the fairly robust effect of digits on morphology, but lack of consistent digits effect on syntax, and we proposed some avenues of research to pull these options apart. Regardless of the outcome of these proposed studies, one conclusion is already clear: The deficits that make up agrammatism do not provide evidence for a localized, domain-specific organ for grammar. The new evidence we provide here forces this conclusion, even under the more restricted definition of agrammatism suggested by Grodzinsky (2000), Mauner et al. (1993), and others (e.g., the core data of actives, subject clefts, passives, and object clefts).

However, as a counterpoint to such a conclusion, Grodzinsky (2000) stated, "It is common, especially in the social sciences, to say that a theory must be accompanied by a clear procedure for falsification. Somehow . . . this requirement is overemphasized and misconstrued in biology (and the social sciences). It is important to note that a theory is a best 'refuted' not by data, as some commentators . . . erroneously contend, but, rather by an alternative proposal" (p. 56). Although we do not agree with the idea that falsifiability is overrated, we do note that our alternative proposal (in the form of the competition model) in fact fits the available data closely and parsimoniously.

As we noted in the introduction, the case against classic localization has often *been stated in negative terms* (i.e., as the absence of localization, transparency of form-function mapping, and domain specificity). Yet, a century of neuroscience research has established that the brain is a highly differentiated mechanism, with considerable division of labor from one region to another. In closing, we offer the following as an alternative to this dilemma.

We now know that evolution of form over species is an extremely conservative process (Gerhart & Kirschner, 1997). Given this fact, we might expect to see that the functional and behavioral attributes defining our particular species will arise out of quantitative "tweaks" of the sensorimotor cerebral organization common to the rest of our mammalian family, with language being the specialization sine qua non. Just as the giraffe has achieved its ability for high leaf eating through quantitative adjustments in a neck that continues to carry out other neck functions (e.g., swallowing, breathing, holding, and moving the head), the human brain may achieve its specialization for functions, such as language through quantitative adjustments to cortical and subcortical regions that continue to carry out the basic sensorimotor functions for which they originally evolved. If this vision of evolution is correct, then we should not expect to find complex functions such as grammar within any single, bounded, and compact region of the brain. We should instead expect to find that many different regions of the brain participate in this function, even though each region may participate in a different way, making a different kind of contribution (see Bates & Dick, 2000, for further discussion of these points).[9]

If this distributive account of brain organization for language is correct, then grammatical deficits following focal brain damage ought to resemble the kinds of deficits we see in a complex system with local or diffuse damage. Importantly, in any complex system, we cannot impute functional mechanism by means of observing localized damage. As Sheila Blumstein has noted (personal communication), if one were to apply a "localizationist" logic to car mechanics, one could easily be drawn to the conclusion that air pressure in the tires is the mechanism by which we steer (because a flat tire makes steering almost impossible). The same conclusion holds true for the uniquely complex system that is our brain; each skill we possess is mediated by a vast number of different processing areas, with some lying at more critical junctions or playing a more critical role than others. Damage to a particular region may result in serious deficits in a particular skill, but by no means can this region be construed as the mechanism mediating the skill in question.

In this article, we have shown that highly selective deficits in grammar can be reproduced by altering the processing climate, changing the sensorimotor conditions under which grammatical processing must be achieved. These results are precisely what we would expect under a distributive scenario for language and other complex cognitive skills. This is not an antilocalizationist view. Rather, it is an alternative in which our understanding of brain organization for language can be informed by many other aspects of neuroscience, including evolutionarily informed models of sensorimotor organization and activity.

[9] Using the kinds of stress conditions presented here, we may be able to better assess the similarities in the ways that complex systems—such as language, motor control, and musical performance—use these neural resources; we are currently conducting experiments in this vein.

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