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SOME ASPECTS OF LANGUAGE PROCESSING REVEALED THROUGH THE ANALYSIS OF ACQUIRED APHASIA: The Lexical System

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INTRODUCTION

Acquired aphasia is the loss of some aspect of language processing consequent to brain damage. The specific form of aphasia observed in a patient is determined by the locus of cerebral insult. However, given the complexity of the language processing system, involving as it does complex linguistic mechanisms—phonological, lexical, syntactic, semantic, and pragmatic—as well as associated cognitive systems (e.g. working memory), a vast number of different forms of aphasia may be observed. Each form of aphasia observed is presumed to result from the particular type of damage to a component or combination of components of the language processing system. It is unrealistic, therefore, in the limited space available here, to attempt a review of the full range of possible language deficits in aphasia. A more manageable task is to focus the review on just one subsystem of the language faculty. This review focuses on the lexical system.

Normally in a review I would at this point move directly to a presentation of the main theoretical and empirical developments in the area of lexical processing and the analysis of diverse forms of aphasia involving lexical deficits. However, developments over the past decade have led to a reconsideration of the theoretical and methodological underpinnings of the once dominant approach in neuropsychological research, with the result that

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the approach has been challenged and the interpretability of most of its empirical findings questioned. The full implications of this challenge are only now becoming apparent. It is necessary, therefore, to consider briefly the nature of this critique so as to motivate the selection of material reviewed here (as well as the exclusion of certain materials).

The organization of this chapter is as follows. First I present a brief critique of the classical approach in neuropsychological research and a discussion of the theoretical and methodological assumptions of a new approach identified as *cognitive neuropsychology*. I then review the major empirical and theoretical developments in the area of lexical processing and lexical deficits in aphasia. A brief discussion of the implications of these results for a functional neuroanatomy of language concludes the review.

METHODOLOGICAL AND THEORETICAL ASSUMPTIONS FOR A COGNITIVE NEUROPSYCHOLOGY OF LANGUAGE

The modern study of acquired language disorders is based on a set of theoretical and methodological principles that distinguish it from, and even put it in opposition to, the classical study of aphasia. This latter approach is primarily concerned with establishing clinico-pathological correlates for different forms of aphasia. By contrast, the modern study of acquired aphasia has as its objective that of specifying the computational structure of normal language processing. Within this framework, those relationships between the cognitive/linguistic mechanisms comprising the language faculty and brain structures that may emerge from the analysis of aphasia, while very important, do not constitute the principal objective of research. That is, although research on aphasia will undoubtedly serve to provide an important source of constraints on a functional neuroanatomy for language processing, it need not, and in much recent work it appears not to, be explicitly committed to such a goal. This does not mean that cognitive neuropsychology is unconcerned with the problem of relating cognitive mechanisms to the brain. To the contrary, the relationship of cognition to the brain is one of its objectives, but such a goal cannot take precedence over that of specifying the nature of the cognitive mechanisms that must be neurally implemented. To state the problem differently, the objective of cognitive neuropsychology is to articulate and to attempt to answer the correct type of empirical questions about brain/cognition relationships—questions that can only be formulated through an explicit theory of cognitive functioning. Thus, a "neuro-sci-

entific" theory of cognitive abilities will not be formulated by directly relating behavior to neural events but through the mediation of cognitive operations. Cognitive neuropsychology rejects as prejudicial the eliminative materialism of some neuroscientists [and philosophers; cf Churchland (1986)] and operates instead with the assumption that a neuroscientific theory of cognition will be a theory about cognitive mechanisms and not directly about behavior; cognitive descriptions of mental events will not be replaced by neural descriptions but may be reduced to this latter level of description, should such a day arrive.

Classical neuropsychological research operated within a medical model framework mostly uninformed by cognitive or linguistic theory, and certainly unconcerned with the objective of developing a computationally explicit account of language processing. The syndromes that were correlated to anatomical sites for clinico-pathological analyses were based on impoverished notions of language processing using clinically derived, common sense classification schemes for language impairments (e.g. Benson 1985, Damasio 1981, Kertesz 1985). The symptoms that comprised the syndromes were grossly nonanalytic behavioral categories such as poor repetition, poor auditory language comprehension, poor naming ability, and so forth—behavioral conglomerates that are subserved by highly complex sets of cognitive and linguistic mechanisms. There are several reasons for rejecting this approach as a framework within which to explore the structure of the cognitive/linguistic mechanisms that subserve language processing and their relationship to the brain. However, before briefly presenting the details of this critical analysis I should like to consider one major accomplishment that has been achieved through research carried out within this framework.

Despite the serious limitations of this approach, what little is known about the functional neuroanatomy for language has come to us principally through clinico-pathological correlations for the aphasias. Although it has been known at least since the time of Hippocrates (ca 400 B.C.) that insult to the brain may result in disturbances of the language faculty, it was not until the detailed analysis of Broca, Wernicke, Charcot, Lichtheim, Dejerine, and others in the second half of the nineteenth century that a firm foundation was laid for relating language processes to the brain. Indeed by the end of that century French and German neurologists had described all the major aphasia syndromes at a level of detail that seemed to allow little opportunity for improvement. These investigators, under the influence of Gall's phrenological hypothesis, which proposed that distinct areas of the cerebral cortex subserve different cognitive faculties, set out to chart the functional burden of distinct parts of the cortex; that is, to localize the language faculty and its principal subcomponents in particular areas of

the brain. Their labors and those of other students of aphasia since that time have not gone unrewarded. Neuropsychologists have amassed a systematic body of observations relating locus of brain damage to patterns of language dysfunction. These observations have estabished not only that language processing is subserved by neural structures in the left hemisphere (in most people) but that there is a highly articulated functional organization within this hemisphere, with different parts assumed to subserve different components of language processing. These results are well known and have been reviewed many times (e.g. Caplan 1987, Caramazza & Berndt 1982, Damasio & Geschwind 1984).

The general picture to emerge from this research program may be summarized thus: The linguistic components of language processing—syntactic, morphological, lexical-semantic, and phonological—are subserved by neural structures in the perisylvian region of the left hemisphere (see Figure 1); other regions of the brain, notably the right hemisphere, play a less important, supportive role in language processing. Thus, there is now considerable evidence that an intact right hemisphere may be needed for subtle interpretation of language, such as the appreciation of irony, metaphor, and humor as well as the emotional content of a linguistic act, but not for strictly linguistic processing (e.g. Brownell et al 1984, Gardner et al 1983).

This general view of the neural representation of language processes has received considerable support from neuropsychological research with other methodologies and techniques. Research with split-brain patients (patients whose two cerebral hemispheres have been disconnected for medical reasons), where the capacities of the two hemispheres may be

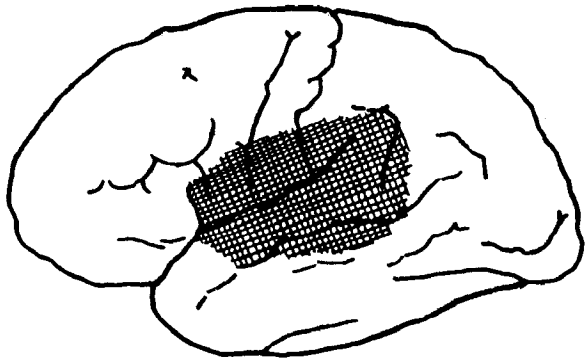

Figure 1 Schematic representation of the lateral surface of the left hemisphere with shading of the perisylvian region.

investigated in relative isolation, has confirmed that linguistic capacities are represented exclusively in the left hemisphere. A similar conclusion has been reached through electrical stimulation research of the exposed cerebral cortex during neurosurgical procedures. This latter research has shown that electrical stimulation of the cortex results in temporary linguistic impairments only when the stimulation is applied to the perisylvian region of the left hemisphere. And, finally, studies of regional cerebral blood flow with emission tomography during language activities in normal subjects have also led to a similar conclusion. These studies, which measure the metabolic (blood flow) activity in different regions of the brain during the performance of language tasks, have shown that it is the perisylvian region of the left hemisphere that is most directly implicated in language processing (cf Caplan 1987).

Without in the least intending to minimize the importance of that which has been learned about the neuropsychology of language through the methods currently at our disposal, I emphasize, nevertheless, that we have only succeeded in providing a gross, nonanalytic mapping of the language faculty onto the brain—at best a gross functional neuroanatomy. Is this the most that may be achieved through the analysis of language disorders consequent to brain damage? This question receives different answers depending on whether we place the focus on the neural or the cognitive part of the brain/cognition equation. Let us consider first the brain part of the equation.

The answer here is not an entirely encouraging one. The effort to relate functional disorders of language to locus of brain damage, no matter how fine-grained an analysis, can only result in a "modern phrenology." This is not to say that such an achievement would be insignificant. Quite the contrary: A fine-grained mapping of component parts of the language processing system onto neural structures would place important constraints on theories of the neurophysiological bases for language. But the type of observation at our disposal cannot lead to a neurophysiology of language. Furthermore, the fact that natural language is a uniquely human ability severely restricts the range of experimental opportunities for exploring the neurophysiological mechanisms for language processing—for example, we cannot use those experimental procedures currently within the armamentarium of the neurophysiologist for the analysis of neural activity in nonhuman animals. Does this mean that we must abandon the hope for a neurophysiology of language? Although current opportunities are limited, there is the hope that technological developments will eventually make it possible to investigate neural activity in humans directly with ethically acceptable means. In the meantime, we are not completely disarmed. We could rely on a bootstrap strategy that exploits whatever

brain/cognition principles might emerge from the analysis of diverse cognitive processes in various nonhuman species to develop a computational neurophysiology of language processing; that is, a theoretical neurophysiology that relies on principles of neuronal functioning to develop neuronal-net models of specific linguistic processes—an approach that has received considerable attention in recent years (Arbib et al 1982, Hinton & Anderson 1981, Rumelhart & McClelland 1986). I return to this general issue in the concluding section of this review.

By contrast to the less-than-optimistic conclusion about the possibility of an experimental neurophysiology of language, the outlook for progress in developing a detailed functional theory of language processing through the analysis of different forms of acquired aphasia is very encouraging. The pragmatic motivation for using language deficits to inform and constrain theories of normal language processing comes from the observation that brain damage does not result in undifferentiated loss of language ability but in the selective loss of some ability in the face of otherwise normal performance. Thus, for example, brain damage may selectively impair language processes while sparing other perceptual and cognitive abilities. However, if brain damage were to result in dissociations of functions that are no finer than global cognitive systems (e.g. language, calculation, etc), the resulting patterns of impaired performance would be of little value in determining the processing structure of these systems. Fortunately for our enterprise, brain damage may result in highly specific patterns of dysfunction, presumably reflecting the componential structure of cognitive systems. We can use these highly articulated patterns of impaired performance to evaluate and develop models of normal language processing. However, such an enterprise cannot be carried out within the framework of classical neuropsychology. To fully appreciate this claim we must consider, albeit very briefly here, the assumptions that motivate the possibility of drawing meaningful inferences about normal language processing from patterns of language disorders (see Caramazza 1986a, for detailed discussion).

As already noted, the object of cognitive neuropsychology is to develop a theory of cognitive functioning through the analysis of patterns of cognitive dysfunction consequent to brain damage. The theoretical assumption that motivates the use of impaired performance as the basis for inferring the structure of normal processes is that the transformations of the normal system under conditions of damage are not indefinite or random but, instead, obey precise constraints determined by the intrinsic structure of the normal system: A pattern of impaired performance reflects a discoverable (and specifiable) transformation of the normal cognitive system (what I have called elsewhere the assumption of "transparency";

Caramazza 1984, 1986a). In this framework, a pattern of impaired performance is taken as support for a theory of the processing structure of a cognitive system (over some alternative theory) if it is possible to specify a transformation—a functional lesion—in the proposed theory (but not in some alternative theory) of the cognitive system such that the transformed system may account for the observed pattern of performance. This procedure allows a precise criterion for the empirical evaluation of a cognitive theory through the analysis of the performance of cognitively impaired brain-damaged patients.

The role played by "functional lesions" in the proposed framework for research is analogous to that played by "experimental conditions" in a typical experimental paradigm; that is, in a regular experiment the relationship between data and theory is mediated by specific experimental conditions, and in research with brain-damaged patients it is mediated by functional lesions (as well as experimental conditions). However, the two situations are disanalogous in one crucial respect: Whereas experimental conditions are under the control of the experimenter (and therefore known a priori), functional lesions are not known a priori but must themselves be inferred from the performance of patients. Thus, although we may consider a brain-damaged patient as constituting an "experiment of nature," where the functional lesion represents some of the experimental conditions of the experiment, these latter conditions are not known a priori, as would be the case in a regular experiment, and therefore they raise particular problems whose solution has important methodological consequences. Specifically, given that functional lesions may only be specified a posteriori—that is, once all the relevant patterns of performance for inferring a functional lesion in a cognitive system are available—there can be no theoretical merit in a classificatory scheme of patients' performance that is based on any arbitrary subset of a patient's performance. Two important consequences follow from these observations: (*a*) patient classification cannot play any significant role in cognitive neuropsychological research and (*b*) patient-group studies do not allow valid inferences about the structure of normal cognitive processes.

On the issue of patient-classification-based research, not only are there methodological arguments against its validity but, in addition, there are theoretical and practical considerations that undermine its usefulness (see Badecker & Caramazza 1985, Caramazza 1984, Caramazza & Martin 1983, Marshall 1982, 1986). The great majority of classification-based research has used theoretically uninformed behavioral categories for patient classification. Patients are classified as being of a particular type on the basis of criteria such as the following: whether or not a patient has poor repetition performance, or poor language comprehension perfor-

mance, and so forth. However, since performance of such complex tasks as repetition or comprehension involves many cognitive mechanisms, impaired performance on these tasks may be due to damage to any one or combination of the cognitive mechanisms implicated in the performance of the task as a whole. Thus, poor performance in such tasks does not guarantee a theoretically useful homogeneity of the patients classified by these criteria. Furthermore, there is little value in reviewing classification-based research on aphasia for strictly pragmatic reasons. This research has led to little if any insight into the structure of normal language processes despite over a century of work.

The second major consequence of recent analyses of the logic of research in cognitive neuropsychology is that valid inferences about the structure of cognitive systems from patterns of cognitive dysfunctions are only possible for single-patient studies (Caramazza 1984, 1986a, Caramazza & McCloskey 1988, Shallice 1979). The arguments for this contention are straightforward but too long to present here. Suffice it to say that the principal argument is based on the observation that functional lesions can only be postulated a posteriori—that is, on the basis of all the relevant evidence needed to fix a functional lesion in a cognitive system.

Thus far I have focused on some negative conclusions of recent methodological and theoretical developments in cognitive neuropsychology; that is, I have presented recent conclusions concerning the impossibility of using the clinically based, classical methods of research on aphasia for learning about the structure of normal language processes and their neural correlates. A focus on these negative conclusions has been found necessary because of the need for clearly identifying the type of theoretical questions that may be profitably addressed through investigations of patients with cognitive deficits and for specifying the attendant methodology for addressing these issues. These developments may also be viewed positively, however: They offer us a theoretically coherent basis for a productive cognitive neuropsychology that increasingly interacts with other subdisciplines of the cognitive and neural sciences.

THE LEXICAL SYSTEM

Even though the focus of this review has been resricted to just a single subsystem of the language processing system, the ground to be covered is still quite extensive. The lexical system is very complex involving many linguistic and cognitive dimensions as well as being implicated in many different types of cognitive functions such as sentence comprehension and production, reading, writing, and naming. Consequently a further restriction of focus is necessary. The primary focus will be on single-word

processing tasks, although an effort will be made to link the account of the lexicon that emerges from the review to the broader issue of sentence processing. Three sets of issues will be dealt with in this review: the general architecture of the lexical system; the representational content in different lexical processing components; and the processing structure within components. Although these issues are not entirely independent, it is useful to draw these distinctions for purposes of exposition.

The Functional Architecture of the Lexical System

The dominant view of the functional architecture of the lexical system is that it consists of a distributed but interconnected set of lexical components (e.g. Allport & Funnell 1981, Caramazza 1986b, Morton 1981, Shallice 1981). Over the past 10 to 15 years an impressive range of theoretical arguments and empirical evidence has been amassed in support of this view. The modal model that has emerged has the following structure. A major distinction is drawn between input and output lexical components; that is, lexical components involved in the comprehension (recognition) or production of words, respectively. A second major distinction is drawn between modality-specific input or output lexical components: The orthographic input lexicon, those mechanisms involved in processing written words, is distinguished from the phonological input lexicon, those mechanisms involved in processing spoken words. These modality-specific input lexicons are distinguished from their corresponding output lexicons, those mechanisms involved in the production of written and spoken words. It is further assumed that modality-specific lexical components are interconnected through a lexical-semantic system that stores the semantic representations for words. A schematic representation (as a visual aid) of these processing components is shown in Figure 2.

The evidence in favor of this view of the architecture of the lexical system is quite compelling. On strictly theoretical grounds the distinction between modality-specific components is unimpeachable—the mechanisms involved in processing visual and acoustic signals and the orthographic and phonological lexical representations these give rise to, are computationally independent. In one case—reading—the computational problem involves computing a lexical representation on the basis of visual information and subsequently letters or graphemes; in the other—listening—the computational problem involves computing a lexical representation on the basis of acoustic information and subsequently phonetic and phonemic information. Obviously, the computed representations must be different objects—orthographic or phonological lexical representations. A similar argument may be made for the input/output lexicon distinction.

The available empirical evidence is no less compelling. Two types of

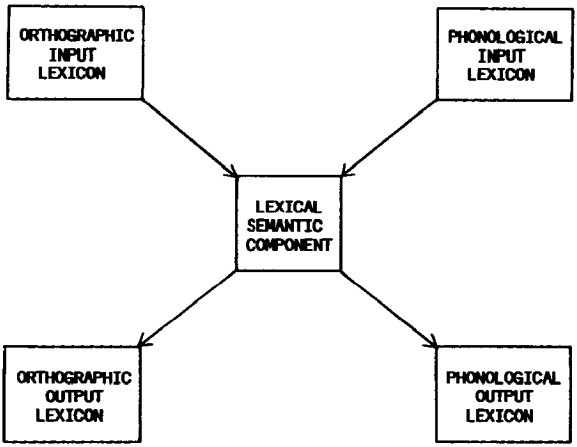

Figure 2 Schematic structure of the lexical system.

evidence have been reported: patients who present with selective damage to one or another lexical component (e.g. Basso et al 1978, Hier & Mohr 1977, Miceli et al 1985, Michel 1979) and patients who present with different patterns of impairments to different components (e.g. Beauvois & Dérouesné 1981, Goodman & Caramazza 1986a). Both kinds of evidence may be taken as support for a distributed view of the lexical system. Thus, for example, Goodman & Caramazza (1986b) have reported a patient who presents with damage to the output graphemic lexicon but who has normal access to other components of the lexical system (e.g. the output phonological lexicon and the lexical-semantic component). This pattern of performance is strong evidence against any theory of the lexicon that assumes a nondistributed, unitary lexical system. Equally supportive of the distributed theory of the lexicon are those patterns of performance in which different types of dysfunctions are found for different components of the lexicon. To give just one example of this type of result, Beauvois & Dérouesné (1979) have reported a patient whose impaired reading performance was radically different from his impaired spelling performance. This patient's impairment in reading involved only those cognitive mechanisms required for converting print-to-sound for novel or unfamiliar words—the patient could not read nonwords but essentially had no difficulty in reading words (see also Goodman & Caramazza 1986a). Thus the graphemic input lexicon must be intact, as must be the phonological output lexicon. By contrast, this patient's spelling impairment resulted from damage to the graphemic output lexicon that spared those mechanisms involved in converting sound-to-print and the phonological input lexicon

(see also Goodman & Caramazza 1986a). This pattern of dissociation of deficits can only be explained by assuming selective damage to different components of a distributed lexical system. There is now a vast cognitive neuropsychological literature that demonstrates differential patterns of impairment for different parts of the lexical system (see also Allport & Funnell 1981, Shallice 1981 for reviews).

Lexical Representations

The distributed lexical system under consideration here distinguishes between modality-specific lexical components. These distinctions capture the most salient (perceptual) features of lexical information—phonological and orthographic information are represented in distinct processing components. However, there are other important lexical features that must be accounted for in a more general theory of lexicon. These include form class or categorial information (i.e. noun, verb, etc), morphological structure (root/stem and affixes), thematic structure (the argument structure of predicates), and semantic information (the meaning of words and morphemes). This information must be captured at some level of the lexical system. In this section I review some of the experimental evidence in favor of these representational distinctions. I also briefly review theoretical arguments and empirical evidence that bear on the issue of which lexical components may be assumed to capture the hypothesized lexical features.

FORM CLASS Although words have an independent status, their primary function is to convey meaning in sentential contexts. It is only when words are used in sentences that the full range of their syntactic, semantic, morphological, and phonological properties become apparent. Thus, for example, the word "jump" may be used as a noun (I watched the jump with trepidation) or as a verb (I watched him jump with trepidation). The two uses of "jump" have distinct grammatical roles (noun vs verb), different meanings, and accept different inflectional affixes (the noun accepts -s for plural, the verb accepts -s, -ing, and -ed to mark person and tense). The grammatical class of a word and its subcategorization features (e.g. transitive/intransitive) also determine the type of derivational affixes it accepts (e.g. only verbs accepts the -able derivational affix as in "enjoyable" but not "*windowable" and, furthermore, this applies only for transitive verbs as in "enjoyable" but not "*appearable"). Clearly, then, the lexicon must represent not only the phonological and orthographic structure of words but also their syntactic, semantic, and morphological properties (e.g. Chomsky 1965).

As indicated above, a crucial property of lexical items is their form (grammatical) class; that is, whether a word is (functions as) a noun, a

verb, an adjective, an adverb, or a function word. These lexical properties play a determining role in the organization of the lexicon. Already in the classical literature there were clear indications for the dissociability of impairment of different form classes of words. The strongest evidence was for the dissociability of function words (articles, auxiliaries, prepositions, etc) from other form classes (nouns, verbs, and adjectives) (e.g. De Villiers, 1978, Goodglass 1976, Stemberger 1984; see Berndt & Caramazza 1980, Lesser 1978 for reviews). Patients clinically classified as agrammatic aphasics—that is, patients whose spontaneous speech is characterized by the relative omission of function words—could be argued to have a selective impairment in lexical access of function words (this position has been argued most forcefully by Bradley et al 1980). However, this type of deficit does not allow us to distinguish between a deficit at some level of sentence production (where a syntactic frame for sentence production is specified) and a pure lexical access deficit (Caramazza & Berndt 1985, Miceli & Caramazza 1988). Nonetheless, independently of whether the deficit in any one patient who makes errors with (or omissions of) function words in sentence production is ultimately found to be at the level of specifying a sentence frame or in lexical access, such patterns of language impairment are prima facie evidence for a representational distinction between function words and other word classes, and hence for a particular form of organization of the lexicon.

More direct evidence exists for the selective impairment of lexical access of function words. There are reports of patients whose performance in single word processing—reading, writing, or repetition of single words—is either relatively poor or relatively good when compared to other form classes (e.g. Bub & Kertesz 1982, papers in Coltheart et al 1980, Friederici & Schoenle 1980, Nolan & Caramazza 1982, 1983). Although it initially appeared that a deficit in function word processing was associated with a more general morphological processing impairment (Beauvois & Dérouesné, 1979, DeBastiani et al 1983, Patterson 1982), it is now clear that these two types of deficits are dissociable (Caramazza et al 1985, Funnell 1983). Thus, we have evidence for at least one type of organizational distinction at some level of the lexicon. (I take up the issues of the level at which these distinctions may be represented, below.)

Evidence for other organizational distinctions within the lexicon has also been obtained. There are numerous reports of patients whose reading or writing performance is differentially affected for nouns, verbs, and adjectives (see papers in Coltheart et al 1980). The typical result reported is for better performance for nouns relative to verbs and adjectives. The systematicity of this result raises the possibility that a lexical dimension other than form class is responsible for this ordering of performance

difficulty. Indeed, patients who present with the form class effect described, typically show greater difficulties in processing abstract than concrete (or high imageability) words. This association of deficits allows the possibility that the relevant dimension affected by brain damage in the patients in question is not form class but concreteness/abstractness. However, an effect of form class has been obtained even when concreteness/abstractness is controlled for (e.g. Baxter & Warrington 1985, Shallice & Warrington 1975). Furthermore, there is evidence in the literature on naming disorders for a double dissociation in naming difficulty for verbs and nouns (Baxter & Warrington 1985, McCarthy & Warrington 1985, Miceli et al 1984). Some patients have considerably greater difficulty naming nouns than verbs, other patients present with the reverse pattern in naming difficulty for nouns and verbs. This result suggests that, at least in some patients, the underlying cause of their naming impairment is selective damage to different subsets of the lexicon—subsets defined by form-class membership. The double dissociation of processing difficulty for nouns and verbs has also been documented for a word comprehension task (Miceli et al 1988). In this latter case the reported dissociation for form class also concerned a dissociation by modality of use. That is, some patients presented with selective impairment in comprehension of verbs without a corresponding difficulty in naming of this class of words.

The results reviewed in this section are unequivocal in one regard: they support the view that the lexicon is organized by grammatical class. They do not provide as compelling a basis, if at all, for determining where in the lexical system form class information is represented. Nonetheless, I propose that presently our best answer to this latter question is that form class information is represented in each modality-specific lexicon (i.e. in the phonological input and output lexicons, and in the orthographic input and output lexicons). Although empirical support for this position—modality specific form class effects in lexical processing (e.g. Baxter & Warrington 1985)—is scanty, there are good theoretical reasons for adopting it. Basically, the argument is that since morphological structure is strictly dependent on form class information, this latter information must be represented at the same lexical level as that at which morphological structure is represented. And, as we will see below, morphological structure is represented in modality-specific lexicons.

MORPHOLOGICAL STRUCTURE Words are not unanalyzable units—they have phonological (and orthographic) and morphological structure. The word "nationalized" is considered to be composed of the verb stem "nationalize" plus the inflectional affix, -ed (past tense). In turn, the verb "nationalize" is derived from the adjective "national" by the addition of

the derivational affix, -ize, which is itself derived from the noun "nation" by the addition of the adjectival derivational affix, -al. Thus, we may analyze words into stems (or roots), derivational affixes [affixes that serve to specify the form class of the derived word; e.g. nation (noun) → national (adjective)], and inflectional affixes that mark the tense, number, and gender of a word (see Scalise 1984 for review). A crucial issue for a theory of the lexicon is whether morphological structure is explicitly represented in the lexicon and how it is represented and used in language processing.

Various theoretical positions have been taken on this issue. A major contrast is between the view that words are represented in the lexicon in morphologically decomposed form (e.g. Taft 1985) versus the view that words are represented as nondecomposed wholes (e.g. Butterworth 1983). A second distinction, relevant only for the case of morphologically decomposed lexical representations, is whether lexical access is only possible after a word stimulus is parsed into its morphological components (stems or roots and affixes) (e.g. Taft 1979) or whether lexical access may proceed through both whole-word and morphemic access procedures (e.g. Caramazza et al 1985). Other issues concern the proper relationship between derivational and inflectional morphology and whether inflectional morphology is represented in the lexical or syntactic system (e.g. Anderson 1982). In the limited space available here I consider only the general issue of morphological decomposition as it emerges through the analysis of the word-processing performance in brain-damaged patients.

Various reports in the literature have dealt with morphological processing in brain-damaged patients. Some of this research has focused on the patterns of omissions (e.g. De Villiers 1978, Gleason 1978, Goodglass 1976, Goodglass & Berko 1960) or substitutions (e.g. Miceli et al 1983) of inflectional affixes in patients clinically classified as agrammatic aphasics. These reports have clearly documented a dissociation in processing inflectional affixes (impaired) versus word stems ("intact"). The reverse pattern of dissociation, impaired stem production and spared inflectional affix production, has also been reported (e.g. Caplan et al 1972). These patterns of results would appear to be prima facie evidence for morphological decomposition in the lexicon. However, as in the case of function word omission (or substitution) in spontaneous sentence production (discussed above), these results are ambiguous with respect to the locus of deficit: A patient may fail to produce (or fail to produce correctly) an inflectional affix because of damage to the inflectional component of a morphologically decomposed representation or because of damage to a component of the syntactic frames computed in the course of sentence production. The relevant data needed to resolve this issue involves patterns of selective impairment in single word processing. Such data are available.

An important source of evidence comes to us from the oral reading errors in patients with acquired dyslexia. An often noted feature in dyslexic patients in the presence of morphological errors—that is, errors such as reading "walked" for "walking" (inflectional error) or "kindness" for "kindly" (derivational error). Errors of this type were first most clearly documented in patients clinically classified as deep dyslexic. These are patients who in addition to morphological errors also make semantic (read "priest" for "minister") and visual (read "bear" for "fear") errors as well as presenting with other processing impairments [i.e. a form class effect, a concreteness/abstractness effect, a frequency effect, and disproportional difficulties in reading nonwords; see Coltheart et al (1980) for review and discussion]. Although the presence of morphological errors as part of the complex clinical picture in these patients may be suggestive, it does not permit an unequivocal conclusion regarding the issue in question; namely, whether or not lexical representations are morphologically decomposed. After all, the putative morphological errors may be no more than visual or semantic errors. However, the existence of "morphological" reading errors may be used in more focused analyses to address the question of concern here.

Patterson (1980, 1982) and Job & Sartori (1984) have described in some detail patients whose reading errors were almost exclusively of the morphological type. These authors interpreted the highly selective impairment in their patients (essentially restricted to the production of morphological paralexias) as evidence for a selective deficit to the morphological component of the lexicon. This conclusion has been challenged, however. Badecker & Caramazza (1988) have argued that the mere production of "morphological" paralexic errors is not sufficient grounds for concluding that the basis for the impairment is a deficit to the morphological component of the lexicon. Equally plausibly these errors could be considered to be highly similar visual errors or highly similar semantic errors. The ambiguity of interpretation could be resolved only if it turned out that a pattern of errors is only explicable by appeal to a morphological and no other lexical (semantic) or perceptual dimension. Note that this objection does not imply that the cases described by Patterson and Job & Sartori may not, after all, truly be cases of selective deficit to a morphological processing component. All that is asserted is that the presented evidence is not sufficient to unambiguously decide the issue. Fortunately, there is at least one case report of a patient whose impaired lexical processing performance is unequivocally the result of a selective deficit to the morphological component of the phonological output lexicon.

Miceli & Caramazza (1988) have described a patient, F. S., who makes morphological errors in spontaneous sentence production and in repetition

of single words. The great majority of this patient's single word repetition errors were morphologically related to the target response. Crucially, these morphologically related responses were almost all inflectional errors (97%). The massive presence of morphological errors restricted to the inflectional category is only explicable by appeal to a morphological principle—a distinction between inflectional and derivational morphology: the evidence for a true morphological processing impairment. The highly selective deficit for inflectional morphology in a single-word processing task reported for F. S. allows the conclusion that lexical entries are represented in morphologically decomposed form—stems (or roots) are represented independently of their inflectional and derivational affixes, which, in turn, constitute independent components within the lexicon.

In this section I have reviewed evidence in support of the view that the lexical system represents words in morphologically decomposed form. As a final issue in this area I argue that morphological structure is represented directly in modality-specific lexicons. However, the evidence for this conclusion is, at best, indirect.

Caramazza et al (1985) have described a patient with a selective deficit in reading nonwords. The patient could read all types of words but made on the order of 40% errors in reading nonwords. However, when his reading performance for "morphologically legal" nonwords (e.g. "walken," composed of the inappropriately combined morphemes, walk- and -en) was assessed, it was found that he read these nonwords much better than comparable nonwords that did not have any morphological structure (e.g. "wolkon"). Since we may safely assume that nonwords do not have permanent entries in the lexical system, the better performance for the "morphologically legal" nonwords must be due to the activation of morphemic representations (e.g. walk- and -en) in the orthographic input lexicon. If this argument is correct, we must conclude that morphological structure is represented in modality specific lexicons.

In conclusion, the evidence from the analysis of language impairments in brain-damaged patients taken together with results in the literature on normal word processing (e.g. Stemberger 1985, Taft 1985) and linguistics (e.g. Scalise 1984) strongly argues for the autonomous representation of morphological structure in the lexical system.

LEXICAL SEMANTICS That of various features of a word its meaning is the most important is quite obvious. Despite this and despite the fact that word meaning is increasingly seen as playing a determining role in linguistic theory (e.g. Chomsky 1981, Wasow 1985), we do not have the detailed theory of lexical meaning that would be commensurate with the crucial role of this dimension of lexical items. The absence of theory has left

empirical work in this area in disarray so that we do not have anything like a coherent research program in the analysis of disorders of lexical meaning. Consequently, in this section I focus on an interesting empirical phenomenon concerning semantic organization of the lexicon, without attempting to provide a general model of this component of the lexicon (in contrast to what I have attempted to do for other components of the lexical system). The phenomenon I consider here is that of category-specific deficits.

We have seen in preceding sections that brain damage may result in highly specific deficits. The patient with a selective deficit of inflectional morphology or the patients with selective deficit in processing function words are cases in point. Results such as these allow us to articulate the functional architecture of the modality-specific lexicons. In recent years Warrington and her colleagues (Warrington 1975, 1981, Warrington & McCarthy 1983, Warrington & Shallice 1984), following an earlier observation by Goodglass et al (1966), have described a number of patients with selective deficits to specific semantic categories. These results provide evidence relevant to the organization of the semantic lexicon.

Goodglass et al (1966) provided a quantitative analysis of a large number of patients in which they show that different patients present with different patterns of relative difficulty in auditory comprehension of semantic categories. Warrington and her colleagues in a series of detailed single-patient analyses have documented selective dissociations between concrete (impaired) and abstract words (spared) (the reserve pattern is commonly reported), inanimate (impaired) and animate words (spared), and living things and foods (impaired) and inanimate words (spared). Perhaps the most striking result in this domain is one reported by Hart et al (1985). The patient, M. D., presented with a very selective disturbance of the ability to name items from two related semantic categories. Despite normal naming performance with the items from many different semantic categories, the patient showed a striking and consistent naming deficit for the categories "fruits" and "vegetables." Thus, as can be seen in Table 1, the patient performed poorly in naming fruits and vegetables in the face of spared ability to name items from other categories.

The patient's difficulties in processing the members of the categories fruits and vegetables extended to a number of other tasks. Thus, the patient presented with difficulties in sorting pictures of fruits and vegetables into the appropriate categories, i.e. sorting together fruits separately from vegetables; he had difficulties in generating the names of members of the two categories when given the category, i.e. producing apple, orange, peach, etc in response to the category "fruits"; and he showed a selective difficulty in naming fruits and vegetables from definition as well as from

Table 1 Number of correct naming responses[a]

	Semantic category		
	Fruit	Vegetables	Other[b]
Line drawings	5/11	7/11	11/11
Colored drawings	4/6	5/7	18/18
Photographs	11/18	12/18	222/229
Real objects	10/13	13/23	11/11
TOTAL	30/48 (0.63)	37/59 (0.63)	262/269 (0.97)

[a] From Hart et al (1985).
[b] The "other" category includes vehicles, toys, tools, animals, body parts, food products, school, bathroom, kitchen and personal items, clothing, colors, shapes, and trees.

tactile presentation. By contrast, he showed normal performance with these categories in a word-picture matching task and in judgments of category, size, texture, and shape when given the name of individual fruits and vegetables. Normal performance on these latter tasks demonstrates that the patient's knowledge of these categories is intact but can only be accessed from the lexicon.

Although the absence of a well-developed theory of lexical semantics makes it difficult to provide a systematic interpretation of these category (semantic)-specific deficits, these latter results provide a provocative source of data on which far-reaching speculations about the structure of lexical organization may be based. Thus, at the very least, these results strongly argue for a highly structured lexical organization based on semantic categories. The implication of these results for neural organization is considered below.

Processing Principles

The material reviewed thus far has allowed us to address issues concerning the architecture of the lexical system and the types and organization of information represented in lexical components. I turn now to a consideration of the processing principles that govern the access of this information.

Two general classes of lexical processing models have been proposed: serial search models and passive, parallel activation models (activation, for short). Of these two classes, the activation models have clearly emerged dominant over the past decade. The basic assumption of activation models is that a stimulus (or input at some level of the lexical system) activates in parallel all stored representations. The degree of activation of any representation is proportional to the overall similarity between the input

and the stored representation. Thus, for example, the stimulus word "car" will activate the representations "cat," "tar", "cart," "cord," etc to different degrees. In this example, "car" will be activated most strongly and "cat" will be activated more than "cord" and so forth. When the level of activation of a representation reaches a set, threshold value, the representation becomes available for further processing to other components of the processing system. Models of this type are known as *serial stage models*. If we relax the assumption that only the representation that reaches a threshold value can serve to activate subsequent stages of processing and we allow all representations that reach a minimal level of activation to activate representations in other components of the system, we have what are called *cascade models of processing* (McClelland 1979). Here I assume, for the sake of simplicity, a serial stage model (although it is quite likely that the cascading principle is a more realistic characterization of the processing sequence).

A distributed model of the lexical system, as that discussed in this review, which operates on the principle of passive, parallel activation, provides a natural framework for considering various features of impaired language performance. Two such features are the ubiquitous frequency effect (words of high usage frequency are in many cases relatively spared in comparison to words of lower frequency) and certain types of error responses produced by patients in single-word processing tasks.

It is a well-established phenomenon in the psychological literature that reaction time to recognize a word or to decide that a string of letters forms a word (lexical decision) is inversely proportional to the frequency of usage of a word (and, similarly, for error rates) (see Gordon 1983 for review). Activation models account for this effect by assuming that the activation threshold of a representation is lowered with repeated presentations of the stimulus or input (Morton 1970). Thus, high-frequency words have lower thresholds than low-frequency words and, therefore, can be activated more easily, resulting in lower reaction times (RTs) and lower error rates, than low-frequency words. This differential effect of word frequency is also found in aphasic patients' performance (see Gordon & Caramazza 1982). To give just one example, many dyslexic patients make more errors in reading low-frequency words than in reading high-frequency words. What is important for our present concern, however, is that the presence of a frequency effect may be associated with certain types of error responses, thus allowing us to identify the locus of deficit responsible for a patient's impaired performance. That is, we may take the presence of a word frequency effect as an indication of a deficit to the lexical system and the type of error (e.g. visual or semantic) as an indication of a deficit at a specific level within the lexical system.

I indicated above that two types of errors produced by dyslexic patients are visual and semantic paralexias. Various accounts have been offered as the basis for these types of errors (e.g. Caramazza 1986b, Marshall & Newcombe 1973, Morton & Patterson 1980, Nolan & Caramazza 1982, Shallice & Warrington 1980). I argue that, at least in some cases, these errors arise from independent deficits to the graphemic input lexicon and the phonological output lexicon for visual and semantic paralexic errors, respectively.

Recall that visual paralexic errors are errors such as reading "bead" for "head" and semantic paralexic errors are errors such as reading "airplane" for "ship." In an indepth investigation of a single patient, F. M., Gordon et al (1987) asked the patient to read several thousand words in order to obtain a reliable data base of errors for detailed analysis. The patient's responses were scored either as correct or as an error of one of the following types: visual, semantic, inflectional, derivational, or other—where this last category consists of ambiguous errors, visual-to-semantic errors or word responses that could not be classified in any of the previously listed error categories. Here I first wish to focus on the evidential role of visual and semantic errors to constrain a model of the lexical system.

A priori it is unlikely that these two types of errors have a common basis: A semantic error can only occur if the correct lexical entry has been activated; that is, in order to produce "minister" for "bishop," the lexical entry for "bishop" had to be activated. There is no such constraint for visual errors. This latter type of error most likely arises from damage to the input graphemic lexicon, where an inappropriate lexical representation is activated. To explore this issue consider the following argument. A word that is read correctly is one that successfully activates a lexical entry in the input graphemic lexicon and the output phonological lexicon. By contrast, a word that gives rise to a visual error is one that fails to activate its lexical entry in the input graphemic lexicon and instead activates a visually similar entry in this lexicon. Similarly, a word that gives rise to a semantic error is one that successfully activates a correct lexical entry in the input graphemic lexicon, but fails to activate its lexical entry in the output phonological lexicon, and instead activates a semantically related entry. Note that this argument makes two obvious, but important assumptions: (*a*) The access procedure for the input graphemic lexicon is orthographically based; (*b*) the access procedure for the output phonological lexicon is semantically based.

This proposed architecture of the lexical system and, more specifically, the assumptions we have made about the address procedures for the input graphemic lexicon and the output phonological lexicon (i.e. parallel activation), allows us to make a precise prediction about F. M.'s per-

formance on re-reading words read correctly, incorrectly produced responses, words to which he made visual errors, and words to which he made semantic errors on the first reading. The prediction is that he should read very well words he read correctly the first time as well as the incorrectly produced responses but should read poorly words to which he previously made errors. Furthermore, the new errors for words that gave rise to visual errors should be predominantly visual whereas those for words that gave rise to semantic errors should be predominantly semantic. These predictions were borne out.

To further substantiate the claim that visual and semantic errors arise due to difficulties in addressing lexical representations in the input graphemic lexicon and the output phonological lexicon, respectively, we assessed F. M.'s ability to comprehend words that were on a previous occasion read correctly or had resulted in visual or semantic errors. The model of the lexical system proposed here leads to the prediction that F. M. should understand both the words he previously read correctly and those with which he made semantic errors, but he should fail to comprehend the words with which he had made visual errors. This prediction too was borne out.

The implication of these results for claims concerning the processing structure of the hypothesized lexical components is clear-cut. It would appear that a visual error is made when a particular lexical entry in the graphemic input lexicon cannot reach threshold and instead a visually similar representation reaches threshold. Similarly, a semantic error occurs when a representation in the phonological output lexicon cannot reach threshold and instead a semantically related response reaches threshold. This interpretation of the basis for F. M.'s visual and semantic errors is only possible if we assume that lexical representations are activated in parallel and in proportion to the similarity between the input and the stored representation.

CONCLUSION

In this all too brief and highly condensed review I have dealt with three aspects of the structure of the lexical system: the general architecture of the system, the types of representational content in each hypothesized component, and the processing principles that allow access of the information stored in the lexicon. The evidence reviewed not only provides empirical support for the model but, in addition, the model serves as a guide for the interpretation and analysis of cognitive/linguistic disorders. The discussion has focused, however, entirely on functional (cognitive) aspects of the process. We may wish to ask, therefore, whether or not the

types of observations available to us from the analysis of cognitive deficits will be relevant to the formulation of a truly *neuro*psychological theory of cognitive functioning. Is a *neuro*psychology of language possible? In a previous section of this review I sounded a pessimistic note with respect to this question. Here, by way of conclusion, I would like to take up this issue in a little more detail.

The classical study of aphasia has failed to lead to any significant insights into the structure of language processing mechanisms and their neural instantiation, other than the gross clinical-pathological mapping already available at the end of the last century. This work clearly established the importance of the perisylvian region of the left hemisphere for language processing but could not go beyond this general phrenological statement. Theoretical and methodological developments over the past decade have introduced the possibility for significant progress for one part of the brain/cognition equation. We have seen that we now have a clearly articulated justification for drawing inferences about normal cognitive processing from the analysis of patterns of cognitive dysfunction, as well as a powerful theoretical and methodological basis for the analysis of cognitive dysfunctions. This development, by itself, is not sufficient to lead to any significant insights into the nature of the neural mechanisms that subserve language processing. It may be sufficient, however, to provide a set of principled constraints on the possible form of a neuropsychological theory of language processing.

Recent work (some of it reviewed here) in cognitive neuropsychology has provided an impressive set of results on the nature of language dysfunctions. It has been possible to demonstrate that language dysfunction may be highly selective, affecting a single component (e.g. Miceli & Caramazza 1988) or even a single representational dimension within a component (e.g. Warrington 1981). Such observations provide a natural set of constraints for a theory of language processing as amply demonstrated above. However, since the observations that enter into this theory-construction process consist of brain/behavior pairs, we may use them to constrain the formulation of a neuropsychological theory of language. Thus far little use has been made of this opportunity. But we may already state an important constraint that has emerged from this research: Given the highly selective and systematic dissociations of function observed in brain-damaged patients, we may conclude that there is a high degree of specialization of cognitive function in the brain; that is, the observations reported support a strong localizationist view of brain organization. This conclusion needs some elaboration.

We have seen that brain pathology may selectively damage one or another component of a distributed lexical system. These results support

the modular theory of lexical components presented above. They also suggest, however, that distinct neural structures subserve the hypothesized lexical components. Indeed, the evidence on hand shows a fine-grained localization of function well beyond the level of gross lexical component all the way down to single representational dimensions. This does not necessarily mean (although such may be the case) that distinct neuroanatomical loci are associated with different components of the lexicon. All that is asserted is that a distinct neural process is associated with different cognitive mechanisms and that these neural processes may be selectively damaged. What is clear, however, is that the neuropsychological data do not support an indefinitely plastic, nonlocalizationist model of neural functioning. This is a nontrivial conclusion about neural processing that has emerged from cognitive neuropsychological research.

Cognitive neuropsychological analyses may also be used to provide a fine-grained mapping of cognitive mechanisms to neural structures or processes. That is, we may be able to go beyond the level of merely specifying general constraints for a neural theory of language processing. This is not possible, however, without a profound transformation of the social organization of scientific investigation in this area.

We have seen that valid inferences about the structure of normal cognition are only possible for single-patient studies. The highly detailed investigation of single patients allows us to infer a functional lesion to a model of a cognitive system and thereby provide support for that model. Although the analysis of single patients is well-suited for drawing conclusions about cognitive structure, this methodology is not sufficient for drawing conclusions about brain/cognition relationships. For this latter purpose we need to accumulate enough cases with "identical" functional lesions in order to correlate the identified cognitive mechanisms with the neural structures that support the identified functions. This entails the accumulation of large numbers of cases. However, since the most useful, clear information is likely to come from patients with highly selective deficits and since such cases are relatively rare, it is extremely unlikely that any single investigator or laboratory will have enough cases to carry out the correlational analysis needed for this purpose. This limitation of the cognitive neuropsychological method in relating cognitive mechanisms to neural structures is not an in-principle limitation of the method but only a practical one that may be overcome if adequate measures are taken. Specifically, as I have argued elsewhere (Caramazza & Martin 1983), cognitive neuropsychologists will have to create research consortia, as have done high energy physicists and astronomers in their respective domains. This step will permit the accumulation of cases with the desired characteristics for the needed correlational analysis. (It must be empha-

sized here, if there is any need, that this proposal in no way implies an indirect justification for the group-study methodology. The frequency analysis proposed here is based on single-patient analyses and does not require the averaging of patients' performance, a methodologically invalid procedure.)

In this concluding section I have identified a procedure for relating language processing mechanisms to brain structures within the methodology of cognitive neuropsychology. We should note, however, that even in the best of all possible worlds this methodology can only lead to a fine-grained, modern phrenology—it will not provide information directly relevant to a neurophysiology of language. This latter goal may be unattainable even with technological developments. The most promising avenue open to us at this time is the development of a computational neuropsychology; that is, the development of neural network models of language processing (e.g. Arbib et al 1982). It is not difficult to imagine how the interaction of increasingly detailed, neurally constrained models of language processing that emerge from cognitive neuropsychological research with neural network models of language processes may lead to a theoretical neurophysiology of language.

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