The Handbook of

Cognitive Neuropsychology

What Deficits Reveal About the Human Mind

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The Future of Cognitive Neuropsychology

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The cognitive deficits resulting from neurological disease, injury, or abnormal development range from the prosaic (e.g., difficulty in spelling common words) to the extraordinary (e.g., loss of ability to perceive motion while other aspects of vision remain largely intact; see Hess, Baker, & Zihl, 1989; Zihl, von Cramon, & Mai, 1983). Cognitive neuropsychologists study deficits with at least three objectives in mind: (a) to gain insights into the structure and functioning of the normal cognitive system; (b) to explore the localization of cognitive functions in the brain; and (c) to achieve a better understanding of the deficits *per se*, as a basis for diagnosis and treatment. The preceding chapters describe how these aims have been pursued, and what progress has been made, in research on a variety of cognitive functions. In this concluding chapter I consider the future of cognitive neuropsychology. For each of the field's major goals I consider two questions: 1) What potential does cognitive neuropsychology hold for significant future contributions to this goal?; and 2) What developments in theory and practice will be needed to realize the potential?

GOAL 1: DEFICITS AS WINDOWS INTO NORMAL COGNITIVE MECHANISMS

As a basis for characterizing normal representations and processes, the study of cognitive deficits has impressed many observers—as crude, indirect, and fraught with interpretive difficulties (e.g., Shallice, 1988; Robertson, Knight, Rafal, & Shimamura, 1993; Seidenberg, 1988). Hence, in assessing the prospects of cognitive neuropsychology for significant future contributions to knowledge about normal cognition, it may be worthwhile to begin with a very basic question: Why study deficits when the aim is to understand normal cognitive functioning?

Why Study Deficits?

One part of an answer is that *all* of the available methods for studying human cognition—including the methods applied in research with normal participants—are indirect and subject to uncertainties of interpretation. Under these circumstances results from multiple methods,

each with different strengths and weaknesses, provide a firmer basis for conclusions than results from any single approach. Another point, which I develop further in a later section, is that cognitive neuropsychological methods, far from being crude, provide a basis for remarkably fine-grained analyses of normal cognitive systems.

However, the fundamental insight underlying cognitive neuropsychological approaches to the study of normal cognition is that complex systems often reveal their inner workings more clearly when they are malfunctioning, than when they are running smoothly. For example, when I use my laser printer, I learn little about how it represents or processes information, as long as nothing goes wrong. Recently, however, a surprisingly informative problem arose when I attempted to print a diagram for use in a class lecture. After apparently accepting input from the computer for some time, the printer signaled an error. When I pressed a 'continue' button, the page shown in Figure 24.1 Panel A emerged. The printer then appeared to accept additional input from the computer, and finally produced the page shown in Figure 24.1 Panel B.

This phenomenon may be interpreted by assuming that the printer accepts input from the computer, and stores it in a limited-capacity memory until either an entire page has been received (in which case the page is printed) or the memory is filled (in which case the printer stops accepting input and signals an error). When the 'continue' button is pressed after an error, the printer produces a page from the information in the (filled) memory, then accepts the remaining information into the now-freed memory, and finally prints this information on a new page. Thus, the printing error provides a basis for inferring some aspects of the printer's internal structure and functioning—for example, that it has a limited-capacity memory, and stores an entire page in memory before printing.

More interesting, however, is what the abnormal output implies about the printer's representation of to-be-printed information. The graphics program with which I created the diagram requires the user to treat elements such as boxes, circles, and arrows as indivisible objects. That is, these elements can be manipulated (e.g., moved, resized) only as units; the component lines or points cannot be referenced individually. In contrast, the abnormal printer output provides compelling evidence that graphics elements are represented in the printer's memory not as objects or even as components such as line segments, but rather as to-be-printed dots. This conclusion follows from the fact that some elements (e.g., the box for orthography-phonology conversion), and even their component line segments, were split across the two output pages. This phenomenon implies that some of the dots making up these elements were stored in the printer's memory before it was filled, whereas other dots could not be stored until memory was freed.

Given that the printer functions properly in printing pages with less graphical material, the error shown in Figure 24.1 also suggests that the printer's memory stores the location of each to-be-printed dot, rather than an on or off (i.e., print or don't-print) value for every possible dot position on the page. (In the latter case the memory demand for a page would be constant regardless of how many dots were to be printed.) Further, the fact that all of the graphics elements were truncated abruptly at the same place on the page implies that information about to-be-printed dots was entered column-by-column into the printer's memory, starting at the leading edge of the page, and proceeding systematically toward the trailing edge. Finally, it is evident that text is somehow treated differently from graphics, given that all of the text was printed on the first output page, including text positioned beyond the truncation point for graphics elements.

Regrettably, space does not permit discussion of the extensive additional testing carried out to confirm and extend these conclusions. Perhaps, however, this brief sketch suffices to show that the printer's abnormal output revealed aspects of its internal representations and processes that could not readily be inferred by observing its normal functioning. Similarly, the impaired performance of people with cognitive deficits can offer insights into normal cognitive

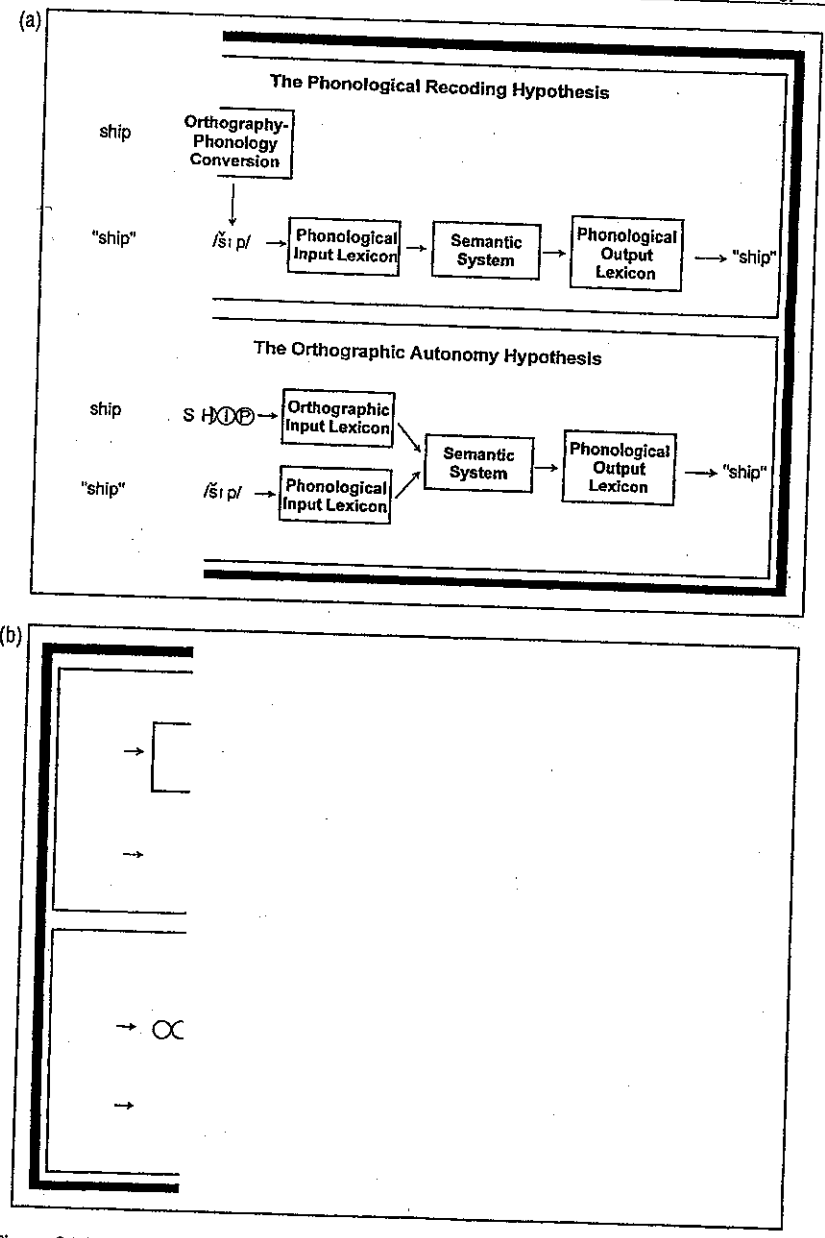


Figure 24.1: A. First page generated by laser printer. B. Second page generated by printer.

representations and processes, perhaps including insights not readily obtained through studies with normal participants.

Studying abnormalities or malfunctions to shed light on normal structures and processes is a well-established research strategy in a variety of scientific domains. For example, research on AIDS has contributed to knowledge of normal immune-system functioning; mutations are studied in part to advance our understanding of normal genetic mechanisms; and observations of damaged ecosystems offer insights into normal ecological processes. More generally, scientists of many stripes study unusual phenomena as a means of gaining insight into more typical conditions or events. For example, studies of supernovae illuminate the processes and products of nuclear fusion in stars; and research on volcanic eruptions offers insights into materials and events in the earth's interior.

Cognitive Deficits as Experiments of Nature

Potentially informative abnormalities or extraordinary phenomena can sometimes be created in the laboratory, as when brain lesions are produced surgically in neuroscience research with laboratory animals, or collisions of subatomic particles are engineered in particle accelerators. Often, however, it is beyond the researcher's power to create the preparations of interest. In such circumstances the study of naturally-occurring phenomena–experiments of nature–has proved to be a powerful tool, providing evidence not obtainable through more usual research methods. Studies of supernovae, volcanic eruptions, and human genetic abnormalities are cases in point.

Human cognitive neuropsychology also falls into this category. Obviously, it is neither possible nor desirable to make experimental brain lesions in humans. Rather, cognitive neuropsychological research takes advantage of the unfortunate natural experiments in which the brain sustains damage through accident or disease, or fails to develop normally.

Like other research with experiments of nature, cognitive neuropsychological research has its disadvantages. For example, the inability to create at will the deficits of interest is an inconvenience at best, and sometimes a serious impediment. (This point does not imply, however, that cognitive neuropsychological research is inherently observational or correlational; rigorous experimental methods can be, and are, applied in studies of cognitive deficits. The researcher's lack of control has to do with the creation of deficits, and not with the investigation of these deficits.)

Balanced against the disadvantages are some significant advantages. One I have already mentioned: Cognitive deficits often reveal in a clear and compelling fashion aspects of normal representation and processing not readily apparent from normal cognitive functioning. Another advantage stems from the inherently opportunistic nature of cognitive neuropsychological research. The cognitive neuropsychologist typically begins by screening individuals with deficits potentially relevant to issues of interest, and then explores in detail those deficits that seem to hold promise for illuminating the issues. This approach looks to nature for clues, and then follows the clues wherever they may lead. Relative to research with normal participants–which typically approaches nature with specific predetermined questions–cognitive neuropsychological research may therefore offer greater opportunity to be surprised by the unexpected. Indeed, cognitive neuropsychology has a long history of turning up remarkable phenomena (e.g., unilateral spatial neglect, blindsight, category-specific deficits, anterograde amnesias) that raise new questions, suggest novel theoretical perspectives, and give rise to productive lines of research (although not always immediately; see, e.g., Zeki, 1993, pp. 33–40). (For further discussion of the advantages and disadvantages of cognitive neuropsychological research see Caramazza, 1986, 1992; Ellis, 1987; Kosslyn & Intriligator, 1992; Kosslyn & Van Kleek, 1990; McCloskey, 1993; Robertson et al., 1993.)

Facilitating Future Progress

Science often moves forward through technological advances, such as the invention of the telescope, or the development of radiometric techniques for dating geological strata and artifacts. Cognitive neuropsychology is no exception, having benefited in recent years from advances in the computing technology available for data collection and analysis, and from the advent of structural and functional brain imaging technologies. At least in the near future, however, developments on a more abstract or conceptual level may be of greater importance for maximizing the field's contribution to knowledge about normal cognition. In this section I consider three potential developments, discussing the first two briefly and the third in greater detail.

Integrating Cognitive Neuropsychology and 'Normal' Cognitive Psychology

One pressing need is for better integration of research using deficits to study normal cognition, and research using normal performance for the same purposes. These two approaches differ only in (some aspects of) methodology, having in common not only the aim of understanding normal cognition and the conception of the mind as a representational/computational system, but also most of the specific theoretical questions under investigation. Nevertheless, studies of cognitively-impaired participants and studies of normal participants are typically conducted by different researchers, presented at different professional meetings, and published in different journals. This segregation by methodology is counterproductive, resulting not only in missed opportunities for fruitful interchange and collaboration, but also in failures of researchers adopting one approach to understand (or even know of) relevant evidence obtained through the other methodology. Some progress has been made toward increased communication between research communities, but much remains to be done.

Strengthening Cognitive Neuropsychological Research

Although much of the research in cognitive neuropsychology is of high quality, some theoretical and methodological shortcomings are relatively common. Addressing these weaknesses would almost certainly increase the rate of progress. One development that would prove beneficial is more explicit articulation of theoretical claims. In current research the theoretical concepts and hypotheses are sometimes so vague that the substance of proposals is difficult to ascertain or evaluate. (I hasten to add that this point applies not only to cognitive neuropsychology, but also, and with equal force, to research with unimpaired participants.)

Cognitive neuropsychological research could also benefit from more thorough and theoretically-grounded testing of patients. Pinning down the nature of a patient's deficit, and exploring the implications of the deficit for issues of normal cognition, often require extensive testing with a substantial number of carefully-chosen tasks. In current research the amounts and types of testing often are not sufficient to provide a firm foundation for inferences.

Finally, researchers need to ensure that the procedures they adopt are suited to the goals of their research. Caramazza and others have argued that the widespread patient-group method, in which data are averaged or otherwise aggregated over the patients in a group, is fundamentally unsound, at least for addressing issues of normal cognition (e.g., Badecker & Caramazza, 1985; Caramazza, 1984, 1986; Caramazza & Badecker, 1989, 1991; Caramazza & McCloskey, 1988, 1991; Ellis, 1987; McCloskey, 1993; McCloskey & Caramazza, 1988, 1991). I will not attempt to recount the arguments in detail. However, the central contentions are (a) that aggregating data over patients requires the assumption that the patients are homogeneous with respect to the nature of their deficits, but (b) that regardless of how patients are selected,

homogeneity of deficits cannot be assumed *a priori* (and indeed is unlikely when deficits are characterized at the levels of detail required for addressing issues of current interest in the study of normal cognition). Caramazza and colleagues argue that although results from multiple patients can and should be considered when addressing issues of normal cognition, the data from each patient must be treated individually. (I will have more to say about this *multiple single-patient study* method in the next section.) These arguments, although to my mind compelling, are controversial; at the least, however, it is clear that researchers should give careful thought to the assumptions underlying their chosen methods, and whether these assumptions are warranted.

Shedding a Conceptual Straightjacket

Many cognitive neuropsychologists, as well as most other cognitive scientists, assume that cognitive deficits can be brought to bear on issues of normal cognition only by applying that I will call *task dissociation logic*. From this perspective, cognitive neuropsychology is a method for drawing inferences of the form, "Task A and Task B involve different processing mechanisms." The method is applied by testing patients on two or more tasks, and interpreting the observed dissociations and associations of deficits according to the following principles.

(1) Single Dissociations. A finding of poor (i.e., impaired) performance on one task with good (i.e., normal, or at least reliably better) performance on the other suggests that the tasks differ in one or more of their underlying processing mechanisms. However, this pattern of results—a single dissociation—does not constitute compelling evidence for different processing mechanisms; another possible interpretation is that both tasks require the same processing mechanisms, but one task demands more from these mechanisms than the other, and consequently shows greater impairment when the mechanisms are damaged.

(2) Double Dissociations. Because single dissociations are subject to this potential *resource artifact* interpretation (Shallice, 1988), stronger evidence is needed to warrant firm conclusions in favor of differences between tasks in underlying processing mechanisms. In particular, what is required is a double dissociation, in which one or more patients show poor performance on Task A with good performance on Task B, while one or more other patients show good performance on Task A with poor performance on Task B. A double dissociation rules out resource artifact interpretations. To interpret the A-good/B-poor dissociation in terms of resource artifacts one would have to assume that Task B places heavier demands than Task A on the required processing mechanisms; but to interpret the complementary A-poor/B-good dissociation one would have to make the contradictory assumption that Task A demands more than Task B from the mechanisms. Double dissociations are therefore the gold standard in cognitive neuropsychological research. (Note, however, that a double dissociation implies only that there is some difference in processing mechanisms between the tasks, and does not by itself say anything about the nature of the mechanisms or how they differ between tasks.)

(3) Associations. Consider finally a pattern of results in which performance is impaired on both Task A and Task B. This pattern—an *association*—might seem to suggest that the tasks share one or more processing mechanisms, given that damage to a shared mechanism would be expected to produce impairment on both tasks. However, the association could also have resulted from two separate deficits, one affecting Task A and the other affecting Task B. As a consequence, associations are uninformative about underlying processing mechanisms.

Task dissociation logic has been widely discussed in the cognitive neuropsychology literature. Some discussions have been aimed at formulating the logic more precisely, addressing

such matters as what specific forms of double dissociation are required to warrant conclusions about separate processing mechanisms (e.g., Jones, 1983; Shallice, 1988). Other commentators have criticized the logic or some of its applications (e.g., Plaut, 1995; Robertson et al., 1993), arguing for example that double dissociations do not necessarily imply a difference in processing mechanisms between tasks (e.g., Chater & Ganis, 1991; Ganis & Chater, 1991; Plaut, 1995; but see Bullinaria & Chater, 1995). Virtually all of the discussions have taken as given that task dissociation logic represents the sole method for relating cognitive deficits to theories of normal cognition. Proponents of the logic have offered their analyses as prescriptions of how to do cognitive neuropsychological research, and opponents have presented their arguments as indictments of cognitive neuropsychology in general.

Although task dissociation logic has clearly played an important role in cognitive neuropsychological research, the tacit assumption that this logic represents the only way of doing cognitive neuropsychology has imposed a conceptual and methodological straightjacket on the field. The nature and source of the self-imposed constrictions become apparent if we examine the presuppositions of the logic.

Presuppositions of Task Dissociation Logic. Task dissociation logic rests upon two—usually implicit—assumptions. The first is that the data obtainable from studies of cognitive deficits are necessarily crude (e.g., Shallice, 1988), and in particular are limited to gross level-of-performance measures (e.g., percent correct) on a small number of tasks. The second assumption is that interpretation of evidence is a fixed mechanical process in which the logical consequences of the data are assessed in isolation from any other empirical or theoretical considerations.

Given these presuppositions, it appears self-evident that cognitive neuropsychology is limited to conclusions of the form, "Task A and Task B differ in at least one processing component," and that these conclusions can only be drawn by applying the principles of task dissociation logic. However, the presuppositions have no basis in canons of scientific method, practice in other areas of cognitive science, or intrinsic characteristics of cognitive deficits. Rather, these self-imposed strictures apparently stem primarily from a failure of imagination on the part of cognitive neuropsychologists. Fortunately, a broader conception of cognitive neuropsychology is emerging.

Cognitive Neuropsychology More Broadly Conceived

A growing body of research demonstrates that the impaired performance of individuals with cognitive deficits is often richly patterned, and that analysis of the patterning can provide a basis for specific, fine-grained inferences about cognitive representations and processes. For example, several recent studies of individual dysgraphic patients converge on conclusions about the orthographic representations underlying the ability to spell words in writing, typing, spelling aloud, and so forth. These studies provide evidence that orthographic representations are not simple linear sequences of letter tokens (e.g., C-R-O-S-S for the word *cross*), but rather are complex multidimensional structures in which (a) orthographic syllable structure is represented (Caramazza & Miceli, 1990); (b) information about the orthographic consonant/vowel status of each letter is represented independently of the letter's identity (Caramazza & Miceli, 1990; Cubelli, 1991; McCloskey, Badecker, Goodman-Schulman, & Aliminosa, 1994); and (c) information about letter doubling is specified separately from the identity of the doubled letter (Caramazza & Miceli, 1990; McCloskey et al., 1994; Tainturier & Caramazza, 1996; Venneri & Cubelli, 1993). The evidence concerning representation of letter doubling is illustrative.

Patient HE (McCloskey et al., 1994) presented with several systematic phenomena in spelling words with double letters, including errors such as the following:

Stimulus	*Response*
shell	sheel
needle	neddle
across	accros
confess	connfes
parrot	parott

In these *doubling shift* errors the letter that should have been doubled appeared in the response as a single letter, and another letter was doubled instead. Several analyses of HE's errors demonstrated that the occurrence of an erroneous doubling was systematically related to the presence of a double letter in the stimulus; for example, errors like *shell* → *sheel* were not simple letter substitutions (e.g., *l* → *e*) that happened by chance to replace one letter of a double-letter pair with a letter that created another double. More generally, HE's doubling shift errors, and other aspects of his performance, were inconsistent with the assumption that double letters are specified in orthographic representations simply by two tokens of the to-be-doubled letter (e.g., S-H-E-L-L). McCloskey et al. (1994) argued that the results could be interpreted only by assuming that double letters are represented by a single letter token associated with a separate specification of doubling.

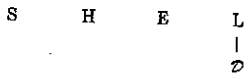
Patient LB (Caramazza & Miceli, 1990) also exhibited a systematic pattern of errors in spelling words with double letters. Among the more striking aspects of this pattern were errors such as the following:

Stimulus	*Response*
pezzo	zeppo
cellula	leccula
blocco	bcollo
passai	sappai

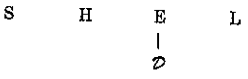
These *doubling exchange* errors are extremely difficult to reconcile with representations in which double letters are specified by two tokens of the to-be-doubled letter (e.g., P-E-Z-Z-O). For example, in the *pezzo* → *zeppo* error how could two letter tokens (Z-Z) change places with a single letter token (P) in such a way that the single token becomes two (P-P) and the two tokens become one (Z)? On the basis of the doubling exchange errors and other phenomena—for example, *double substitution* errors such as *marrone* → *mazzone*—Caramazza and Miceli (1990) concluded that orthographic representations specify double letters by a single letter token plus doubling information.

Results from other patients also suggest that double-letter representations are in some sense special. Venneri and Cubelli (1993) found that patient EZ, in spelling words with double letters, omitted one of the letters in the double over 80% of the time (e.g., *Dalla Villa* → *Dala Vila*). Omissions were far less frequent for letters in other types of clusters, including digraphs corresponding to a single phoneme (e.g., *sc* in *pesce*). Also, Tainturier and Caramazza (1996) reported several interesting double-letter phenomena exhibited by a patient, FM, whose spelling responses were often grossly incorrect (e.g., fate → *frich*). For example, in spelling to dictation FM made no errors that "split" a double-letter sequence (e.g., *pull* → *plul*). In contrast, control letter sequences matched to the double-letter sequences were frequently split (e.g., *rn* in *barn* → *bron*). Tainturier and Caramazza (1996) interpreted this finding as further evidence that double letters are not represented by two tokens of the to-be-doubled letter (e.g., P-U-L-L).

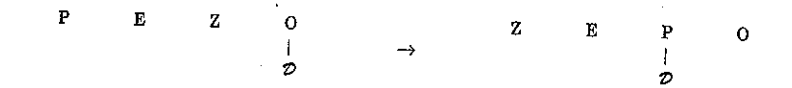
Two specific hypotheses have been proposed concerning the form of the doubling representations (Caramazza & Miceli, 1990; McCloskey et al., 1994). For simplicity, I will describe only Caramazza and Miceli's (1990) *doubling mark* hypothesis, which assumes that double letters are represented by a single letter token associated with a doubling marker, as illustrated below for the word *shell*:



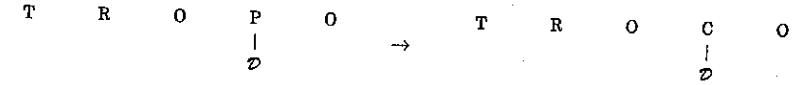
This hypothesis about normal orthographic representations provides a basis for interpreting the various observed patterns of impairment. HE's doubling shift errors (e.g., *shell* → *sheel*) may be explained by assuming that his orthographic representations were sometimes disrupted (during retrieval from an orthographic lexicon or subsequent retention in a buffer memory) in such a way that the doubling mark became associated with the wrong letter token:



LB's double exchanges (e.g., *pezzo* → *zeppo*) can also be interpreted, by assuming that the single token of the to-be-doubled letter was exchanged with the token for another letter in the word:

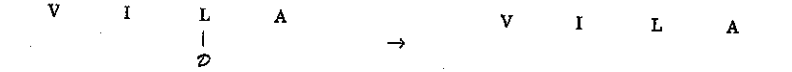


Similarly, LB's double substitution errors (e.g., *troppo* → *trocco*) can be attributed to substitution of an incorrect letter token for the single token of the to-be-doubled letter:



(As expected from these interpretations LB also made letter substitution and exchange errors not involving double letters.)

Patient EZ's frequent deletion of one letter in a double letter pair (e.g., *villa* → *vila*) may be interpreted by assuming that she was impaired in retaining the doubling mark during the spelling process:



Finally, the absence of split-double errors (e.g., *pull* → *plul*) in patient FM's error corpus is straightforwardly explained by the assumption that orthographic representations of double letters include only one token of the to-be-doubled letter, and not two tokens that could become separated.

Taken together, the results from these studies make a compelling case against the assumption that orthographic representations specify double letters by two tokens of the to-be-doubled letter, and argue strongly in favor of the hypothesis that double-letter representations involve a single letter token associated with a separate specification of doubling. This conclusion forms one pillar of the broader argument that orthographic representations are complex multi-dimensional structures, rather than simple linear sequences of letter tokens.

I have developed this example in some detail to illustrate several points. First, in cognitive neuropsychological research addressing issues of normal cognition the evidence need not be

limited to dissociations between tasks in gross performance-level measures, and the inferences drawn from the evidence need not be limited to coarse-grained conclusions of the form, "Task A and Task B involve different processing mechanisms." The impaired performance of individuals with cognitive deficits is often richly structured, and careful analysis of the structure can provide a basis for fine-grained conclusions about cognitive processes and the representations upon which they operate.

Detailed analyses of deficits can be especially powerful when, as in the present example, results from several individual patients can be brought to bear on the theoretical issues of interest. In this multiple single-patient study method the goal is to interpret results from all of the patients in terms of the same assumptions about normal cognitive mechanisms, by specifying for each patient a form of damage that would lead to the particular pattern of impairment observed for that patient. Note that this research strategy is not aimed at finding multiple patients with exactly the same type of deficit. Cognitive deficits, when characterized at levels of detail commensurate with fine-grained conclusions about normal representations and processes, prove to be not only richly-structured but also diverse, and it is unusual to find two or more patients whose performance is the same in all potentially-relevant respects. The multiple single-patient study method acknowledges and in fact exploits this diversity, requiring that a theory be capable of accounting for each of the various forms of impairment that may result from damage to the cognitive mechanisms under investigation.

The double-letter example also illustrates the point that interpretation of evidence need not—and indeed should not—involve the mechanical application of task-dissociation logic. In the first place many forms of potentially-relevant data (e.g., the error patterns exhibited by HE and LB) do not fit neatly into any of the evidential categories defined by this logic (i.e., single dissociation, double dissociation, association). Even for results straightforwardly describable as dissociations or associations, the principles of task dissociation logic are usually not an appropriate guide to interpretation; these principles are valid only when the data are limited to gross performance-level measures, and are interpreted in a theoretical vacuum. For example, the principle that associations do not provide evidence of shared processing mechanisms may have merit in the context of results consisting solely of poorer-than-normal performance on two tasks, because such an association might plausibly have resulted from two separate deficits, each affecting one of the tasks. However, when an association involves a close correspondence across tasks in a specific and richly articulated performance pattern—such as the finding (McCloskey et al., 1994) that patient H.E.'s written spelling and oral spelling showed virtually identical accuracy, effects of variables (e.g., word frequency), and distribution of errors across error types—it strains credibility to suggest that the association reflects the accidental co-occurrence of two separate deficits. Therefore, such associations may properly be taken as evidence of shared processing mechanisms. (See Hillis, Rapp, Romani, & Caramazza, 1990, for another example.)

Similarly misconceived are the task dissociation principles for interpreting dissociations (i.e., single dissociations are weak evidence, and double dissociations are required before firm conclusions can be drawn). To be sure, a double dissociation usually constitutes stronger evidence than one of the constituent single dissociations, simply because the former represents more evidence than the latter. However, given a single dissociation, it is by no means uniformly true either that the single dissociation is weak evidence, or that the most valuable piece of additional evidence is the complementary dissociation that completes the double dissociation (Caramazza, 1986). The task-dissociation principles may apply when (a) the issue under consideration is whether two tasks involve different processing mechanisms, and (b) the single dissociation could plausibly be attributed to a resource artifact (i.e., one task demanding more than the other from the same processing mechanisms). Under such circumstances the complementary dissociation serves the important function of ruling out the resource artifact interpretation, and therefore greatly strengthens the case for separate processing mechanisms.

However, dissociations may be brought to bear on issues other than those concerning shared versus separate processing mechanisms (e.g., issues concerning the form of representations), in which case the resource artifact issue may not even arise. Furthermore, even when the focus is on shared versus separate mechanisms a resource artifact interpretation for a single dissociation may not be plausible, or even possible, when account is taken of the particular form of the dissociation, the particular cognitive processes under investigation, the particular hypotheses being entertained about these processes, and so forth (see Caramazza, 1986; Caramazza & McCloskey, 1991; McCloskey, 1993; Sokol & McCloskey, 1988). For example, Rapp, Benzing, and Caramazza (1997) reported a single dissociation in which patient PW, on some trials of a picture naming task, made semantic errors in naming the picture orally, yet wrote the name correctly. Thus, shown a picture of an owl and asked to say and then write the name several times in succession, PW responded as follows:

Spoken: "turtle"
Written: OWL
Spoken: "turtle"
Written: OWL
Spoken: "turtle"
Written: OWL

Arguing from other aspects of PW's performance that the oral-naming errors reflected an impairment in accessing phonological word representations, Rapp et al. (1997) interpreted the dissociation between written and spoken naming as evidence against the *phonological mediation* hypothesis, which assumes that the orthographic representations underlying written word production can be accessed only via phonological representations (i.e., semantics phonology orthography). Rapp et al. instead endorsed an *orthographic autonomy* hypothesis, according to which orthographic representations may be accessed directly from semantic representations, without phonological mediation.

According to task dissociation logic, we should consider Rapp et al.'s (1997) argument questionable on grounds that their single dissociation might have resulted not from a difference in processing mechanisms between spoken and written naming, but rather from a resource artifact (i.e., spoken naming demanding more than written naming from the same processing mechanisms). Further, we should believe that the Rapp et al. conclusion would be buttressed by results providing the other half of the double dissociation (i.e., good oral but poor written naming). However, this application of task dissociation logic would be seriously misguided. To attribute the Rapp et al. (1997) findings to a resource artifact one would have to assume that retrieval of phonological word representations is a more demanding process in the spoken naming task than in written naming task. However, such an assumption would be not only highly unmotivated but also highly implausible, especially given that the principal alternative to Rapp et al.'s interpretation—the phonological mediation hypothesis—would presumably hold that the phonological retrieval process is identical in spoken and written naming.

Furthermore, results providing the other half of the double dissociation would do very little to strengthen the Rapp et al. (1997) argument. Suppose we had results from one or more patients showing impaired written naming in the presence of intact spoken naming, and more specifically impaired access to orthographic representations in the presence of normal access to phonological representations. Although these results would certainly be consistent with Rapp et al.'s orthographic autonomy hypothesis, they would be equally consistent with the phonological mediation hypothesis, and indeed with virtually any hypothesis positing that access to orthographic representations is implicated in written naming but not in spoken naming. Hence, the findings would add little to the support for the orthographic autonomy hypothesis, and would be far less useful than additional evidence suggesting specifically that access to phonological representations is not required for access to orthographic representa-

tions (e.g., results from oral and written sentence completion similar to those reported by Rapp et al. for oral and written picture naming).[1]

This example drives home the point that contrary to the assumptions of task dissociation logic, the weight of various categories of cognitive neuropsychological evidence need not, and should not, be assessed in a vacuum. Like other forms of evidence in cognitive science (or indeed in any science) cognitive neuropsychological data can and should be evaluated in the light of such considerations as the particular theoretical issues under investigation, the tenable alternative positions on these issues, and the specific form of the evidence. Once this point is recognized it becomes clear that circumscribing *a priori* the forms of data to be considered, or the reasoning to be used in linking data with theory, is neither possible nor desirable.

GOAL 2: FUNCTIONAL LOCALIZATION

Much of our knowledge about instantiation of cognitive functions in the brain has come from research with brain-damaged patients. The study of lesion-deficit correlations has played a central role both in establishing that the brain exhibits functional specialization (e.g., Finger, 1994; Kertesz, 1994), and in linking particular cognitive functions to particular brain regions. Aphasia research, beginning with Broca and Wernicke, has taught us most of what we know about brain specialization for language; studies of organic amnesias–notably, patient HM's profound anterograde amnesia after bilateral temporal lobectomy (Scoville & Milner, 1957)–have established the importance of the hippocampus and other brain regions for memory; observations of patients with parietal lesions contributed to the recognition that this brain area is implicated in spatial processing; and so forth and so on. (For further discussion see, e.g., Banich, 1997; Farah & Aguirre, 1999; Kolb & Whishaw, 1996; McCarthy & Warrington, 1990.)

However, with the advent of functional neuroimaging comes the question of whether lesion-deficit correlation research has outlived its usefulness. Indeed, functional neuroimagers are often at pains to point out the disadvantages of the lesion-deficit correlation method relative to their newer techniques, noting that structural brain lesions are often quite large, that lesions may not respect functional boundaries in the brain, and that the possibility of functional reorganization complicates the interpretation of lesion-deficit relationships.

Functional neuroimaging methods are, without doubt, extraordinarily useful tools for probing brain localization of cognitive functions, offering improved spatial resolution and the ability to study normal participants. However, it would be a mistake to conclude that the lesion-deficit correlation method is no longer useful. The reasons are analogous to those I cited in discussing cognitive neuropsychological research that addresses issues of normal cognition. First, as long as all of the available methods are imperfect–and this is certainly true of current functional localization methods–results from multiple methods will almost always provide a firmer basis for conclusions than results from a single method.

Second, lesion-deficit correlation methods are not limited to establishing crude brain-cognition relationships (e.g., the parietal lobes are implicated in spatial cognition). Consider, for example, the multiple single-patient study of visual field defects carried out many years ago by Gordon Holmes and W. T. Lister. Holmes, a neurologist, and Lister, an ophthalmic surgeon, were officers in the British army during World War I. Stationed at a base hospital in France, they treated thousands of soldiers with head wounds. In 1916 Holmes and Lister published a study of relationships between visual field defects and brain lesion loci in 23 patients with

[1]It is interesting to note that in some sense PW himself provided the other half of the double dissociation. In addition to the trials in which he responded to a picture with an incorrect spoken name but a correct written name, Rapp et al. (1997) also observed trials in which PW's spoken response was correct but his written response was erroneous. These latter trials, like results from other patients showing good spoken but poor written naming, contribute little if anything to the strength of the conclusions Rapp et al. drew in favor of the orthographic autonomy hypothesis.

occipital lobe damage resulting from "penetrating and perforating wounds of the cranium by rifle bullets, shell fragments, and shrapnel, as well as local concussions and depressed fractures" (Holmes & Lister, 1916, p. 38). The x-ray techniques available at the time were of little use in delineating the location or extent of brain damage. Although in a few cases Holmes and Lister were able to observe a patient's brain during surgery, they usually had to infer the lesion locus from the location of entrance and (in some cases) exit wounds, interpreted in light of their knowledge about skull/brain relationships. Assessment of visual fields was also crude; most patients were confined to bed, and were tested with a small hand perimeter.

Despite these difficult conditions Holmes and Lister (1916) were able to establish systematic relationships between lesion loci and visual field defects; for example, patients with damage to the upper bank of the calcarine fissure consistently showed a lower quadrantanopia in the contralesional visual field. On the basis of their observations Holmes and Lister drew the following conclusions:

> The upper half of each retina is represented in the dorsal, and the lower in the ventral part of each visual area. (p. 72)
>
> The centre for macular or central vision lies in the posterior extremities of the visual areas, probably on the margins and the lateral surfaces of the occipital poles. (p. 72)
>
> The centre for vision subserved by the periphery of the retinae is probably situated in the anterior end of the visual area, and the serial concentric zones of the retina from the macula to the periphery are probably represented in this order from behind forwards in the visual area. (p. 73)

Finally, speaking to an issue that had aroused some controversy, Holmes and Lister (1916) concluded that "in common with every other part of the retina, the macula is not represented bilaterally" (p. 71). These conclusions have been confirmed many times over in subsequent studies with more sophisticated methods. Clearly, fine-grained conclusions are possible in multiple single-patient studies of lesion-deficit correlations.

A third reason for continuing to apply the lesion-deficit correlation approach is that relative to functional neuroimaging methods this approach has advantages as well as disadvantages. In particular, interpretation of lesion-deficit correlations is in some respects more straightforward than interpretation of data from functional neuroimaging methods. I refer here not to the uncertainties about relationships between brain activity and the variables assessed in functional neuroimaging (e.g., blood oxygenation), or to the complex statistical issues arising in the analysis of imaging data; rather my point has to do with differences between activation and damage as bases for inferring a brain region's involvement in a cognitive process. Results showing a consistent relationship between a particular cognitive process and activation of a particular brain area may suggest that the area is involved in carrying out the process. However, given our current state of knowledge about the brain, the possibility remains open that brain areas playing no functional role in a cognitive process may become activated when the process is executed (Farah & Aguirre, 1999). Consider, for example, results suggesting that V1 and even LGN (the lateral geniculate nucleus) are activated when subjects engage in visual imagery (e.g., Chen et al., 1998; Kosslyn et al., 1993). These results may indicate that V1 and LGN play functional roles in imagery; on the other hand it is also possible that these areas become activated during imagery (perhaps via feedback connections from higher visual areas) while playing no functional role. In contrast, results showing a consistent relationship between damage to a particular brain area and disruption of a particular cognitive process argue strongly that the area is required for execution of the process.

Also, whereas current functional neuroimaging methods typically impose significant restrictions on testing time and methods, lesion-deficit correlation studies usually allow far more extensive testing with fewer methodological restrictions. This advantage is significant. Isolating a cognitive process of interest is not trivial, and much of the functional neuroimaging

research conducted to date is weak in this respect. That is, the behavioral data collected in many of the studies are not sufficient to permit clear conclusions about the cognitive processes associated with the activated brain regions.

I do not intend to suggest that lesion-deficit correlation methods are superior to functional neuroimaging methods; as mentioned above, the lesion-deficit approach has its own disadvantages. My point is simply that functional neuroimaging and lesion-deficit correlation approaches should be viewed not as competitors, but as complementary approaches to localization issues.

Facilitating Future Progress

At least two developments might contribute to progress in lesion-deficit correlation research: finer-grained characterization of lesions and deficits, and more widespread application of reversible lesion techniques.

Finer-Grained Lesion and Deficit Characterizations

Most lesion-deficit correlation studies have relied upon coarse-grained descriptions of brain lesions (e.g., left posterior, right parietal), and/or crude characterizations of cognitive deficits (e.g., dysgraphia, constructional apraxia) that refer to affected behaviors or tasks rather than underlying cognitive processes. However, current structural imaging methods allow more precise localization of brain lesions; and the theoretical and methodological tools of cognitive neuropsychology allow specific, theoretically-grounded characterizations of cognitive deficits. Exploiting these capabilities in lesion-deficit correlation studies should enhance our understanding of how and to what extent specific cognitive processes are realized by specific brain mechanisms.

Reversible Lesion Techniques

Recently-developed techniques for creating temporary local disruption of brain function also hold promise for fruitful applications of the lesion-deficit correlation approach. In one such technique grids of electrodes are surgically placed on the surface of the cortex (for clinical diagnostic purposes), and left in place for periods ranging from a few days to a few weeks. Mild electrical stimulation applied via pairs of adjacent electrodes creates a temporary disruption of cortical function in the neighborhood of the electrode pair, and cognitive testing is undertaken during stimulation to assess effects of the disruption. The cortical grid technique has produced several intriguing results showing specific relationships between stimulation sites and cognitive impairments (e.g., Boatman, Hall, Goldstein, Lesser, & Gordon,1997; Boatman, Lesser, & Gordon, 1995; Hart, Lesser, & Gordon, 1992) For obvious reasons, however, this technique is unlikely to see widespread application in research.

Transcranial magnetic stimulation (TMS) provides a less invasive alternative. In this technique a temporary local disruption of cortical function is created by applying a brief but strong magnetic field at the scalp to induce an electric current in the underlying cortex. Unlike the grid technique, TMS can be used (with appropriate safeguards; see Wasserman, 1998) in studies involving normal participants. Although relatively new, the TMS technique has already been applied in a large number of studies on perception, attention, memory, language, and motor control. Some researchers consider the method especially useful for probing the time course of cognitive processing. (See Walsh & Rushworth, 1999, for a general introduction.)

A significant advantage of reversible lesion techniques is that participants can serve as their own controls. In studies of patients with structural lesions it may be difficult to determine whether a patient's performance reflects the effects of the lesion, or whether instead the patient would have shown comparable performance even prior to brain damage. However, in a reversible lesion study participants can be tested both with and without stimulation, yielding performance measures reflecting both the normal and the disrupted functioning of the affected brain region. However, reversible lesion methods also have disadvantages, including uncertainties about the extent to which effects of stimulation are localized to the targeted brain regions, and limits on trial durations and total amounts of testing under stimulation.

GOAL 3: UNDERSTANDING DEFICITS

Thus far I have considered the study of cognitive deficits for purposes of characterizing normal cognitive mechanisms, and exploring brain localization of cognitive functions. The third major objective of cognitive neuropsychology is to achieve a better understanding of the cognitive deficits themselves, among other reasons to provide a basis for improved diagnosis and treatment.

For most purposes deficits are best characterized in terms of damage to, or abnormal development of, normal cognitive mechanisms. For example, in diagnosing reading deficits for purposes of selecting appropriate treatment strategies, one needs to determine which aspects of the normal reading process are disrupted (e.g., visual processing of stimulus words, syntactic analysis, retrieval of lexical-semantic representations, or so forth), and the nature of the disruption(s).

Traditionally, characterization of deficits has involved deficit categories and diagnostic tests not grounded in well-articulated theories of normal cognition. In some instances deficit types have been defined primarily on the basis of clinical observations suggesting that certain symptoms often co-occur (e.g., Broca's aphasia, defined by a system complex including reasonably preserved comprehension, non-fluent speech, and impaired repetition). In other instances the deficit categories have been grounded in very coarse-grained analyses of normal cognitive functions (e.g., receptive and expressive aphasia, based on the analysis of language processing into comprehension and production components). Diagnostic assessment has typically involved administering a few brief tasks chosen to reveal the critical symptoms or assess the status of the grossly-defined cognitive functions.

This approach to defining and diagnosing deficits typically yields heterogeneous deficit categories, with the individuals sorted into a category differing widely in the nature of their underlying dysfunctions (e.g., Badecker & Caramazza, 1985; Miceli & Silveri, 1989). Categories of this sort do not provide an adequate foundation for research on, or implementation of, treatment strategies. For example, research on remediation of developmental reading disorders has been hampered by reliance on the coarse, pretheoretical category of developmental dyslexia. Whereas this category is almost certainly heterogeneous, most developmental dyslexia research implicitly assumes that the underlying cognitive dysfunction is the same in all (or at least most) dyslexics (Martin, 1995; McCloskey & Rapp, 2000). As a consequence, most remediation studies have examined undifferentiated groups of dyslexic individuals, and have been aimed at formulating a single set of methods for across-the-board application. Among the results of this approach are disappointing success rates, and widespread failures to replicate. (See Martin, 1995, for more detailed discussion.)

Crude pretheoretical classifications of deficits are also a serious impediment in research aimed at uncovering genetic, neuropathological, or other bases for deficits. Here again research on developmental dyslexia is an example: Clarification of genetic and neurological bases for developmental reading deficits is unlikely to be forthcoming as long as most researchers assume that dyslexia is a unitary disorder.

Fortunately, recent years have seen a growing recognition that methods for diagnosis and treatment of cognitive deficits should be grounded in specific theoretical assumptions about normal cognitive representations and processes (e.g., Bishop & Byng, 1984; Byng, 1988; Byng & Black, 1995; Caramazza & Hillis, 1993; Castles & Coltheart, 1993; Davis & Coltheart, 1999;

Hillis, 1993, 1998; Kay, Lesser, & Coltheart, 1996; Margolin, 1992; McCloskey, Aliminosa, & Macaruso, 1991; Mitchum & Berndt, 1995; Mitchum, Haendiges, & Berndt, 1993; Nickels, 1995; Nickels, Byng, & Black, 1991). The resulting work has produced some useful diagnostic instruments (e.g., Kay et al., 1996), as well as some potentially promising treatment outcomes (e.g., Davis & Coltheart, 1999; Mitchum, Haendiges, & Berndt, 1995; Nickels, 1995).

Facilitating Future Progress

The increased emphasis on cognitive theory in research concerning diagnosis and remediation of cognitive deficits is encouraging. However, at least two additional developments will be needed if the research is to have a significant impact on clinical practice.

Balancing the Ideal and the Practical in Diagnosis

Theory-based assessment of cognitive deficits can be extremely time-consuming and labor-intensive when carried out at a sufficiently fine grain to be useful in targeting remediation efforts. At present, ascertaining which cognitive mechanisms are intact and which are impaired, and clarifying the nature of the impairment(s), often requires far more testing—perhaps even an order of magnitude more—than is feasible on a large scale in clinical settings. Accordingly, if theory-based assessment methods are to have broad application, ways will have to be found to streamline the process without major loss of detail or accuracy.

Theory of Remediation

Practical theory-based assessment methods, although probably necessary for effective treatment, are certainly not sufficient; knowing what is wrong is not the same as knowing how to fix it. At least one other element is crucial: a theory of remediation (e.g., Byng & Black, 1995; Caramazza & Hillis, 1993; Mitchum & Berndt, 1995). A remediation theory, which might have both cognitive and neurophysiological elements, would address such questions as, What sorts of changes to a cognitive system are and are not possible after the system has been damaged in various ways?; and, How can potentially-beneficial changes be brought about? Theoretical treatment of these questions would be a major step toward transforming the treatment of cognitive deficits from an art to a science.

Conclusion

Cognitive neuropsychology has made, and continues to make, major contributions to the study of normal cognition; indeed, some of the most exciting results in cognitive science are coming from studies of deficits. Furthermore, the lesion-deficit correlation method has been our major source of knowledge about localization of cognitive processes in the brain, and should continue to be valuable as a complement to the newer functional neuroimaging methods. Finally, cognitive neuropsychological research has advanced our understanding of cognitive deficits, and holds promise for contributing to the development of treatments. If theory and method continue to develop along the lines discussed in this chapter, the future of cognitive neuropsychology should be bright. The potential of the approach is just beginning to be tapped.

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