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Cognitive Neuropsychology Is More Than Single-Case Studies

Lynn C. Robertson, Robert T. Knight, Robert Rafal, and Arthur P. Shimamura

S. M. Sokol et al. (1991) claim that "The Cognitive Neuropsychology Approach" (p. 355) is limited to the single-case study design. The present article takes issue with this claim. Contrary to the beliefs of Sokol et al., we argue (a) that cognitive modularity is best studied by group design, (b) that the possibility of neural reorganization in patients should be tested through converging evidence from different populations using various methods, and (c) that cognitive neuropsychology can benefit from being a part of cognitive neuroscience where both neural and cognitive mechanisms underlying normal cognition are relevant.

Recently, Sokol, McCloskey, Cohen, and Aliminosa (1991) began their article concerning cognitive modularity of calculation processes with a section entitled "*The* [italics ours] Cognitive Neuropsychological Approach" (p. 355). The theme of the section was that there is only one valid approach in cognitive neuropsychology, and that is the single-case study. Group studies using patients within cognitive neuropsychology were said to be futile.

> The appropriate methodology to adopt in using data from brain-damaged patients to develop and test cognitive models is that of the single-patient study (Caramazza, 1986; Caramazza & McCloskey, 1988; McCloskey & Caramazza, 1988; Rapp, 1990). In studies involving normal subjects, data are averaged across subjects to reduce the influence of measurement error (or "noise"). Such a strategy is licensed by the assumption that the members of a group of normal subjects are homogeneous with respect to the relevant functioning of their cognitive systems (i.e., the so-called "universality assumption," see Caramazza, 1986; McCloskey & Caramazza, 1988). However, because brain damage may disrupt a cognitive system in a variety of different ways, performance disparities among brain-damaged patients cannot be dismissed as noise. Consequently, averaging data over a group of patients is inappropriate (Sokol et al., 1991, p. 356).

The pros and cons of the single-case-only approach have been widely debated in journals devoted to neuropsychology (on the pro side—Badecker & Caramazza, 1985; Caramazza, 1984, 1986, 1991; Caramazza & Badecker, 1989; McCloskey & Caramazza, 1988; and on the con side—Bates, Appelbaum, & Allard, 1991; Bub & Bub, 1988; Caplan, 1988; Newcombe & Marshall, 1988; Shallice, 1988; Whitaker & Slotnick, 1988; Zurif, Gardner, & Brownell, 1989; Zurif, Swinney, & Fodor, 1991). This controversy has been curiously one-sided. The inclusion of single-case studies in cognitive neuropsychology has not been critically questioned (although the single-case-only approach has). Many of us who conduct group studies have also reported data from single cases. Presumably, the purpose of Sokol et al. (1991) in including their introductory section was to put their case to a general cognitive psychology audience. As a result, we felt a duty to respond.

Our primary purpose in this article is not to question the conclusions of Sokol et al. (1991) concerning the modular structure of calculation processes. Rather, the issue we address is whether the general approach of Sokol et al. is sound enough to exclude patient group data in testing models of normal cognition.

It appears that Sokol et al. (1991) want to ignore many basic issues in the field of cognitive neuropsychology, including the value of biological evidence for theories of normal cognition. In addition, they have no recommendations for how one might refute evidence collected from an isolated case, since they present no objective criteria for subject selection except on the basis of the phenomenon itself. They do offer a way of supporting the evidence, and this is by finding another single case with the same deficits as the first case (see Sokol et al., 1991, p. 363, Footnote 7).

Our arguments add to the increasing size of the group of critics against the single-case-only or "radical" view.[1] We

Lynn C. Robertson, Departments of Neurology and Psychiatry, University of California, Davis, and Veterans Administration Medical Center, Martinez, California; Robert T. Knight and Robert Rafal, Department of Neurology, University of California, Davis, and Veterans Administration Medical Center, Martinez, California; Arthur P. Shimamura, Department of Psychology, University of California, Berkeley.

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Correspondence concerning this article should be addressed to Lynn C. Robertson, Neurology Service 127, Veterans Administration Medical Center, 150 Muir Road, Martinez, California 94553 or Center for Neuroscience, University of California, Davis, California 95616.

[1] Shallice (1988) referred to the single-case-only approach as Ultra Cognitive Neuropsychology, which has a similar meaning to Radical Cognitive Neuropsychology in the sense that both connote an extreme and unorthodox position. In the Sokol et al. (1991) case, the radical position is that no group studies are allowed. We use the term *radical* for historical reasons. Many of the arguments set forth by Sokol et al. and others who agree with them are similar in kind to arguments concerning group designs that radical behav-

argue that group studies in cognitive neuropsychology are, if anything, better at articulating functionally distinct components or modules than the single-case designs. Again, we are not proposing the rejection of single-case experiments as Sokol et al. (1991) proposed the rejection of group studies. Rather, we are advocating that each approach has its own usefulness, and each should complement the other; however, for issues of discovering cognitive modularity, group designs that demonstrate behavioral separability and converging evidence of biological separability are preferred. A more general goal is to develop a vision of how cognitive psychology and especially cognitive neuropsychology (defined as the use of neuropsychological data for the understanding of normal cognition) fit into the contemporary goals of cognitive neuroscience (the use of biological evidence for the understanding of normal cognition).

Modularity

As stated by Sokol et al. (1991), one of the major assumptions of cognitive neuropsychology is that cognition is "mediated by a complex information processing system comprising a number of functionally distinct components" (p. 355). In other words, the information processing system is modular.[2] In general, cognitive neuropsychologists accept this assumption. However, the radical cognitive neuropsychological approach accepted by Sokol et al. is based on the argument that only the study of single cases can uncover these components. They claim that their data support this assumption by showing that 2 patients with very different etiologies and neural damage could perform one task normally (or actually relatively normally) while performing a second task poorly. One task required multiplication of nonzero digits by 0 as in 0×7 ($0 \times N$ problems), while the other required multiplication of two nonzero single-digit numbers as in 8×7 ($M \times N$ problems). The two patients had difficulty with the $0 \times N$ problems (nearly 100% errors, at least in the first testing session),[3] but their performance on $M \times N$ problems was relatively unimpaired (15% and 25% errors). On the basis of the difference in performance between these two types of problems, Sokol et al. claimed that the two tasks are performed by different cognitive mechanisms in normals. In other words, one system could be affected in a single case (or two cases), while the other system remained relatively intact.

We agree that this conclusion is possible. There is nothing in the data of Sokol et al. (1991) to refute it. Where we part company with the radical cognitive neuropsychologists is with the claim that group studies are misleading and cannot improve the data they present (Caramazza, 1986; Caramazza & McCloskey, 1988).

We begin by exploring the benefits of group studies in cognitive neuropsychology for purely cognitive issues and specifically for questions of cognitive modularity. Suppose

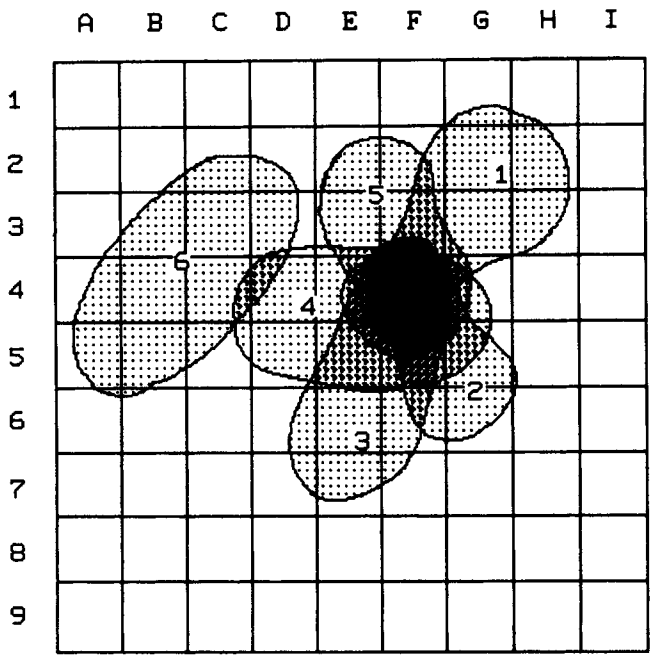

Figure 1. The grid represents a set of hypothetical cognitive modules wherein each cell represents a functional model in some cognitive space.

the grid in Figure 1 represents a set of cognitive modules. The grid need not represent the brain, although there is no reason it could not. For the moment, we set aside the assumption that the grid can be superimposed on anatomical, physiological, or neurochemical systems, and we simply let each cell of Figure 1 represent a functional module in some cognitive space. Suppose a variable number of cognitive functions have been disrupted in 6 patients. Each of the subject's func-

iorists made in previous decades. Obviously, this is not meant to imply that radical cognitive neuropsychologists and radical behaviorists have similar conceptual approaches in any other meaningful way.

[2] Fodor (1983) proposed that modules were encapsulated functional systems. In his view, modules must be cognitively unpenetrable in the sense that their functions are basically automatic and their computations cannot be changed by other cognitive processes. He further argued that modules are input systems. We concur that modularity refers to functionally distinct systems that are encapsulated, but only in the sense that a module can perform its functions without necessarily accessing other modules. We leave open the possibility that a module can be penetrated under certain task conditions (e.g., attention may modulate sensory input). We also do not agree that modular systems must be limited to input systems. We do agree with Fodor that different modules should be associated with different neural systems that can be independently disrupted.

[3] It was a bit peculiar that both patients were able to multiply by 0 in multiple digit problems (e.g., 9,103 × 80). Sokol et al. (1991) explain this difference in terms of different rule-governed responses. Multiplication by 0 of a single digit uses a different rule than multiplication by 0 of multidigit numbers where several columns are needed. Although this argument is not entirely convincing (note the difference in the 2 patients' approaches), it is not our intent to evaluate the data as they relate to the theory, but only to evaluate the claim that only data from single-case studies can be used to test the dissociation and its value to normal cognition.

tional lesions could be represented in the space as shown in Figure 1 where all but one of the subjects had a deficit in Cell 4F. It is important to realize that the distributions shown in Figure 1 are not known beforehand, and thus we do not know beforehand whether Cell 4F is involved in every subject or not. One point of testing for modularity is to determine whether there are distinct systems, either in single subjects or in groups of subjects. If Cell 4F in Figure 1 represented $0 \times N$ problems, then Subjects 1 through 5 should show some (although variable because we should expect variability within modules) performance deficits on such problems. If Subject 6 were included in the group (say because the subject's brain lesion overlapped with those of the other 5 subjects, although the subject's functional lesion did not), it would not disqualify group analysis. Subject 6 could be the extreme case on the same distribution as Subjects 1–5, which means that the functional lesion of Subject 6 really did extend into Cell 4F and that the test was not sensitive enough to observe it in this subject. In this case, the representation in Figure 1 would be wrong. Alternatively, the deficits of Subject 6 could be as shown in Figure 1 (i.e., no overlap of functional lesions at all). Rigorous experimental procedures should be able to discriminate between these two possibilities, but that is an issue concerning Subject 6 per se, and how representative this subject is of the group. It is not an argument for disqualifying the group design.

However, the radical view is stronger than this and claims that even grouping Subjects 1 through 5 is inappropriate because there is heterogeneity of functional lesions. Yet notice that in Figure 1 there is homogeneity of functional deficit for these 5 subjects *on the function of concern*, which means that a statistical test between this group and normals would likely be significant. Yet, Sokol et al. (1991) would still maintain that Subjects 1–5 can only be tested in separate single-case experiments (p. 356), because we cannot know in advance that the functional lesion is in Cell 4F. Of course, we cannot know in advance what any one subject in any group design will do on an experimental task before we test them. In the radical approach, subjects are chosen on the basis of their performance on the experimental tasks themselves with performance on one task within normal range and within the abnormal range on another task when the issue is modularity. Under such conditions, it is guaranteed that the experiment will be successful in supporting any hypothesis of independence. There are at least two critical questions that arise. First, of all the cognitive modules that have been affected by the neural damage in a single subject, which one produced the differences? Second, is a subject an outlier or a representative sample of some group?

In Figure 1, Subject 1 has functional lesions in many cells including 4F. Under such conditions, it is not possible to know whether the deficit observed is due to Cells 4F, 3G, 3H or any of the other modules that have been affected by neural damage in this subject. For instance, suppose that the functional lesions of Subject 1 were only in Cells 4F and 3G and that Cell 4F represents a calculation process and that Cell 3G represents syntax production. If Subject 1 had multiplication difficulties only when multiplying by 0 and no difficulty with $N \times M$ problems, then it would be reasonable to conclude that $0 \times N$ and $N \times M$ calculations use separate processes in this subject. Conversely, if Cell 4F represents the rules of calculating single digit numbers including $0 \times N$ problems and Cell 3G represents discrimination processes between 0 as a number and *O* as a letter, then the performance of Subject 1 would be relatively worse on $0 \times N$ problems than on $N \times M$ problems, and these were the Sokol et al. findings. The fact that no deficits were observed on other tasks testing for 0 comprehension may be deceptive because the means of measurement may not have been sensitive enough to observe such a deficit. For instance, Sokol et al. (1991) found no deficits when 0 was used in addition problems. However, addition may be a simpler task than multiplication, and the dual representation of 0 may not be as problematic in a simple task as it is in a complex task. Of course, these are questions that can be examined by experimentation. Our only point here is that a patient with multiple sources of neural damage is likely to have multiple behavioral deficits, many of which are difficult to detect and may be context dependent.

An investigator may believe a priori that the task selected requires the participation of Cell 4F but not Cell 3G for theoretical or logical reasons. However, this presupposes that 4F and 3G are already known to be distinct functional modules. If we already know this, then the experiment is not worth performing at all, neither by single-case nor by group design.

This problem can be reduced (a) when modularity is tested by group design and (b) where the subjects are not included on the basis of their performance on the experimental tasks themselves. Rather, the subjects are grouped according to some objective criteria (e.g., they all have evidence of acalculia, they all have anatomical involvement of cortical region X). If the subjects in Figure 1 showed a deficit in the task of interest as a group, then one can be substantially more confident that 4F represents a distinct module and does so across individuals. If the functional architecture is truly modular, in the sense that modules are isolated units that need not affect one another in a given stage of processing, then variability around but including 4F would still produce a deficit in any task that requires the computation that 4F performs. Variable dysfunction in modules over a group of subjects strengthens the case for functionally distinct components as opposed to weakening it as the radical single-case view proposes. The irony of this increased confidence in the independence of different functions through group design is that the radical cognitive neuropsychologists' main reason for asserting that patients cannot be grouped is that these patients have heterogeneous functional deficits (presumably reflecting different functional components). Clearly, if subjects in a group are too heterogeneous, then variability in performance will be high, and significant results will not be observed. When significant differences are obtained, it can be assumed that subjects are homogeneous on the factor of interest, or at least homogeneous enough for the effect to be observed statistically.

Certainly, there is the possibility that a group of patients may perform as if there were a $0 \times N$ module, but in fact each individual's performance deficit would be due to a different pattern of functional deficits. That is, averaging over subjects

can be misleading, because no individual subject may show the dissociation of interest for the reason proposed, which is a historically difficult problem for group studies overall. However, this type of error is far more likely to occur in a series of single-case studies where there is no known common etiology, neuropsychological profile, and/or neurobiological involvement.

Cognitive Modularity and Neural Modularity

We propose that for the development and testing of models of normal cognition, a single patient's deficits are generally most useful as a first approximation that should be followed by group studies, if possible.[4] Often the hypothesis in cognitive neuropsychology is that a given physical lesion is likely to produce a given cognitive deficit (i.e., a given functional lesion), while a different physical lesion will not. If the hypothesis is supported, then there is evidence for functional modularity, at least in the sense that these two functions do not rely on each other to perform the task.

We use an example from our own work where both cognitive and neural modularity were supported. Robertson, Lamb, and Knight (1988) showed that, in a group of subjects with lesions centered in the left superior temporal gyrus and adjacent temporal-parietal junction, the analysis of local information in a compound visual stimulus was disrupted, but a similar area in the right hemisphere disrupted global analysis. If a group of subjects with left temporal-parietal damage is different from a group with right temporal-parietal damage, then more confidence can be given to the conclusion that the behavioral dissociations are due to different mechanisms. For purposes of demonstrating cognitive modularity, the location of the lesions is not as important as the fact that the lesions are distinct. If the location of the lesions has implications for issues in neuroscience as well, this does not change their relevance for cognitive theory.

What would Sokol et al. (1991) do with the positive findings from the Robertson et al. (1988) group study? From our reading of the arguments, they would dismiss them as having no bearing on theories of normal cognition, because the group as a whole is functionally heterogeneous. "Functionally heterogeneous on what?" we ask. In each group, the subjects were homogeneous enough to produce significant group differences on the functions of concern (or at least on the functions that were assumed to be tested by the experimental procedures). Is the argument of Sokol et al. with the different number of functions affected between subjects or with the different distributions? Is it in the variable magnitude of the effect of the function being tested, or is it in some other variable as yet unidentified? There is no question that between-subjects heterogeneity can be eliminated as a source of error by choosing to study a selected case, but this position is extreme, and it neglects the central issue of generalizability. We know there is heterogeneity in all groups, and our statistics take that fact into account.

As an exercise in the approach of Sokol et al. (1991), we selected Subject R. H. from Robertson et al.'s (1988) left-hemisphere group and Subject H. S. from their right-hemisphere group (see Table 1). As is often done in single-case reports, (whether intentionally or not), these two patients were selected as the best candidates. In other words, they are the extremes in their groups in terms of being normal when responding to one level but outside the normal range when responding to the other level. As extremes, are their data more compelling in addressing questions of functionally distinct components than the group as a whole? Perhaps to the layperson they are, because their effects are exaggerated. However, this practice would be analogous to selecting the 1 or 2 subjects in any group, including a group of normals, that support a particular theory. A second subject from each side of the distribution could be selected, and each subject could be presented as a separate experiment to demonstrate that extremes can be replicated. The rest of the subjects could be dropped from the experiment. It is obvious that the rest of the subjects can increase noise and reduce the probability of finding a significant difference between the groups. But a significant effect was found between the groups in the Robertson et al. (1988) study despite the variability inherent in group studies using patient populations.

Table 1
Mean Reaction Time in Milliseconds for One Right- and One Left-Hemisphere-Damaged Patient and a Group of Matched Normal Controls

Subjects	Target level	
	Global	Local
Patients		
H. S. (right hemisphere)	973	634
R. H. (left hemisphere)	687	1,188
Controls		
Mean	688	637
Standard deviation	91.4	87.3
Range	588–806	531–786

Perhaps the radical cognitive neuropsychologists' position is best understood by an example from research meant to support their position. Arguing against group studies, Miceli, Silveri, Romani, and Caramazza (1989) showed that collapsing over 20 subjects with the neuropsychological diagnosis of agrammatism did not produce reliable differences between patient and normal groups. When Miceli et al. examined the individual data, they found 2 subjects who (they argued) were qualitatively different from each other and therefore represented two different populations. Miceli et al. presented these 2 subjects' data as evidence for distinct grammatical processes and argued that the group design blanketed the important effects in individuals. In response, Bates et al. (1991) applied well-established, stricter screening criteria that were based on the use of critical biological variables to

[4] Sometimes there is only 1 or a handful of patients who show a particular deficit. Their deficits are often dramatic, just as it is dramatic when one finds a person with true eidetic imagery or an idiot savant. In such cases, a thorough case study may be the only option, but unless other means of testing similar functions in neurologically intact or other patient populations are found, the universality of the phenomenon must remain in question.

reanalyze the data from the 20 subjects reported by Miceli et al. (e.g., no patients in the acute stage, no crossed aphasics), and they demonstrated that reliable and meaningful group differences were observed in the smaller subset of patients when such criteria were applied. Bates et al. also formally demonstrated that the two extreme cases that Miceli et al. reported to support conclusions about distinct processes could occur by chance in a group of 20 subjects.

When one reads the examples that the single-case-only advocates use to support their position, the examples are often in the form of obtaining a null effect that is due to variability in a group defined by a clinical syndrome. In other words, they are mainly concerned with Type II errors. If the radical cognitive neuropsychology advocates are simply arguing that the practice of using clinical classifications to justify group inclusion (as opposed to behavioral or biological criteria), then their arguments are limited to the question of what should be included in a clinical syndrome, which is a question that is outside the focus of cognitive neuropsychology but is of central importance to the field of clinical neuropsychology. Clinical syndromes were not developed to act as a criterion for experimental groups. They were developed for clinical purposes to allow a common language between clinicians. Therefore, it should be no surprise that rigorous testing of component operations could show enormous variability between subjects that are selected solely on the basis of a clinical syndrome. Although this state of affairs suggests that clinical classification might profit from further subdivision, the only bearing that it has on cognitive neuropsychological research is to alert cognitive neuropsychologists to the increased possibility of a Type II error when these syndromes are used to form groups. This is not a position that warrants the elimination of group studies. When differences are found, concern over Type II errors must be replaced with concern over Type I errors.

In summary, we fully agree with the radical cognitive neuropsychologists in that studies of brain-injured patients as a group are difficult to use to address the null hypothesis (Caramazza, 1991), but this problem is magnified in a single case. On the other hand, both group and single-case studies can be used to show a dissociation between tasks, and when dissociations occur, the group design is superior for two reasons. First, it can demonstrate that significant differences occur despite variable functional deficits in processes outside the ones of concern, which increases our confidence in the universality of distinct operations. Second, group studies typically offer objective subject inclusion criteria for experimenters who wish to replicate, extend, or refute the results of other studies. Finally, if common physical lesions in a group disrupt one process but not another, then the physical evidence, at the very least, could be used as a reasonable starting point for group inclusion whether one's interest is only in cognitive theory or not. It is difficult to know what criteria one should use within the single-case-only approach.

Both G. E. and P. S. in the Sokol et al. (1991) study had a deficit in multiplying by 0, yet each had very different neuropathology and etiology. To pursue this finding experimentally, either one would have to give the tasks of Sokol et al. to every patient who presented with neuropsychological problems in an attempt to find another case having such a deficit (a self-fulfilling replication) or one would have to search for other inclusion criteria, one of which could be the common neuropathology between G. E. and P. S.

The fact that 2 separate subjects tested by Sokol et al. (1991), who were probably selected because they had a clinical diagnosis of acalculia (although we are not informed whether this was the selection criterion or not), both had difficulty with multiplying by 0s (but only in single-digit problems) does not solve the averaging problem of group design. We cannot help but wonder whether the dissociation seen in two very different patients who are clearly not homogeneous (except that they have difficulty multiplying by 0) could result from unexpected sources. Are the amygdala and hippocampus of 1 subject compromised, and could the 0 calculation simply be more difficult to remember? Is there orbital frontal damage? P. S. or G. E. may know the answer to $0 \times N$ problems but be unable to inhibit the incorrect response. Are 0s easier or harder to discriminate than other digits? Do subtle deficits not detected in baseline measures of comprehension of 0 interact with other deficits to form the pattern of results?

Conversely, if no other patient is ever found who has such a dissociation, what does this say about these 2 individuals specifically and about the radical approach in general? It seems the conclusion would be that these 2 subjects could be exceptions to the rule. They show that differences can occur between tasks (i.e., they are existence proofs). They show that $0 \times N$ and $M \times N$ calculations can be affected differentially in 2 subjects. By definition, they do not represent a population because there can be no group study of the phenomenon in radical cognitive neuropsychology. Finally, if only some patients with the diagnosis of acalculia show the dissociation and others do not, then this is an issue concerning clinical classification and has no bearing for cognitive neuropsychologists except to warn us that the use of that particular clinical classification to form experimental groups without additional screening criteria can reduce the probability of observing a significant effect. The radical cognitive neuropsychological solution to this problem is to use the existence of the dissociation itself as the major inclusion criterion, which makes the findings irrefutable. If we test 100 patients and select the 2 or 3 who show the effect and report their data as independent studies that replicate one another, then the data in the literature are guaranteed to support the hypothesis.

Selective Dysfunction and Its Relation to Normal Function

If the only goal of cognitive neuropsychology were to demonstrate that modularity can exist in the universe of cognition, then the radical cognitive neuropsychological approach might be reasonable. But as most cognitive neuropsychologists believe, whether radical or not, the question is not simply whether some functions are distinct in some patients, but more importantly, are the functions distinct in most people? What do the modules do? And how and when do they interact with one another?

Over a hundred years of study in neuropsychology using both group and single-case designs has shown that modularity does exist in the human brain, but at the same time neural systems are highly interactive. Also, modularity, as we use the term here, can exist at one stage of processing but not at another. Evidence for modularity is strong in many domains, and it is an empirical problem to determine which functions exhibit strong independence and which do not. Single-case studies can be an important initial clue. They can be used to formulate hypotheses about both cognitive and brain processes, which can then be tested by group design whether in normals or other brain-damaged subjects.

For example, visual loss need not disrupt auditory language comprehension or vice versa. If this were not already well known, then the study of 1 patient with visual loss but no language comprehension deficits and another with the reverse would be exciting and would demonstrate that such modularity can exist. It would then be reasonable to design studies to test for modularity of the same systems in normals as well as in other brain-damaged groups, and without much work, methods to reveal it would probably be found. For instance, the findings of Robertson et al. (1988) that supported independence between global and local processing advantage in the right and left hemisphere received converging support in studies using visual field presentation in both normals and patients with full commissurotomy (Martin, 1979; Sergent 1982; Robertson, Lamb, & Zaidel, in press). It is doubtful that testing 1 or 2 brain-damaged subjects would be as convincing.

Returning to our example of evidence for modularity of vision and language comprehension in a single case, we may want to go on to ask other questions. One question might be, which tasks require both functions (e.g., pointing to an object upon command) and which require only one function? Note that the task is crucial in determining what is modular, what is not, and when it is. Another question might be, what information is essential in the auditory signal for normal language comprehension? One way to approach this question is through studies of normals that vary the different parameters of the signal to discover when comprehension breaks down. Another way is to test patient populations who have comprehension difficulties to determine which parameters can still be heard. If the two sets of data converge on the same conclusion, then scientific advancements have been made in understanding both normal and abnormal comprehension, and the two sets of data strengthen confidence in arguments concerning what is required for normal language comprehension.

We argue that studying various populations with converging evidence is a better test of the second assumption made by Sokol et al. (1991), which states that "brain damage may selectively disrupt this information processing system without bringing about a qualitatively different organization of function *de novo*" (p. 355), than is the single-case-only approach. This does not mean that the exhaustive study of a single case, either normal or brain injured, cannot lead to important and interesting hypotheses. However, it is no secret that people vary enormously, and it is because of this variability that more than 1 subject is tested, if possible. If the effects are found in groups—whether the effects support modularity, reveal an algorithm by which the module functions, or point to neural substrates—then there is obviously more generalizability and predictability than when the effect is limited to 1 or 2 individuals; especially if these individuals are selected because they are exceptions rather than the rule.

Where Is the "Neuro" in Radical Cognitive Neuropsychology?

One final problem in the approach of Sokol et al. (1991) is that the neural correlates of cognitive deficits are deemed to be virtually irrelevant. Although this problem may not be a major concern for cognitive psychologists, it is a major concern for investigators in areas within cognitive neuroscience including those in cognitive neuropsychology. The goal of cognitive neuroscience is to understand the biological basis of normal cognition. This is not the goal of most cognitive psychologists, although several have found it advantageous to use biological evidence to support their theories (Kosslyn, 1988; LaBerge, 1990; Posner & Petersen, 1990; Schacter, 1989). The use of the term *cognitive neuropsychology* implies that it is a part of cognitive neuroscience in the sense that one of its goals is to understand the normal relationship between cognition and the brain.

Neuropsychology has always been, and continues to be, the study of brain–behavior relationships. The radical cognitive neuropsychologists acknowledge this goal when they agree that cognitive neuropsychology includes the investigation of "brain/cognitive mechanism relationships" (McCloskey & Caramazza, 1988, p. 612) and when they conclude that "cognitive neuropsychology is a fast-growing, vibrant field of study with increasingly significant contributions to our understanding of normal cognitive processes and the functional organization of the brain" (Caramazza & McCloskey, 1988, p. 527). However, the use of the term *cognitive neuropsychology* is misleading within the framework outlined by Sokol et al. (1991), because their method leaves the brain out of the relationship. Although the definition of behavior and cognitive mechanisms and what should be included in such concepts can be debated, the definition of brain is quite clear. The role of the brain and the way in which single-case studies can articulate brain function are in need of clarification. Sokol et al. can always say that this relationship was not the goal in their article, and indeed, it was not. However, the fact that "The Cognitive Neuropsychological Approach" (p. 355) was stated explicitly in their article to be limited to single-case studies and the fact that one of the authors of the article (McCloskey) has acknowledged that cognitive neuropsychology includes the study of brain–cognitive mechanism relationships mandate an answer to the question of how a cognitive neuropsychology that is based only on single cases can address the role of neural substrates in normal cognition. What methodology is appropriate to answer questions concerning this relationship?

Of course, it is true that cognitive processes can be studied independently of the neural processes that support them, just

as neural processes can be studied without relating them to cognitive or behavioral systems. In neither case does the word *neuropsychology* seem appropriate. In radical cognitive neuropsychology the neural substrates seem to be of interest only in some abstract sense where the neurological status of the single case is reported, but nothing more is made of it. This can be seen in the description of G. E. in the Sokol et al. (1991) article:

> G. E. is a right-handed man who sustained a closed-head injury as a result of a fall. . . . At the time of hospital admission he exhibited right-sided weakness, confusion, and agitation. CT [computerized tomography] scans revealed a left frontal contusion. G. E.'s medical history also includes seizure disorder (controlled with medication) and alcohol abuse (p. 368).

Are G. E.'s deficits due to orbitofrontal or anterior temporal damage related to the fall, to the chronic effects of alcohol, to the history of seizures, to the seizure medication, or to some chance interaction between his multiple medical problems? The fact that P. S. showed a similar deficit with a completely different etiology and neurological profile only confuses the issue.

> P. S. is a right-handed woman who suffered a left-hemisphere cerebral vascular accident (CVA) in 1985, at the age of 38. Arteriograms revealed an intracranial bleed resulting from either an arteriovenous malformation or small aneurysm. As a consequence of her CVA, P. S. retains right-sided weakness and a rather severe verbal dysfluency (Sokol et al., 1991, p. 357).

If cognitive neuropsychology is the study of brain–cognitive mechanism relationships, what are we to make of the differences in neuropathology? Is there any specific neural injury that would systematically affect $0 \times N$ problems? The data for P. S. and G. E. are more confusing than helpful. They also provide no objective criteria on how one might find other subjects with the same problem. How many patients were tested on the task before P. S. and G. E. were discovered? How did those who were rejected, if any, perform and what were their etiologies and neurological profiles? On what basis were P. S. and G. E. recruited for testing? Without this type of information, there has been no apparent advance concerning the question of brain–cognitive mechanism relationships. Whether brain mechanisms are of interest and whether brain–cognitive mechanism relationships can be addressed by only single-case studies need to be addressed explicitly if the radical cognitive neuropsychologists wish to define a methodology that applies to the goals of the entire field.

Summary

There are scientific, theoretical, and pragmatic reasons to define "The Cognitive Neuropsychological Approach" as including both the single-case and the group design but not to limit it to single cases only. It is useful to collapse patients into groups, and this is especially the case when testing for modularity of cognitive functions. The single-case-only approach described by Sokol et al. (1991) is limited in its stated goals. It solves the problem of between-subjects variability by sacrificing generalizability, predictability, and the possibility of refutation. Finally, it does not address issues within brain sciences, and this position challenges the definition of cognitive neuropsychology as including the study of brain–cognitive mechanism relationships. Certainly, there can be real methodological problems in selecting a group of patients for study in cognitive neuropsychology, but at least part of the solution requires a sound foundation in neuropathology and rigorous screening that is based on neuropsychological and biological criteria. Cognitive science and neural science are converging on the common goal of understanding the computational and biological basis of normal human cognition. If this is to be a serious enterprise, a convergence of scientific disciplines is needed in which various approaches support and stimulate one another. If for no other reason, the position of the radical cognitive neuropsychologists is disturbing because it may limit young investigators entering the field. Students may be tempted to stay safely within their paradigms and may be discouraged from looking to other disciplines that may contribute the answers they seek.

However, the most important limitation of the approach advocated in the Sokol et al. (1991) article is the potential elimination of a source of data. Given such a position, cognitive psychologists should be very cautious about accepting such a radical view.

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